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

Appendix B: Methods and Results
B3: Wildlife Risk Assessment

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LIST OF ACRONYMS

ATSDR	Agency for Toxic Substances and Disease Registry
BMR	Basal metabolism rate
BRCC	Bushy Run Research Center
COPCs	Constituents of Potential Concern
CSO	Combined Sewer Overflow
EEC	Estimated exposure concentrations
EED	Expected environmental dose
HQ	Hazard quotient
IRIS	Integrated Risk Information System
LOAEL	Lowest observed adverse effect level
NAS	National Academy of Sciences
RTECS	National Institute for Occupational Safety and Health
NOAEL	No observed adverse effect level
NRC	National Research Council
NTP	National Toxicology Program
ORNL	Oak Ridge National Research Laboratory
PAHs	Polycyclic aromatic hydrocarbons
PCBs	Polychlorinated biphenyls
PDF	Probability distribution function
RTECS	Registry of Toxic Effects of Chemical Substances
TBT	Tributyltin
TRV	Toxicity reference value
U.S. EPA	U.S. Environmental Protection Agency
USFWS	U.S. Fish and Wildlife Service
WERF	Water Environment Research Foundation

1. INTRODUCTION

This appendix presents the methods and results used in the wildlife risk assessment. This includes the information used to determine the receptor-specific exposure concentrations that would result from wildlife eating food and drinking water from the study area. The four wildlife receptors (great blue heron, bald eagle, spotted sandpiper, and river otter) and the two scenarios (baseline condition and without CSOs) evaluated here were first identified and developed in Appendix A1 - *Problem Formulation*.

Section 2 discusses the approaches and data used to select the toxicity reference values (TRVs) for each receptor. Section 3 presents the spatial and temporal use of study area by wildlife receptors along with the unique elements of receptor biology that determine the doses to which each receptor is exposed. Section 4 details the risk characterization methods used to probabilistically combine the results of the wildlife effects and exposure assessments. Section 5 summarizes the results of the risk characterization, while Section 6 presents the conclusions of the wildlife risk assessment. Interpretation of the data and results presented here can be found in Volume 1 – *Overview and Interpretation* report.

2. WILDLIFE TOXICOLOGICAL EFFECTS CHARACTERIZATION

The methodology used to select toxicological effects data for the wildlife receptors (river otters, great blue heron, bald eagle, and spotted sandpiper), are discussed below and are followed by the data used in the risk assessment. For a general discussion of these selection methods, see Issue Paper No. 7, "*Aquatic Life and Wildlife Toxicology*" in Appendix C.

2.1 Methodology

No U.S. EPA or State of Washington wildlife criteria or standards currently exist. Consequently, wildlife TRVs were obtained from the following information sources:

- U.S. Fish and Wildlife Service Contaminant Review series (e.g., Eisler 1988),
- the Agency for Toxic Substances and Disease Registry (e.g., ATSDR 1991) documents,
- the Oak Ridge National Laboratory database (Sample et al. 1996), and
- the scientific literature.

Chronic toxicological effects data (i.e., data on effects observed after test animals were exposed over a significant portion of their lifetime) were the objective of this search. A chronic effect threshold generally is based on the highest dose resulting in no-observed-adverse-effect (the no observed adverse effect level or NOAEL) or the lowest dose resulting in an observed adverse effect (the lowest observed adverse effect level or LOAEL). If a NOAEL was not available for a chemical, it was estimated to be ten percent of the LOAEL (U.S. EPA 1994). These values (NOAEL and LOAEL) actually represent the lower and upper bounds of the true TRVs (or thresholds for toxic effects). However, the process by which the upper and lower bounds are identified provides no guide to where the true TRV lies. Thus, it was assumed for the purposes of this risk assessment that the true toxicity value had an equally likely probability of lying anywhere between the NOAEL and LOAEL. This probably was represented in the risk characterization by the use of a uniform distribution (Ross 1985).

All toxicity studies were evaluated on the relevance of toxic endpoints investigated, and the dosing regime and dosing medium used to expose test organisms. As described in Appendix A1 – *Problem Formulation*, wildlife risk assessments typically assess risks to populations, but as individual bald eagles and spotted sandpiper are protected under the Endangered Species Act and Migratory Bird Act, respectively, risks to individuals were assessed for these receptors. Risks to the great blue heron and river otter were assessed at the population level. Population-level toxicity endpoints include reproduction, development, and survival, while endpoints for assessing risks to individuals also include growth reductions and systemic effects such as organ damage. In the absence of toxicological data for the preferred population-level toxicity endpoints for the great blue heron or river otter, impacts on growth or other systemic effects were substituted.

A dosing regime is the method for delivering a dose. Typical methods are ingestion, gavage (use of a stomach feeding tube), or intraperitoneal injection (injection into the abdominal cavity). Acceptable dosing regimes that most closely approximate actual environmental exposures include ingesting food or water. Animal dosing regimes that are not considered representative of environmental exposure scenarios include gavage and intraperitoneal injection. However, when information is only available from gavage studies, such as for polycyclic aromatic hydrocarbons (PAHs), then these alternative studies are used. All toxicity data were expressed as milligrams per kilograms of body weight per day. The wildlife toxicity data used were based on daily dose levels normalized to the body weight of the test species. This is necessary to allow evaluation of wildlife toxicity data across tests and species (Sample et al. 1996).

The dosing medium is the form of the chemical used in the experimental study. An example of an acceptable dosing medium would be inorganic mercury salts, such as mercuric chloride. Mercury-containing fungicides (e.g., Ceresan, methyl mercury dicyandiamide) were not considered relevant dosing media due to the possible additive effects of the non-mercury components, and many bird studies were excluded for this reason.

Finally, due to the lack of toxicity studies for our specific receptors, surrogate species were used. Additionally, many wildlife studies available in the scientific literature could not be used because individual effects were related to chemical residue levels in animal tissue instead of known dosing regimes. Therefore, clear-cut dose-response relationships could not be identified. Toxicological effects data (doses) were more readily available for domestic and laboratory animals, such as rats, mice, chickens, mallards, and quail. Whenever possible, mammalian toxicity data were used to represent mammalian receptors and avian data to represent avian receptors. When data were only available for mammals, the mammalian toxicity data were used to estimate the NOAEL for the avian receptor. Use of surrogate species introduced an additional level of uncertainty in our assessment of the potential toxicity of a chemical to a wildlife species. To address this uncertainty, an additional margin of safety was applied either by adding a safety factor, or by scaling the toxicity data based on test and receptor species body weight.

For mammals, scaling the toxicity dose based on the body weight of the test and receptor species is recommended (Travis and White 1988, Travis et al. 1990, U.S. EPA 1992). Research has demonstrated that numerous physiological functions, such as metabolic rates and responses to toxic chemicals, are functions of body size (Sample et al. 1996). Differences in metabolic rates can lead to more resistance to toxic chemicals because of the rate of detoxification through metabolism and excretion of the chemical (Sample et al. 1996). Body weight scaling, however, has not been found to be appropriate for birds (Fischer and Hancock 1997). For birds, differences in toxicological reactions appear to

be more a factor of whether the species is passerine[1] or nonpasserine (Fischer and Hancock 1997). Additional methods applied specifically to the mammalian versus the avian receptors are discussed below.

2.2 Great Blue Heron, Bald Eagle, and Spotted Sandpiper

As noted previously, potential risks were evaluated to great blue heron *populations* and bald eagle and spotted sandpiper *individuals*. No toxicity data were available for any of these receptors. Toxicity data for surrogate species (e.g., chickens, mallards and quails) were used instead. For some chemicals (e.g., PAHs and 1,4-dichlorobenzene), toxicity data for birds were not available, and therefore, toxicity data from mammalian test species were used. To account for potential differences in species sensitivities, safety factors derived using best professional judgment were applied to the selected NOAEL and LOAEL data. For the great blue heron, a safety factor of two was applied, while a safety factor of five was applied for the bald eagle and spotted sandpiper. A larger safety factor was used for the bald eagle and spotted sandpiper to protect sensitive individuals in their general populations. With these safety factors, it was conservatively estimated that the general receptor population was two times more sensitive and specific individuals five times more sensitive than the test species. It should be noted that the specific relationships between receptors and test species are unknown.

2.3 River Otter

There were no toxicity data available from laboratory studies of river otters. Therefore, surrogate species were used to estimate the toxicity of chemicals to the river otter. The surrogate species used included mink, rat, and mouse. Mink data were used preferentially over rat and mouse data when available, because mink are more closely related (common family) to otters than rats or mice. As noted above, toxicity data based on these surrogate species were adjusted using body weights of test species and river otters. The following formula was used for scaling toxicity data based on body size (Sample et al. 1996):

$$\text{NOAEL}_w \text{ or } \text{LOAEL}_w = \text{NOAEL}_t \text{ or } \text{LOAEL}_t \left(\frac{\text{BW}_t}{\text{BW}_w} \right)^{1/4} \qquad \text{(Equation 2-1)}$$

Where:

NOAEL_w = No observed adverse effects level for mammalian wildlife receptor

[1] Passerines are the perching songbirds (such as starlings) and account for approximately half of all known bird species. These birds have a very different physiology that influences their response to toxicants and distinguishes them from the nonpasserine birds (such as ducks and chickens).

$LOAEL_w$ = Lowest observed adverse effects level for mammalian wildlife receptor
$NOAEL_t$ = No observed adverse effects level for mammalian test species
$LOAEL_t$ = Lowest observed adverse effects level for mammalian test species
BW_w = Body weight of mammalian wildlife receptor
BW_t = Body weight of mammalian test species

This equation is the appropriate one to use when the test organisms have smaller body weights than the receptor. The body weights used are presented below in Table 2-1.

Table 2-1. Average Body Weights for River Otters and Mammalian Test Species Used in the Body Weight Scaling

Type	Species	Body Weight (kg)	Reference
Wildlife Receptor	River otter	8.6	U.S. EPA (1993)
Test Species	Rat	0.25	RTECS (1985)
	Mouse	0.025	U.S. EPA (1988)
	Mink	1.7	U.S. EPA (1993)

2.4 Selected Toxicity Values

The TRVs for mammals and birds are shown in Table 2-2 and Table 2-3, respectively. The toxicological endpoints for the otter included reproductive effects, such as decreased litter size, and reduced fertility, and kidney and liver degeneration. The endpoints for the avian receptors included reproductive effects, such as reduced hatchability and eggshell thinning, kidney damage, and growth reductions. For most chemicals and receptors, reproductive effects were the most sensitive toxicological effect endpoint. For zinc, a separate value was selected to protect the individual eagle and sandpiper because effects on growth occurred at a lower level then those for reproduction. Toxicity data were available for all chemicals except some PAHs. For these, the toxicological effect data for another PAH, benzo(a)pyrene, were substituted. For the avian receptors, PAH toxicity data were based on mammalian test species for all but fluoranthene and pyrene. For these chemicals, mallard toxicity data were available.

2.5 Effects Characterization Uncertainty

The effects characterization treated uncertainty about toxicity reference values (TRVs) two ways. First, safety factors were applied when we had to extrapolate toxicity data, for example from one species to another or one toxicity endpoint to another. After safety factors were applied, we had a range of possible values for each wildlife TRV. Second,

we applied probability distributions to these ranges. At the upper end of each TRV's range was the lowest observed adverse effects level (LOAEL), and at the lower end the no observed adverse effects level (NOAEL), both adjusted by the safety factors. We had no toxicity data between the NOAEL and the LOAEL, so we used a uniform probability distribution to characterize uncertainty. The uniform distribution assumes the TRV lies between the NOAEL and LOAEL, but that we don't know where it falls within this range.

Table 2-2. TRVs for the River Otter

Analyte	Literature LOAEL (mg/kg/d)	Scaled LOAEL (mg/kg/d)	Literature NOAEL (mg/kg/d)	Scaled NOAEL (mg/kg/d)	Test Organism	Effect	References
Metals/Metalloids							
Arsenic	1.26	0.52	0.126[a]	0.052	Rat	Decreased litter size	ATSDR (1991); Schroder and Mitchener (1971)
Cadmium	1.9	0.4	1	0.2	Mouse	Reproductive failure	ATSDR (1991); Schroeder and Mitchener (1971); ORNRL (1996)
Copper	15.1	10.1	11.7	7.8	Mink	Kit mortality	ORNRL (1996); Aulerich et al. (1982)
Lead	1.5	0.3	0.15[a]	0.04	Mouse	Reproductive success of implanted ova	Eisler (1988); Clark, (1979)
Mercury (inorganic)	3	1.2	0.09	0.06	Rat, mink	Kidney damage (rat), no clinical/ pathological signs of tox. (mink)	Carmignani et al. (1989); Wobeser et al. (1976)
Nickel	80	33	40	17	Rat	Decreased offspring per litter	ORNRL (1996); Ambrose et al. (1976)
Zinc	320	132	160	66	Rat	Increased fetal resorption	Schlicker and Cox (1968)
Organometallics							
Tributyltin	3.4	1.4	0.34[a]	0.14	Rat	Decreased pup weight	IRIS (1998)
Polychlorinated Biphenyls							
Aroclor 1016	3.43	2.29	1.37	0.91	Mink	Reproductive effects	Eisler (1986); Ringer (1983)
Aroclor 1221	3.43	2.29	0.447	0.298	Mink	Reproductive effects	Eisler (1986); Ringer (1983)

Table 2-2. TRVs for the River Otter (continued)

Analyte	Literature LOAEL (mg/kg/d)	Scaled LOAEL (mg/kg/d)	Literature NOAEL (mg/kg/d)	Scaled NOAEL (mg/kg/d)	Test Organism	Effect	References
Aroclor 1232	0.34	0.23	0.14	0.09	Mink	Fertility, whelping, number of kits	Wren et al. (1987); Hornshaw et al. (1983)
Aroclor 1242	1.12	0.75	0.447	0.298	Mink	Reproductive failure	Eisler (1986); Ringer (1983)
Aroclor 1248	0.34	0.23	0.14	0.09	Mink	Fertility, whelping, number of kits	Keplinger et al. (1971)
Aroclor 1254	0.34	0.23	0.14	0.09	Mink	Fertility, whelping, number of kits	Wren et al. (1987); Hornshaw et al. (1983)
Aroclor 1260	6	2.48	0.06	0.03	Rat	Stillborns and pup survival	NAS (1979); Burke and Fitzhugh (1970); Keplinger et al. (1971).
Total PCBs	0.34	0.23	0.14	0.09	Mink		
Organics							
1,4-Dichlorobenzene	600	139	40[a]	17	Mouse, rat	Liver degeneration, decreased white blood cell count (mouse), no effects on liver or immune system (rat)	ATSDR (1991); Gaines and Linder (1986); NTP (1987)
4-Methylphenol	N/AV[b]	N/AV[b]	450	186	Rat	Reproduction	ATSDR (1990); BRRC (1989)
Benzo(a)anthracene[c]	10	2.3	1[a]	0.2	Mouse	Reproductive effects	MacKenzie & Angevine (1981)
Benzo(a)pyrene	10	2.3	1[a]	0.2	Mouse	Reproductive effects	MacKenzie & Angevine (1981)
Benzo(e)pyrene	10	2.3	1[a]	0.2	Mouse	Reproductive effects	MacKenzie & Angevine (1981)
Benzo(b)fluoranthene[c]	10	2.3	1[a]	0.2	Mouse	Reproductive effects	MacKenzie & Angevine (1981)

Table 2-2. TRVs for the River Otter (continued)

Analyte	Literature LOAEL (mg/kg/d)	Scaled LOAEL (mg/kg/d)	Literature NOAEL (mg/kg/d)	Scaled NOAEL (mg/kg/d)	Test Organism	Effect	References
Benzo(g,h,I)perylene	10	2.3	1[a]	0.2	Mouse	Reproductive effects	MacKenzie & Angevine (1981)
Benzo(k)fluoranthene[c]	10	2.3	1[a]	0.2	Mouse	Reproductive effects	MacKenzie & Angevine (1981)
Bis(2-Ethylhexyl) phthalate	183.3	42.56	18.3	4.25	Mouse	Reproductive effects	ORNRL 1996; Lamb et al. (1987)
Chrysene[c]	10	2.3	1[a]	0.2	Mouse	Reproductive effects	MacKenzie & Angevine (1981)
Dibenzo(a,h)anthracene[c]	10	2.3	1[a]	0.2	Mouse	Reproductive effects	MacKenzie & Angevine (1981)
Fluoranthene	250	58.1	125	29.0	Mouse	Systemic	IRIS (1998)
Indeno(1,2,3-cd)pyrene[b]	10	2.3	1[a]	0.2	Mouse	Reproductive effects	MacKenzie & Angevine (1981)
Pyrene	125	29.0	75	17	Mouse	Systemic	HEAST (1995)
Phenanthrene[b]	10	2.3	1[a]	0.2	Mouse	Reproductive effects	MacKenzie & Angevine (1981)

[a] The NOAEL was estimated from the LOAEL using an uncertainty factor of 10.

[b] N/AV = Not Available

[c] LOAEL and NOAEL estimated using benzo(a)pyrene as a "surrogate" PAH.

Table 2-3. TRVs for Avian Receptors

Analyte	Literature LOAEL (mg/kg/d)	Heron LOAEL[a] (mg/kg/d)	Eagle, Sandpiper LOAEL[b] (mg/kg/d)	Literature NOAEL (mg/kg/d)	Heron Level NOAEL[a] (mg/kg/d)	Eagle, Sandpiper NOAEL[b] (mg/kg/d)	Test Organism	Effect	References
Metals/Metalloids									
Arsenic	12.8	6.4	2.6	5.14	2.57	1.03	Mallard	Mortality	ORNRL (1996); USFWS (1964)
Cadmium	4.4	2.2	0.9	1.45	0.73	0.29	Chicken, mallard	Decreased egg production	NRC (1980); Leach et al. (1979); Scheuhammer (1987); White and Finley (1978)
Copper	61.7	30.9	12.3	47	24	9.4	Chicken	Weight gain and mortality	ORNRL (1996); Mehring et al. (1960)
Lead	0.72	0.36	0.14	0.072	0.036	0.014	Japanese quail	Delayed egg production	Scheuhammer (1987); Edens et al. (1976)
Mercury (inorganic)	0.74	0.37	0.15	0.37	0.19	0.07	Japanese quail	Eggshell thinning	Stoewsand et al. (1971)
Nickel	107	53.5	21.4	77	39	15	Mallard	Mortality and reduced growth	ORNRL (1996); Cain and Pafford (1981)
Zinc	137	68.5	27.4	131	65.5		Chicken	Reduced hatchability	Eisler (1993); Stahl et al. (1990)
Zinc				90		18	Chicken	Decreased growth (individual effect)	Roberson and Schaible (1960)

Table 2-3. TRVs for Avian Receptors (continued)

Analyte	Literature LOAEL (mg/kg/d)	Heron LOAEL[a] (mg/kg/d)	Eagle, Sandpiper LOAEL[b] (mg/kg/d)	Literature NOAEL (mg/kg/d)	Heron Level NOAEL[a] (mg/kg/d)	Eagle, Sandpiper NOAEL[b] (mg/kg/d)	Test Organism	Effect	References
Organometallics									
Tributyltin	16.9	8.45	3.38	6.8	3.4	1.36	Japanese quail	Reduced hatchability and egg weight	ORNRL (1996); Schlatter et al. (1993)
Polychlorinated Biphenyls									
Aroclor 1016	0.91	0.46	0.18	1.83	0.92	0.37	Chicken	Egg hatchability, teratogenic effects	Cecil et al. (1974)
Aroclor 1221	0.91	0.46	0.18	1.83	0.92	0.37	Chicken	Egg hatchability, teratogenic effects	Cecil et al. (1974)
Aroclor 1232	0.91	0.46	0.18	0.46	0.23	0.09	Chicken	Egg hatchability	Lillie et al. (1975)
Aroclor 1242	0.91	0.46	0.18	0.46	0.23	0.09	Chicken	Egg hatchability	Lillie et al. (1975)
Aroclor 1248	0.91	0.46	0.18	0.46	0.23	0.09	Chicken	Egg hatchability	Lillie et al. (1975)
Aroclor 1254	0.99	0.50	0.20	0.46	0.23	0.09	Ringed turtle dove, chicken	Egg hatchability	Heinz et al. (1984); Lillie et al. (1975); Hill et al. (1976); Scott (1977)
Aroclor 1260	0.91	0.46	0.18	0.46	0.23	0.09	Chicken	Egg hatchability	Lillie et al. (1975)
Total PCBs	0.91	0.46	0.18	0.46	0.23	0.09	Chicken	Egg hatchability	Lillie et al. (1975)

Table 2-3. TRVs for Avian Receptors (continued)

Analyte	Literature LOAEL (mg/kg/d)	Heron LOAEL[a] (mg/kg/d)	Eagle, Sandpiper LOAEL[b] (mg/kg/d)	Literature NOAEL (mg/kg/d)	Heron Level NOAEL[a] (mg/kg/d)	Eagle, Sandpiper NOAEL[b] (mg/kg/d)	Test Organism	Effect	References
Organics									
1,4-Dichlorobenzene	600	300	120	40	20	8.0	Rat, mouse	Liver degeneration, decreased white blood cell count (mouse), no effects on liver or immune system (rat)	ATSDR (1991); Gaines and Linder (1986); NTP (1987); Carlson and Tardiff (1976).
4-Methylphenol	22.6[c]	11.3	4.5	9.42[c]	4.71	1.88	Red-winged blackbird	Mortality	RTECS (1995); Schaeffer et al. (1983)
Benzo(a)anthracene	10	5	2	1[d]	0.5	0.2	Mouse	Reproductive effects	MacKenzie & Angevine (1981)
Benzo(b)fluoranthene	10	5	2	1[d]	0.5	0.2	Mouse	Reproductive effects	MacKenzie & Angevine (1981)
Benzo(k)fluoranthene	10	5	2	1[d]	0.5	0.2	Mouse	Reproductive effects	MacKenzie & Angevine (1981)
Benzo(a)pyrene [a, c]	10	5	2	1[d]	0.5	0.2	Mouse	Reproductive effects	MacKenzie & Angevine (1981)
Benzo(e)pyrene	10	5	2	1[d]	0.5	0.2	Mouse	Reproductive effects	MacKenzie & Angevine (1981)
Benzo(g,h,i)perylene	10	5	2	1[d]	0.5	0.2	Mouse	Reproductive effects	MacKenzie & Angevine (1981)

Table 2-3. TRVs for Avian Receptors (continued)

Analyte	Literature LOAEL (mg/kg/d)	Heron LOAEL[a] (mg/kg/d)	Eagle, Sandpiper LOAEL[b] (mg/kg/d)	Literature NOAEL (mg/kg/d)	Heron Level NOAEL[a] (mg/kg/d)	Eagle, Sandpiper NOAEL[b] (mg/kg/d)	Test Organism	Effect	References
Bis(2-Ethylhexyl) phthalate	N/AV[e]	N/AV[e]	N/AV[e]	1.11	0.56	0.22	Ringed dove	Reproductive effects	ORNRL (1996); Peakall (1974)
Chrysene	10	5	2	1[d]	0.5	0.2	Mouse	Reproductive effects	MacKenzie & Angevine (1981)
Dibenzo(a,h) anthracene	10	5	2	1[d]	0.5	0.2	Mouse	Reproductive effects	MacKenzie & Angevine (1981)
Fluoranthene	250	125	50	125	63	25	Mallard	Reproductive effects	HEAST (1995)
Indeno (1,2,3-cd) pyrene	10	5	2	1[d]	0.5	0.2	Mouse	Reproductive effects	MacKenzie & Angevine (1981)
Phenanthrene	10	5	2	1[d]	0.5	0.2	Mouse	Reproductive effects	MacKenzie & Angevine (1981)
Pyrene	125	63	25	75	38	15	Mallard	Reproductive effects	HEAST (1995)

[a] The population level NOAEL or LOAEL is based on the NOAEL or LOAEL divided by an uncertainty factor of 2 to account for interspecies variability.

[b] The individual level NOAEL or LOAEL is based on the NOAEL or LOAEL divided by an uncertainty factor of 5 to account for potentially more sensitive endpoints such as systemic effects of growth.

[c] The LOAEL and NOAEL are based on an uncertainty factor of 5 and 12, respectively, for the ratio of acute and chronic effect doses for 3-methylphenol in rats (it was assumed the ration is the same for birds).

[d] The NOAEL was estimated from the LOAEL using an uncertainty factor of 10.

[e] N/AV = Not Available

3. WILDLIFE EXPOSURE ASSESSMENT

The four wildlife receptors being evaluated in the Water Quality Assessment are exposed to constituents of potential concern (COPCs) through water, sediment, and food. A combination of receptor-specific activity patterns, ingestion rates, and body weights determines the cumulative dose received from these media. This section presents the methods and receptor-specific data used to determine the wildlife COPC exposure concentrations. An initial step in determining wildlife exposures involves identifying where and when the receptors are present in the study area. The prey requirements of wildlife in these areas are also discussed here, as well as the specific prey items used to determine exposure in the wildlife risk assessment. Last, the equation and approaches used to quantify exposures are presented.

3.1 Spatial and Temporal Use of Study Area by Wildlife Receptors

The preferred aquatic habitat of each wildlife receptor is defined as a "patch." The patches correspond to a group of cells in the model grid overlay of the Water Quality Assessment study area. These receptor-specific cell patches determine chemical exposure for each receptor as only the concentrations from these cells were used to calculate receptor exposure levels. The following section discusses the biology of each receptor and how this was used to define the receptor-specific patches.

3.1.1 Great Blue Heron (*Ardea herodias*)

The great blue heron is a year-round fish-eating resident of the study area. They are often seen wading and feeding in or near eelgrass in Elliott Bay but can be found in any intertidal habitat in the Duwamish River. Kellogg Island is a particularly important habitat for great blue heron. They were the most numerous of shore/wading birds recorded by Cordell et al. (1996) on the Duwamish River over the period of June to September 1995.

Heron colonies (rookeries) are usually located close to their primary feeding areas. In the study area, a heron colony (rookery) is located in nearby West Seattle a few hundred meters west of Kellogg Island. This site is used by up to 40 birds (Norman 1995). Another rookery in Renton, 12 km distant, contains 28 nests and may contribute birds to the study area. On lakes in Minnesota, the distance between rookeries and feeding areas ranged between zero and 4.2 km, averaging 1.8 km (Mathisen and Richards 1978). Parnell and Soots (1978) found that rookeries in North Carolina were located an average of 7 to 8 km from feeding grounds.

While three to seven eggs are laid over a period from early March to May, seldom more than two chicks fledge (Norman 1995). In late summer after fledging, the juveniles disperse widely and do not return to their natal area until adulthood (Butler 1995). They exploit any small body of water where fish are abundant but tend to spend their winters in

upland areas feeding on invertebrates and mice (*Microtis* sp.). Butler (1991) suggests that this is because they can't meet their food requirements in coastal estuaries in fall and winter. Birds that are observed within the study area tend to be adults. Consequently, a heron patch has been defined for the period of adult feeding during the fledging period and a second patch for the remainder of the year.

Shiner perch (*Cymatogaster aggregata*) is a major food source of the chick and female herons (Butler 1993). Adult shiner perch are particularly abundant in the Duwamish River in May and June during the spawning season (Matsuda et al. 1968). Juvenile shiner perch are more abundant in the river the other months of the year. Great blue herons eat fish up to 20 to 25 cm in length (Kirkpatrick 1940; Hoffman 1978). Adult herons provide the same food to their nestlings as they consume, although partially digested (Kushlan 1978).

The patches where exposure to great blue herons will be calculated include the surface areas of most shorelines in the study area. Year-round exposure could occur over essentially the entire length of the Duwamish River from Harbor Island to the Turning Basin. In Elliott Bay, exposure areas include the West Seattle shoreline between Seacrest and Duwamish Head, and the intertidal habitat near Myrtle Edwards Park, Elliott Bay Pier, and Smith Cove. During the nesting season (March through July), exposure for the birds nesting at the rookery near Kellogg Island will occur in grid cells close to the rookery, generally no more than a mile either north or south of the rookery along the Duwamish River. Model cells that make up the heron patches are presented in Table 3-1.

3.1.2 Bald Eagle (*Haliaeetus leucocephalus*)

The bald eagle is primarily a carrion feeder (dead and dying fish) but also will catch live fish (Brown and Amadon 1968). Spawned-out salmon are a particularly important food item in the Pacific Northwest. Shiner perch (*Cymatogaster aggregate*) are a major prey item for bald eagles in the study area. In eating carrion, they may ingest small amounts of sediment. Although eagles feed mainly on fish, waterfowl make up a significant portion of their food during winter months. Eagles have been observed to kill Western grebe in the Duwamish River in winter (J. Strand, Department of Natural Resources, King County, personal communication). Eagles also have been reported to prey on great blue heron chicks (Norman et al. 1989).

Resident birds are found in the study area in the summer but this may be limited to two or three pair. The closest active eagle nest is located in West Seattle, only a few hundred meters from water and in our study area (K. Stenberg, Department of Natural Resources, King County, personal communication). Other nests in Seattle are located in Discovery Park and Lincoln Park (Hadley 1998). Migrant (wintering) birds are routinely observed in the study area beginning in October. They migrate north in late March. Because eagles forage over a large area (U.S. EPA 1993), we have assumed that the surface layer of the entire study is within their home range. The patch size for which exposure to bald eagle will be calculated is the entire study area. Depth of each model cell is restricted to the uppermost layer.

3.1.3 Spotted Sandpiper (*Actitis macularia*)

Spotted sandpipers have been observed within the study area from late June through September (Cordell et al. 1996) but also are known to winter in protected embayments of Puget Sound (Paulson 1993). Over the period June through September 1995, Cordell et al. (1996) observed spotted sandpiper routinely on intertidal habitat exposed at the Turning Basin, on Kellogg Island, and at Terminal 105.

Table 3-1. Model Cells Found in Each Wildlife Patch

Heron, August—April	1, 2, 3, 44, 46, 48, 49, 51, 52, 54, 55, 57, 58, 60, 61, 63, 64, 66, 67, 69, 70, 72, 73, 75, 76, 78, 79, 81, 82, 84, 85, 87, 88, 90, 91, 93, 94, 96, 97, 99, 100, 102, 103, 105, 106, 108, 109, 111, 112, 114, 115, 117, 118, 119, 120, 123, 123, 124, 125, 129, 130, 131, 132, 136, 137, 140, 141, 144, 145, 146, 166, 175, 188, 204, 220, 237, 254, 270, 285, 299, 312, 313, 326, 327, 340, 355, 389, 407, 408
Heron, May—July	100, 102, 103, 105, 106, 108, 109, 111, 112, 113, 114, 117, 118, 119, 120, 123, 124, 125, 129, 130, 131, 136, 137, 140, 141, 144, 145, 146, 147
Bald Eagle	1, 2, 3, 44, 45, 46, 47, 48, 49, 50, 51, 52, 53, 54, 55, 56, 57, 58, 59, 60, 61, 62, 63, 64, 65, 66, 67, 68, 69, 70, 71, 72, 73, 74, 75, 76, 77, 78, 79, 80, 81, 82, 83, 84, 85, 86, 87, 88, 89, 90, 91, 92, 93, 94, 95, 96, 97, 98, 99, 100, 101, 102, 103, 104, 105, 106, 107, 108, 109, 110, 111, 112, 113, 114, 115, 116, 117, 118, 119, 120, 121, 122, 123, 124, 125, 126, 127, 128, 129, 130, 131, 132, 133, 134, 135, 136, 137, 138, 139, 140, 141, 142, 143, 144, 145, 146, 147, 148, 149, 150, 151, 152, 153, 154, 155, 156, 157, 158, 159, 160, 161, 162, 163, 164, 165, 166, 167, 168, 169, 170, 171, 171, 172, 172, 173, 173, 174, 174, 175, 176, 177, 178, 179, 180, 181, 182, 183, 184, 185, 186, 187, 188, 189, 190, 191, 192, 193, 194, 195, 196, 197, 198, 199, 200, 201, 202, 203, 204, 205, 206, 207, 208, 209, 210, 211, 212, 213, 214, 215, 216, 217, 218, 219, 220, 221, 222, 223, 224, 225, 226, 227, 228, 229, 230, 231, 232, 233, 234, 235, 236, 237, 238, 239, 240, 241, 242, 243, 244, 245, 246, 247, 248, 249, 250, 251, 252, 253, 254, 255, 256, 257, 258, 259, 260, 261, 262, 263, 264, 265, 266, 267, 268, 269, 270, 271, 272, 273, 274, 275, 276, 277, 278, 279, 280, 281, 282, 283, 284, 285, 286, 287, 288, 289, 290, 291, 292, 293, 294, 295, 296, 297, 298, 299, 300, 301, 302, 303, 304, 305, 306, 307, 308, 309, 310, 311, 312, 313, 314, 315, 316, 317, 318, 319, 320, 321, 322, 323, 324, 325, 326, 327, 328, 329, 330, 331, 332, 333, 334, 335, 336, 337, 338, 339, 340, 342, 343, 344, 345, 346, 347, 348, 349, 350, 351, 352, 353, 354, 355, 360, 361, 362, 363, 364, 365, 366, 367, 368, 369, 370, 371, 380, 381, 382, 383, 384, 385, 386, 387, 388, 389, 402, 403, 404, 405, 406, 407, 408
Spotted Sandpiper	1, 2, 3, 46, 51, 54, 66, 69, 75, 112, 118, 124
River Otter	1, 2, 3, 44, 46, 48, 49, 51, 52, 54, 55, 57, 58, 60, 61, 63, 64, 66, 67, 69, 70, 72, 73, 75, 76, 78, 79, 81, 82, 84, 85, 87, 88, 90, 91, 93, 94, 96, 97, 99, 100, 102, 103, 105, 106, 108, 109, 111, 112, 113, 114, 115, 118, 119, 120, 123, 124, 125, 126, 129, 130, 131, 136, 137, 140, 141, 144, 145, 147, 148, 149, 150, 151, 152, 153, 154, 155, 156, 157, 158, 159, 160, 161, 162, 163, 164, 165, 166, 167, 168, 169, 170, 171, 171, 172, 173, 174, 175, 182, 183, 184, 185, 186, 187, 188, 199, 200, 201, 203, 204, 219, 220, 235, 236, 237, 253, 254, 285, 299, 312, 313, 326, 327, 327, 340, 355, 371, 389, 408

Exposure to COPCs in the study area occurs primarily through feeding activities. Spotted sandpiper feed on invertebrates (e.g., amphipods, polychaetes) by probing and picking the intertidal sediments. Leon (1980), Parametrix (1990), and Cordell et al. (1996) determined that *Corophium* was one of the most abundant amphipods on Kellogg Island mudflats. Breeding in the study area may occur but has not been documented.

The most important habitats (patches) for estimating exposure to spotted sandpiper include intertidal mudflats and beaches in the Duwamish River and Elliott Bay. In the Duwamish River, patches containing these habitats occur on either side of the river at the Turning Basin, on the east side of the river immediately above Slip #4, on Kellogg Island, and on the west side of the river adjacent to Kellogg Island. In Elliott Bay, these patches occur from Seacrest north to Duwamish Head, at Myrtle Edwards Park and the Elliott Bay Pier, and near Smith Cove. Therefore, the sandpiper patch was defined as the surface layer of the model cells corresponding to these areas (see Table 3-1).

3.1.4 River Otter (*Lutra canadensis*)

From largely anecdotal information, it is known that a family of river otters lives year-round on Kellogg Island. Otters also have been anecdotally observed in Elliott Bay at Myrtle Edwards Park and near Duwamish Head. River otters in Puget Sound feed largely on fish but also will feed on crabs and sometimes mussels and clams (J. Strand, King Department of Natural Resources, King County, personal communication). They are more likely to eat non-game than game species. In eating invertebrates, they may ingest sediment and other material. Food requirements for otters in captivity have been estimated at 700 to 900 grams of food daily (Harris 1968). In Oregon, they have been reported to eat adult coho salmon during the period of salmon spawning (Toweill 1974). Waterfowl, gulls, and rails, particularly eggs and nestlings, comprise a significant part of their diet in Pacific coast states (Toweill 1974; Grenfell 1974; Hayward et al. 1975; Verbeek and Morgan 1978).

Little is known about size of the otter's home range. It is likely dependent on habitat and the availability of food and dens. On rivers or streams, their home range may be a long strip along each shoreline. In a wetland or area with many small streams, the home range may resemble a polygon. In Sweden, the home range for a female and young was an area 7 km in diameter (Erlinge 1967). The home range for an adult male was 15 km in width with a highly variable length. Male otters also were found to forage 9 to 10 km a night and up to 16 km have been recorded. Because otters have a relatively large home range, their patch size includes all shoreline cells in the study area and to all depths (see Table 3-1).

3.2 Wildlife Exposure Assumptions

Potential risks to wildlife receptors were estimated by comparing the average daily chemical dose to wildlife chronic TRVs. To estimate the average daily chemical dose the receptors may receive from each exposure pathways, information on food, water, and sediment ingestion rates was collected for each wildlife receptor, as well as body weights.

The food, water, and sediment ingestion rates used in this risk assessment were all a function of the receptors' body weights. Because potential risks were evaluated probabilistically, mean body weights and associated standard deviations or standard errors were identified from the literature. This is described below in more detail by receptor.

3.2.1 Great Blue Heron (*Ardea herodias*)

In contrast with the bald eagle and spotted sandpiper, potential risks were assessed to the great blue herons' entire population. This difference requires the use of the standard error to estimate the normal body weight distribution of herons, rather than the standard deviation as used for eagles and sandpipers. (The standard error is a measure of the uncertainty in the mean body weight, while the standard deviation is a measure of the uncertainty in body weights in a population of individuals.) Great blue heron body weights were identified in the U.S. EPA's Wildlife Exposure Factors Handbook (1993). Because adult males appear to be slightly larger than adult females, body weight distributions were identified for both sexes. The body weights and standard errors are shown in Table 3-2.

Table 3-2. Great Blue Heron Body Weight (kg) Summary Statistics

Sex	Mean	SD	SE	n	Reference
Male	2.576	0.299	0.0725	17	U.S. EPA (1993)
Female	2.204	0.337	0.0870	15	U.S. EPA (1993)

The food ingestion rates of male and female great blue herons were estimated using an allometric equation[2] (U.S. EPA 1993):

$$IR_{food} = 10^{0.966 \times \log(BW) - 0.640} \times 0.001\,kg/g \qquad \text{(Equation 3-1)}$$

Where:

IR_{food} = Food ingestion rate (kg/day-wet)
BW = Body weight (g)

No empirical water ingestion rate data were identified for great blue heron so an allometric equation based on body weight was used (U.S. EPA 1993). This equation is:

[2] An allometric equation expresses a particular animal attribute (in this case drinking water) as a function of another attribute (e.g. body weight).

$$IR_{water} = 0.059 \times BW^{0.67} \qquad \text{(Equation 3-2)}$$

Where:

IR_{water} = Water ingestion rate (L/day)

BW = Body weight (kg)

No data on sediment ingestion rates of great blue herons were found in the literature, but sediment ingestion rates are likely to be low based on their foraging behavior. The U.S. EPA (1993) states that when fishing, great blue herons use two fishing techniques: standing still and waiting for fish to swim within striking distance, or slow wading to catch more sedentary prey. Therefore, to fish they require shallow water with a firm substrate, and larger prey are usually immersed in water before they are swallowed. All of these factors support the assumption of a low sediment ingestion rate; in this risk assessment, it was assumed sediment ingestion was equal to 2 percent of their dietary intake.

3.2.2 Bald Eagle (*Haliaeetus leucocephalus*)

Body weights of adult bald eagles were identified in the literature (Dunning 1993), and assumed to represent the body weights of eagles in the study area. To account for the potential range of individual eagle body weights that may exist, it was assumed that the distribution of individual eagle body weights is normally distributed. Because female eagles tend to be larger than male eagles, body weight distributions were identified for each sex. Mean eagle body weights were available for both females and males, but the standard deviations on these means were not available (Table 3-3). Therefore, the standard deviations were estimated based on the range of body weights that contributed to each mean. Because the body weight sample sizes were fairly large (n = 35 and 37 for males and females, respectively), it was assumed the range represented 99 percent of adult eagle body weights, or approximately plus or minus three standard deviations about the mean[3].

Chemical doses to bald eagles were estimated assuming that there were an equal number of males and females at the site. Accordingly, the male- and female-specific exposure parameters (e.g., body weight) were given equal weight in deriving the chemical dose distribution.

According to independent studies reported in U.S. EPA (1993) and Stalmaster (1987), the daily food ingestion rate of adult eagles is equivalent to approximately 12 percent of body

[3] Ninety-nine percent of the measurements in a normal population lie with the mean plus or minus 2.6 standard deviations (Zar 1984).

weight on a wet weight basis. The water ingestion rate for the bald eagle was calculated using the same allometric equation as for the great blue heron.

Table 3-3. Bald Eagle Body Weight (kg) Summary Statistics

Sex	Mean	SD[a]	Range	n	Reference
Male	4.13	0.197	3.637-4.819	35	Dunning (1993); U.S. EPA (1993)
Female	5.35	0.462	3.631-6.4	37	Dunning (1993); U.S. EPA (1993)
Male	4.325	NA	NA	52	Stalmaster (1987)
Female	5.268	NA	NA	54	Stalmaster (1987)

[a] Standard deviation estimated as 1/6 the range of body weights

No data were available on eagle sediment ingestion rates, although it is likely they will ingest some sediment when scavenging along shorelines. In this assessment, it was assumed that the sediment ingestion rate is equal to 1 percent of the eagle food diet.

3.2.3 Spotted Sandpiper (*Actitis macularia*)

Body weights of male and female spotted sandpipers were taken from the literature, and were different enough to preclude combining them into one distribution. The standard deviations on the mean body weights identified were not available, so they were assumed based on the range of body weights reported. Due to small sample sizes (n = eight and nine for males and females, respectively), it was assumed the body weight ranges captured 95 percent of the potential adult spotted sandpiper body weights, or approximately plus or minus two standard deviations about the mean[4]. The body weights and standard deviations are shown in Table 3-4.

Table 3-4. Spotted Sandpiper Body Weight (kg) Summary Statistics

Sex	Mean	SD[a]	Range	n	Reference
Male	0.0379	0.0018	0.034-0.041	8	U.S. EPA (1993); Maxson and Oring (1980)
Female	0.0471	0.0018	0.043-0.050	9	U.S. EPA (1993); Maxson and Oring (1980)

[a] Standard deviation estimated as 1/4 the range of body weights

[4] Ninety-five percent of the measurements in a normal population lie with the mean plus or minus 2.0 standard deviations (Zar 1984).

The sandpiper food ingestion rate was estimated using an allometric equation dependent on body weight (U.S. EPA 1993). The dry weight ingestion rates calculated by this equation were converted to wet weights to ensure conformity with other data used in estimating spotted sandpiper risks. The wet weight ingestion rate was estimated based on 80 percent moisture in sandpiper food items (20 percent solids based on data reported by Meador [1997]). The allometric equation used was:

$$IR_{food} = \left(0.0582 \times BW^{0.651}\right) \times \frac{1\,\text{kg wet matter}}{0.2\,\text{kg dry matter}} \qquad \text{(Equation 3-3)}$$

Where:

IR_{food}	=	Food ingestion rate (kg/day-wet)
BW	=	Body weight (kg)

The spotted sandpiper water ingestion rate was estimated using the same equation for the bald eagle shown above. Due to their probing feeding habits, spotted sandpipers were assumed to have a significant sediment ingestion rate. While spotted sandpiper sediment ingestion rates were unavailable, they were available for the semipalmated, western, stilt, and least sandpipers (U.S. EPA 1993). Sediment ingestion rates for these four sandpipers, estimated as the percent soil in diet on a dry weight basis, averaged 18 percent. Sediments were assumed to be 50 percent moisture based on data collected by King County from the Duwamish River (S. Michelson, King County Environmental Laboratory, personal communication.)

3.2.4 River Otter (*Lutra canadensis*)

Potential risks to the river otter population were assessed. Body weights were identified in the U.S. EPA's Exposure Factors Handbook (U.S. EPA 1993). As for the other wildlife receptors, separate distributions were fit to male and female body weights due to apparent differences in their weights. The mean body weights and standard errors assumed in this risk assessment are shown in Table 3-5.

Table 3-5. River Otter Body Weight (kg) Summary Statistics

Sex	Mean	SE	n	Reference
Male	9.2	0.6	4	U.S. EPA (1993); Melquist and Hornocker (1983)
Female	7.9	0.2	6	U.S. EPA (1993); Melquist and Hornocker (1983)

Using the model of Iversen (1972), as cited in U.S. EPA (1993), the river otter food ingestion rate was estimated as a function of body weight, mean caloric content of prey (k_{prey}) (kcal/g), and the ratio (r_{met}) of free living to basal metabolism rates (BMR):

$$IR_{prey} = \frac{(BMR \times r_{met})}{(k_{prey} \times 1000)} \quad \text{(Equation 3-4)}$$

Where: BMR (kcal/day) is given by the equation:

$$BMR = 84.6 \times BW^{0.78} \ (\pm 15\%) \quad \text{(Equation 3-5)}$$

$$BMR = \text{Uniform}[0.85 \times 84.6 \times BW^{0.78}, 1.15 \times 84.6 \times BW^{0.78}] \quad \text{(Equation 3-6)}$$

The term "Uniform" in the preceding equation is a mathematical statement used to generate a uniform probability distribution using these data inputs.

The value of (r_{met}) was assumed to be between three and five, based on U.S. EPA (1993):

$$r_{met} = \text{Uniform}(3,5) \quad \text{(Equation 3-7)}$$

The mean caloric content of prey was estimated by a normal probability distribution function (PDF) with a mean and standard error from Table 4-1 of U.S. EPA (1993):

$$k_{prey} = \text{Normal}\left(1.2, \frac{0.24}{\sqrt{18}}\right) \quad \text{(Equation 3-8)}$$

The term "Normal" in the preceding equation indicates a mathematical statement used to generate a normal probability distribution using this mean and standard error.

No data were available on the water ingestion rate of otters, so an allometric equation was used (U.S. EPA 1993):

$$IR_{water} = 0.099 \times BW^{0.90} \quad \text{(Equation 3-9)}$$

Where:

IR_{water}	=	Water ingestion rate (L/day)
BW	=	Body weight (kg)

As data were not available on the sediment ingestion rate of river otters, it was assumed that they had a sediment ingestion rate of 2 percent.

3.3 Sources of Environmental Concentration Data

Exposures were estimated for the wildlife receptors for the baseline, without CSOs, and reference scenarios based on water and sediment concentrations predicted in their study area patches as well as specific tissue types believed to be potential prey species. Estimated exposure concentrations for water and sediment in each receptor patch are summarized in Tables 3-6 to 3-10. Fish and invertebrate tissue concentrations were measured analytically by the King County Environmental Laboratory from samples

collected from the study area by the County, working with the Washington State Department of Fish and Wildlife. The estimated exposure concentrations (EECs) in prey were estimated from those measured tissue concentrations (Tables 3-11 to 3-16). The EECs were estimated based on composite samples taken from multiple animals.

Compositing was necessary to obtain a sufficient volume of tissue for planned chemical analyses. A minimum of 130 grams was required for analyses of metals, organics including PAHs and PCBs, the organometallics (i.e., TBT), and conventionals including lipids and percent moisture. Individual specimens of most of the targeted species weighed considerably less than 130 grams. The number of composite samples and number of animals per composite are given below in Table 3-16. An assumed normal distribution of the mean EECs was developed based on the arithmetic mean and standard error of the measured concentrations in tissue. The methods used to develop distributions for exposure parameters are presented below.

Table 3-16. Tissue Samples Used to Estimate Wildlife Exposure Concentrations (EECs)

Tissue Type	Number of Organisms Per Composite	Location	Number of Composite Samples	Tissue Analyzed
Shiner Perch *(Cymatogaster aggregate)*	10	Duwamish River Elliott Bay Port Susan	3 3 3	Whole body
Intertidal Amphipods (*Traskorchestia traskiana)*	2,000 approx.	Duwamish River Nisqually Delta	2 2	Whole body
Dungeness Crab *(Cancer magister)*	3	Duwamish River Elliott Bay Port Susan	2 4 3	Edible muscle & hepato-pancreas
Mussel *(Mytilus trossulus)*	50	Duwamish River Elliott Bay Totten Inlet	23 3 13	Soft parts
chinook Salmon (*Oncorhynchus tshawytscha)*	N/AV	Duwamish River	N/AV	Muscle
coho Salmon (*Oncorhynchus kisutch)*	N/AV	Duwamish River	N/AV	Muscle

N/AV = Not available

3.3.1 Great Blue Heron Prey Species

Shiner perch *(Cymatogaster aggregate)* are a major food source for great blue herons in the study area, and are important in juvenile survival. Therefore, tissue concentrations in shiner perch were used to estimate exposures to heron by the food consumption pathway.

Shiner perch collected from the Port Susan area were used to estimate reference shiner perch tissue concentrations.

3.3.2 Spotted Sandpiper Prey Species

As described above, the spotted sandpiper feeds on invertebrates, including amphipods and polychaetes in intertidal sediments. Therefore, amphipods (*Traskorchestia traskiana)* in the study area were assumed to be prey species for the spotted sandpiper. Tissue concentrations of amphipods collected from the study area were used to estimate exposures to the spotted sandpiper. McCallister Creek in the Nisqually Delta was used as a source of reference amphipod tissue concentrations.

3.3.3 Bald Eagle Prey Species

Shiner perch *(Cymatogaster aggregate)* and several salmon species are major food sources for bald eagles in the study area. Therefore, tissue concentrations in shiner perch, individual coho and chinook salmon, and combined salmon were used to estimate exposures to bald eagles by the food consumption pathway. Shiner perch collected from the Port Susan area were used to estimate reference shiner perch tissue concentrations.

3.3.4 River Otter Prey Species

River otters feed on a combination of fishes and aquatic invertebrates found in the study area. Therefore, shiner perch, crabs, and mussels were all assumed to be prey species for the river otter. Tissue concentrations of each of these species collected from the study area were used to estimate exposures to the river otters. Crab tissues were separated into edible muscle and hepatopancreas for this analysis.

3.4 Methods Used to Calculate Exposures to Wildlife Receptors

Exposures for all wildlife receptors were derived from ingestion of food, surface water and sediment. The equations used to estimate these exposures are presented below. The intake (dose) represents the amount of chemical ingested, and it is expressed as the estimated environmental dose or EED. Chronic exposures were evaluated for all receptors. Additionally, it was assumed that all chemicals were equally bioavailable in the field as in those toxicity tests used to establish effects thresholds (TRVs). This a typical assumption in wildlife risk assessments (U.S. EPA 1994). As discussed earlier, body weights and food, water, and sediment ingestion rates can differ for males and females. Therefore, EEDs were calculated assuming the receptor populations in the Duwamish River and Elliott Bay are 50 percent male and 50 percent female.

The drinking water ingestion EEDs were computed as shown in Equation 3-10:

$$EED_{water} = EEC_{water} \times \left(\frac{0.5 \times WIR_m}{BW_m} + \frac{0.5 \times WIR_f}{BW_f} \right) \quad \textbf{(}\text{Equation 3-10}\textbf{)}$$

Where:

EED_{water}	=	Water dose to wildlife species of interest (mg/kg BW/day)
EEC_{water}	=	Estimated exposure concentration (mg/L)
WIR	=	Water ingestion rate (L/day)
BW	=	Body weight of receptor (kg)
M	=	Male
F	=	Female

The food ingestion EEDs were estimated using chemical concentrations in their food and prey consumption rate (e.g., fish) for each of the receptor species as calculated in Equation 3-11:

$$EED_{food} = EEC_{prey} \times \left(\frac{0.5 \times FIR_m}{BW_m} + \frac{0.5 \times FIR_f}{BW_f} \right) \quad \textbf{(}\text{Equation 3-11}\textbf{)}$$

Where**:**

EED_{food}	=	Food dose to wildlife species of interest (mg/kgBW/day)
EEC_{prey}	=	Expected tissue concentration in prey species tissue (mg/kg wet weight)
FIR	=	Food consumption rate (kg wet weight/day)
BW	=	Body weight of receptor (kg)
m	=	Male
f	=	Female

Similarly, sediment ingestion EEDs were calculated for each of the wildlife receptors. The daily EED from the incidental ingestion of sediment is calculated as shown in Equation 3-12:

$$EED_{sediment} = EEC_{sediment} \times \left(\frac{0.5 \times SIR_m}{BW_m} + \frac{0.5 \times SIR_f}{BW_f} \right) \quad (\text{Equation 3-12})$$

Where:

$EED_{sediment}$	=	Sediment dose to wildlife species of interest (mg/kgBW/day)
$EEC_{sediment}$	=	Expected environmental concentration in sediment (mg/kg wet weight)
SIR	=	Sediment ingestion rate (kg wet weight/day)
BW	=	Body weight of receptor (kg)
M	=	Male
F	=	Female

The total dose to wildlife receptors is estimated by combining the food, water and sediment ingestion EEDs described above. Risks are calculated based on total chemical exposure from each of these sources. The equation to estimate the expected environment dose (EED) to each of the wildlife receptors is described by Equation 3-13:

$$EED_{total} = + EED_{water} + EED_{food} + EED_{sediment} \quad \text{(Equation 3-13)}$$

Where:

EED_{total}	=	Total expected environmental dose to wildlife receptor (mg/kgBW/day)
$EED_{sediment}$	=	Sediment dose to wildlife species of interest (mg/kg BW/day)
EED_{water}	=	Water dose to wildlife species of interest (mg/kg BW/day)
EED_{food}	=	Food dose to wildlife species of interest (mg/kg BW/day)

3.5 Exposure Assessment Uncertainty

Several assumptions introduce uncertainties into the exposure assessment. These have to do with chemical concentrations in the water, sediments and food to which wildlife receptors are exposed, and with the characteristics and behaviors of the wildlife receptors that affect the magnitude of their exposures. Many of the uncertainties were treated probabilistically, so they are accounted for in the exposure assessment results. Those that were not treated probabilistically were generally small compared to the uncertainties that were treated probabilistically, so they have little influence on exposure estimates. The specific probability distributions we used to characterize uncertainty about the characteristics and behaviors of the wildlife receptors were presented in the Section 3.2 Wildlife Exposure Assumptions. The specific probability distributions we used to characterize uncertainty in the water, sediment, and tissue EECs for wildlife were presented in Tables 3-6 through 3-11. Uncertainty about dietary composition was evaluated through sensitivity analysis. Specifically, we estimated risk separately for each prey species collected (i.e., assuming each prey species in turn comprised 100 percent of the receptor's diet). Bald eagle exposure estimates were based on shiner perch and (adult) salmon data. We did not have waterfowl tissue data, though waterfowl may be part of the bald eagle's diet. If waterfowl tissue concentrations are higher than shiner perch and salmon tissue concentrations, our use of the fish data to estimate the bald eagle's exposure would introduce an underestimation bias. Conversely, if waterfowl tissue concentrations are lower than the shiner perch and salmon tissue concentrations, our exposure estimates are biased to overestimate the bald eagle's risk.

4. WILDLIFE RISK CHARACTERIZATION METHODS

Potential risks to river otters, great blue herons, spotted sandpipers, and bald eagles from COPCs in surface water, sediment, and prey items were estimated using the HQ (hazard quotient) approach, where:

$$\text{Hazard Quotient} = \frac{\text{Expected Environmental Dose}}{\text{Toxicity Reference Value}} \qquad \text{(Equation 4-1)}$$

The expected environmental dose was defined above as the chemical dose received from water ingestion, sediment ingestion, and food ingestion (water, sediment, and food). HQs were determined for each exposure pathway separately, and then summed to determine the HQ for all exposure pathways combined. HQs were determined by exposure pathway to identify which pathway contributed most to the total risk for each species.

HQs were calculated probabilistically to quantitatively assess uncertainty in the dose estimates (due to natural variability and lack of site-specific information on receptor body weights and ingestion rates) and in the TRVs (because the threshold for effects is uncertain. As identified above (Section 3.2), receptor body weights (and ingestion rates as a function of body weight) were assumed to be normally distributed. In addition, variability in prey tissue concentrations was addressed assuming that variability in the estimates of the mean tissue concentrations was also normally distributed. Lastly, it was assumed the true TRVs (or thresholds for toxic effects) were uniformly distributed between the NOAEL and LOAEL, meaning that it was equally chance that any value between the NOAEL and LOAEL could be the true toxicity reference value. These distributions of body weights, ingestion rates, prey tissue concentrations, and TRVs were then randomly sampled in calculating the HQ using a Monte Carlo analysis.

Monte Carlo analysis involves running a model (e.g., the HQ model shown in Equation 4-1 above) and repeatedly performing the calculation using randomly selected sets of input values each time. While the input values are randomly selected, the selection of values is a function of their probability of occurrence. Using the Microsoft Excel-compatible computer program @Risk (Palisade Corporation 1996), approximately 2,000 to 3,000 sampling iterations were conducted for each wildlife receptor and scenario. The number of sampling iterations varied because an auto-stop feature was used which discontinues the sampling once the output distributions converge (i.e., once the sample percentiles, mean, and standard deviation all change by less than 1.5 percent over two consecutive sampling intervals of 100 iterations).

5. WILDLIFE RESULTS

This section summarizes the results of the wildlife risk assessment. As described above, potential risks to wildlife receptors from chemical stressors were estimated assuming current baseline conditions and if CSOs were removed from the Duwamish River and Elliott Bay. Additionally, potential risks to wildlife at reference sites in Puget Sound were estimated for comparison to the risk estimates for the Duwamish River and Elliott Bay.

Potential risks (estimated using HQs) to receptors were evaluated probabilistically, meaning distributions of HQs were calculated. Accordingly, HQs will be discussed in this section as means or as different percentiles. Specifically, the percentiles given greatest attention are the 5^{th} and 95^{th} percentile HQs. These represent lower and upper bound HQs, respectively, that bracket the range of HQs that may be observed. The minimum and maximum HQs were not used as lower and upper bounds because they are at the ends of the tails of the HQ distributions and are highly unreliable. The HQ results are presented and discussed below by receptor.

5.1 Great Blue Heron

It was assumed herons are exposed to chemical stressors from ingestion of small fish (shiner perch), sediment, and water. Overall (total exposure) HQs were weighted for two different portions of the year: (1) May through July during the fledgling season when adults will feed in a more localized area of the Duwamish River and (2) the remainder of the year when adults will feed over a larger area including Elliott Bay.

5.1.1 Metals/TBT

For both baseline conditions and the without CSO scenario, none of the mean HQs for any metal or TBT over the one-year duration exceed 1.0; however, the 95^{th} percentile HQ for lead is 1.8 and 1.7 for baseline conditions and without CSOs, respectively (Table 5-1). The exposure pathway contributing the most to these HQs is sediment ingestion. The 95^{th} percentile HQs for all other metals and TBT are less than 1.0. At reference sites, all 95^{th} percentile HQs were less than 1.0 for herons (Table 5-5).

5.1.2 Organics

All heron HQs (including 95^{th} percentile) for organics are less than 1.0 under baseline conditions, without CSOs, and for reference sites (see Table 5-1 for the Duwamish River and Elliott Bay and Table 5-5 for reference sites).

5.2 Bald Eagle

Eagle prey items were considered to consist of perch and salmon (where data were available). When chemical data were available for both perch and salmon, they were assumed to contribute equally to the diet, however, HQs are also shown for individual prey items so one can see the influence each has on the overall HQ. As shown in Table 5-2, HQs were calculated for "combined salmon" (chinook plus coho), and chinook and coho individually. It is the combined salmon HQs that were used in the calculation of the overall HQ.

5.2.1 Metals/TBT

Under both baseline conditions and the without CSO scenario, no mean HQs are greater than 1.0 (see Table 5-2). The 95th percentile HQ for lead exceeds 1.0 (HQ = 2.04), with the driving exposure pathway being sediment ingestion. The 95th percentile lead HQs for eagles using reference data is less than 1.0 (Table 5-7).

5.2.2 Organics

All eagle HQs (including 95th percentile) for organics are less than 1.0 under baseline conditions, without CSOs, and for reference sites (see Table 5-2 for the Duwamish River and Elliott Bay and Table 5-6 for reference sites).

5.3 Spotted Sandpiper

The sandpiper diet was assumed to consist of sediment-dwelling invertebrates (represented by amphipods). More mean HQs exceeded 1.0 for the sandpiper than for any of the other receptors.

5.3.1 Metals/TBT

Mean HQs exceed 1.0 for copper, lead, and zinc (baseline and without CSOs) (Table 5-3). The 5th percentile, mean, and 95th percentile HQs are 16, 22, and 27 for copper; 46, 112, and 279 for lead; and 0.5, 1.4, and 2.4 for zinc. The overall HQs for copper and zinc are driven by the dietary exposure, while for lead the overall HQ is driven mostly by the diet, but sediment ingestion is contributing fairly significantly as well. HQs for copper, lead, and zinc at the reference site are also fairly high: mean HQs are 20 for copper, 60 for lead, and 4.5 for zinc (Table 5-7).

5.3.2 Organics

Mean HQs exceed 1.0 for PCBs and bis(2-ethylhexyl)phthalate (Table 5-3). The 5th percentile, mean, and 95th percentile HQs are 1.5, 2.5, and 3.7 for PCBs and 0.4, 2.3, and 4.2 for bis(2-ethylhexyl)phthalate. The overall HQs for both of these organics are driven by dietary exposure. HQs at reference sites are less than 1.0 (Table 5-3). All sandpiper

HQs (including 95th percentile) for organics are less than 1.0 under baseline conditions, without CSOs, and for reference sites (see Table 5-3 for the Duwamish River and Elliott Bay and Table 5-7 for reference sites).

5.4 River Otter

As explained above, it was assumed wildlife receptors are exposed to chemical stressors through food, water, and sediment ingestion. It was assumed the river otter feeds primarily on small fish (represented by shiner perch), crabs, and mussels. In the overall HQ calculation for otters (i.e., summing the exposure from food, water, and sediment), it was assumed otters eat equal proportions of fish, crab, and mussels. However, because HQs were calculated for each individual food type, one can see the influence of different food items on the overall HQ and infer how the overall HQ would change if different dietary fractions were assumed.

5.4.1 Metals/TBT

Under baseline conditions, the only metal with an overall mean HQ exceeding 1.0 is lead (HQ = 1.6) (Table 5-4). The 5th and 95th percentile HQs are 0.7 and 3.8, respectively. Although the 95th percentile HQ for ingestion of mussels exceeded 1.0, the HQs for the other dietary fractions are much lower, thereby diluting the overall contribution from food (because it was assumed otters feed equally on the other food items). The exposure pathway contributing most to the overall HQ is sediment ingestion (mean HQ = 1.3, 5th percentile = 0.5, 95th percentile = 3.5). The lead HQs expected with removal of CSOs are only slightly lower (see Table 5-4). At reference sites the overall 95th percentile HQ for lead is 0.5 (see Table 5-8).

The 95th percentile overall HQ for arsenic (both baseline and without CSO scenario) slightly exceeded 1.0 (HQ = 1.1). This suggests there is only slightly greater than a 5 percent probability that the arsenic HQ exceeds 1.0. In contrast to lead, the overall arsenic HQ is driven by the contribution from food, and specifically from crabs. At reference sites the overall 95th percentile HQ for arsenic 0.5 (see Table 5-4).

5.4.2 Organics

None of the HQs for organics exceed 1.0 (see Table 5-4 for the Duwamish River and Elliott Bay and Table 5-8 for reference sites). The highest mean overall HQ is 0.5 for PCBs.

5.5 Risk Characterization Uncertainty

The wildlife risk assessment found that lead in amphipods eaten by spotted sandpipers could cause exposures hundreds of times higher than the sandpiper's lead TRV. The range of uncertainty in the spotted sandpiper's lead HQ was 24 to 481, with a sample mean of 112. This uncertainty distribution accounts for uncertainty in the average concentration in the spotted sandpiper's diet and uncertainty about average body weight

and food ingestion rate. Lead hazard quotients were ten times higher in the study area than at reference sites, but still greater than one at the reference sites. Details of how uncertainties were treated in the exposure and effects characterizations were presented in those sections of this appendix.

Not all sources of uncertainty are accounted for in the analysis. Most notably, there is a model structural uncertainty that is not accounted for. Specifically, the lead TRV is based on reproductive effects, but spotted sandpipers generally are thought not to breed in the Duwamish Estuary or Puget Sound. Our exposure model does not take into account lead depuration that may occur between exposure in the Duwamish Estuary, and nesting elsewhere. As such they contain an unquantified overestimation bias. Another source of uncertainty we did not account for is uncertainty about bioavailability. We assumed bioavailability was the same in the field as in laboratory toxicity tests. This assumption probably creates a small overestimation bias in the risk characterization. Nonetheless, the range of spotted sandpiper lead HQs is sufficiently high to clearly indicate potential risks to wildlife in the Duwamish Estuary. Lead risk estimates are the same for baseline and without CSOs, because the source of the lead is historically contaminated sediments near Kellogg Island.

The same sources of uncertainty were evaluated for the other three wildlife receptors as for the sandpiper. These include uncertainty about exposure concentrations, uncertainty about body weight and food ingestion rate, and uncertainty about the TRV. These uncertainties were treated probabilistically. Uncertainty about dietary composition was also evaluated through sensitivity analysis. Specifically, we estimated risk separately for each prey species collected (i.e., assuming each prey species in turn comprised 100 percent of the receptor's diet). This allowed us to see how variability in prey species body burdens affected wildlife risk estimates, although as noted above, it was done as a sensitivity analysis. Final risk estimates were computed using an overall average prey concentration for each chemical of potential concern.

We estimated HQs greater than one for the bald eagle, great blue heron, and river otter for lead (all three receptors) and also arsenic for the river otter only. The probability of the arsenic HQ exceeding one for the river otter was less than ten percent, with an estimated minimum of 0.2 and maximum of 2.5. The results were the same for baseline and without CSOs. The lead HQs for river otter ranged from about 0.5 to 6, with about a two-thirds probability of exceeding one. The lead HQs for the great blue heron ranged from about 0.4 to 4 during fledgling season, with about a 25 percent probability of exceeding one. The lead HQs for the bald eagle ranged from about 0.3 to 3, and also had about a 25 percent probability of exceeding one. These risk estimates do not contain any intentional biases, other than safety factors on the TRVs for inter-and intra-species variability and the possibility of a more sensitive endpoint than measured (decreased litter size for arsenic and reproductive endpoints for lead). Removing these safety factors would reduce the maximum HQs below one, indicating that the presence or absence of risk to the eagle, heron and otter is uncertain.

Uncertainty about the conclusion that removing CSOs would have no discernable effect on risks to wildlife is low. We have a reasonably good understanding that sources other

than baseline CSO discharges are principally responsible for the arsenic and lead to which wildlife are exposed. Therefore, removing CSOs has little effect on risks.

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