Appendix D: Defining Injuries to Natural Resources in Hylebos Waterway

Prepared For

The Commencement Bay Natural Resource
Co-Trustees

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INTRODUCTION

The Hylebos Waterway Habitat Equivalency Analysis (HEA) evaluates and quantifies natural resource injuries in areas where surficial sediments contain Substances of Concern (SOCs) at or above concentrations defined as thresholds for natural resource injury. This report focuses on describing how injuries are defined and how increasing concentrations of SOCs cause greater injury. The report also addresses how percent service loss is used as the injury metric for natural resource damages in Hylebos Waterway.

A companion document by Cacela *et al.* (2001) thoroughly discusses the concept of percent service loss. We will only discuss that topic in this report in the context with how it supports our HEA. Most of the following will concentrate on describing what information is used to determine service loss and how increasing service losses are estimated.

It should be noted that the process and results described in this report represent a means to establish groundwork for dialog. More rigorous scientific scrutiny will likely result in injury threshold values different than those reported here. However, this document provides a useful basis for determining injury levels for discussing settlement. This report discusses the concept of percent service loss and provides plausible examples.

BRIEF DISCUSSION ABOUT ASSOCIATING ECOSYSTEM SERVICE LOSS WITH SEDIMENT CONTAMINATION

The concept of "service" is central to HEA. What is needed is a clear understanding of what "service" is being evaluated or modeled and how reductions in services are determined (Cacela *et al.* 2001). Habitats typically provide many and varied types of ecological services (Strange *et a*l, in prep), plus consumptive and nonconsumptive uses to humans, and promote ecological sustainability through complex biotic and abiotic interactions (Holmlund and Hammer, 1999). Although some human use services are easy to evaluate (e.g., recreational beach use), others are not and it is difficult to interconnect services associated with human use to services associated with ecological function. Due to this difficulty, in our HEA, we focus all evaluations of service solely on aspects of ecological function.

"Ecological service" is a term that may embrace several different structural or functional attributes of an ecosystem. Specific measures must be defined to enable quantification of this service (Cacela *et al.* 2001). In the context of HEA, ecological services are used as an "exchange rate" for determining what amount of habitat must be restored to compensate for some harm to the environment. Equivalency between injured and restored habitat is gauged through the level of service provided by the habitats. Although the injured and restored habitats may include human use services as well as ecological services, it is assumed that a focus on restoring ecological function value will result in a concomitant restoration of human use services.

It is important to consider how an ecological service is lost, how much is lost, and how that loss can be represented. For the purposes of our HEA, ecological services are lost when an organism or organisms are adversely affected by the presence of a specified concentration of a SOC. These adverse effects range from subcellular alteration to lethality, and likely represent service losses ranging from minor to major amounts. Lethality may result in a population level effect; however, quantifying population effects is very difficult. Consequently, we focus on effects to groups of organisms (a sample), individual organisms, and processes within an organism.

Organisms live on a finite energy budget. Because the energy budget is limited, any stressor that occurs in addition to "natural" (i.e., non-anthropogenic) stressors is detrimental to that organism. Organisms must redirect energy to mitigate the effects of the stressor (Rowe *et al.*, 1998). Examples of stress-induced energy redirection include physiological detoxification (e.g., metallothionein induction) and additional caloric expenditures for motility (e.g., due to habitat avoidance behaviors or enlarged home range) (Cacela *et al.* 2001). Redirecting energy for these activities comes at the expense of other biological processes, such as growth, reproduction, and/or avoiding predation. If a particular habitat is more stressful than a reference site, it provides less service, and portion or percent of service is lost. That lessened service is an injury. Because of the functional role that ecological services play in HEA, service reductions are expressed on a percentage basis. The ecological service index of a unit of habitat is reduced by a fractional amount that reflects the difference in habitat quality at the injured site relative to an uninjured reference location.

One way to evaluate service loss within a habitat is to identify the extent and types of injuries to resources utilizing that habitat. Such information is found in various scientific literature; however, the breadth of information on injuries varies greatly between chemicals. Two examples of differing levels of information and associated service loss estimates are provided for Hexachlorobenzene (HCB) and Polycyclic Aromatic Hydrocarbons (PAHs). Following the discussion of each of these SOCs is information about other SOCs that are handled similarly.

HCB INJURIES AND THE USE OF APPARENT EFFECTS THRESHOLDS

The only data sets used in this report to evaluate injuries from HCB and several other SOCs are benthic community analyses and bioassay information associated with the State of Washington's Sediment Management Standards (SMS, Chapter 173-204 WAC, revised 12/95). The State of Washington Department of Ecology maintains a database that determines Apparent Effects Thresholds[1] (AETs) associated with various invertebrate bioassays and benthic community data. The seven AETs considered in this report address five invertebrate phyla and the benthic (invertebrate) community (Table 1). These include a benthic community analysis, and bioassays for an echinoderm, Microtox™, amphipod, *Neanthes*, "bivalve", and oyster. Since the "bivalve" (includes both mussels and oysters) and oyster bioassays both represent organisms that are mollusks, the bivalve AET is used only when oyster data is not available. This decision is based on the fact that the oyster AET was promulgated in the Washington SMS rule and underwent extensive review during that process. The more recently estimated "bivalve" AET has not yet undergone a similar evaluation.

Although the SMS information is expressed in both dry weight and organic carbon (OC) normalized concentrations, this report focuses solely on the dry weight AETs. This decision is based on concern that the total carbon content of some sediment samples from Hylebos Waterway is artificially elevated from some human activities that result in deposition of organic substances (e.g., petroleum hydrocarbons, wood chips, etc). OC normalization of these carbon-enhanced sediment samples may result in inappropriately low normalized values (Michelsen, 1992).

The lowest AET establishes Marine Sediment Quality Standards (MSQS) for Puget Sound that "correspond to a sediment quality that will result in no averse effects on biological resources…".

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[1] An Apparent Effects Threshold is defined as the concentration of a single chemical (or chemical class) in sediments above which a particular biological effect has always been observed in a particular biological test.

Table 1. Apparent Effects Thresholds (AETs) for a variety of substances of concern. These AETs are derived from data used to promulgate State of Washington (Marine) Sediment Management Standards. All concentrations are expressed at dry weight values.

SUBSTANCES OF CONCERN	BIOASSAYS[1]						
	Amphipod Bioassay	***Benthic Community Analysis***	***Bivalve Bioassay***	***Echinoderm Bioassay***	***Microtox™ Bioassay***	***Neanthes Bioassay***	***Oyster Bioassay***
	[1994]	[1988]	[1994]	[1994]	[1986]	[1998]	[1986]
Metals (mg/kg or ppm)							
Antimony	200	150	5.9	9.3	na	21	na
Arsenic	450	57	35	130	700	63	700
Cadmium	14	5.1	3.6	2.7	9.6	3.0	9.6
Chromium	>1,100	260	63.5	>96	na	94	na
Copper	1,300	530	298	390	390	270	390
Lead	1,200	450	336	430	530	360	660
Mercury	2.3	2.1	1.7	1.4	0.41	1.3	0.59
Nickel	>370	>140	>82	110	na	150	na
Silver	6.1	>6.1	3.0	8.4	>0.56	3.3	>0.56
Tributyltin	>180	na	na	na	na	>460	na
Zinc	3,800	410	839	460	1,600	530	1,600
Nonionizable organic compounds (ug/kg or ppb)							
Total LPAHs	29,000	13,000	3,825	1,200	5,200	3,700	5,200
2-Methylnaphthalene	1,900	1,400	120	64	670	190	670
Acenaphthene	2,000	730	660	130	500	960	500
Acenaphthylene	1,300	1,300	150	71	>560	130	>560
Anthracene	13,000	4,400	1,500	280	960	1,700	960
Fluorene	3,600	1,000	500	120	540	410	540
Naphthalene	2,400	2,700	180	230	2,100	1,300	2,100
Phenanthrene	21,000	5,400	2,000	660	1,500	3,400	1,500
High molecular weight PAHs (ug/kg or ppb)							
Total HPAHs	69,000	69,000	13,080	7,900	12,000	8,100	17,000
Benz[a]anthracene	5,100	5,100	1,100	960	1,300	3,300	1,600
Benzo[a]pyrene	3,500	3,600	1,000	1,100	1,600	2,200	1,600
Benzo[ghi]perylene	3,200	2,600	640	920	670	1,300	720
Total benzofluoranthenes	9,100	9,900	na	1,800	3,200	>6,600	3,600
Chrysene	21,000	9,200	1,600	950	1,400	10,000	2,800
Dibenzo[a,h]anthracene	1,900	970	250	240	230	na	230
Fluoranthene	30,000	24,000	3,300	1,300	1,700	10,000	2,500
Indeno[1,2,3-cd]pyrene	4,400	2,600	590	760	600	1,300	690
Pyrene	16,000	16,000	4,100	2,400	2,600	>9,600	3,300
Chlorinated organic compounds (ug/kg or ppb)							
1,2,4-Trichlorobenzene	51	na	10	>4.8	31	na	64
1,2-Dichlorobenzene	>110	50	6	na	35	na	50
1,3-Dichlorobenzene	>170	>170	21	>4.4	>170	na	>170
1,4-Dichlorobenzene	120	110	97	na	110	na	120
Hexachlorobenzene	130	22	6	na	70	>120	230

Table 1. Continued

SUBSTANCES OF CONCERN	BIOASSAYS						
	Amphipod Bioassay	***Benthic Community Analysis***	***Bivalve Bioassay***	***Echinoderm Bioassay***	***Microtox Bioassay***	***Neanthes Bioassay***	***Oyster Bioassay***
	[1994]	[1988]	[1994]	[1994]	[1986]	[1998]	[1986]
Phthalates (ug/kg or ppb)							
Bis[2-ethylhexyl] phthalate	>8,300	1,300	2,200	1,700	1,900	2,000	1,900
Butyl benzyl phthalate	970	900	100	200	63	>580	>470
Di-n-butyl phthalate	1,400	>5,100	58	>31	1,400	na	1,400
Di-n-octyl phthalate	>2,100	6,200	61	>98	na	na	>420
Diethyl phthalate	>1,200	200	6	>62	>48	na	>73
Dimethyl phthalate	>1,400	>1,400	6	85	71	na	160
Miscellaneous Extractables (ug/kg or ppb)							
Dibenzofuran	1,700	700	140	110	540	630	540
Hexachlorobutadiene	180	11	6	1.3*	120	na	270
Hexachloroethane	140	na	73	na	na	na	na
N-Nitrosodiphenylamine	48	28	na	>25	40	na	130
Volatile Organics							
Ethylbenzene	50	10	na	4	33	na	37
Tetrachloroethene	>210	57	na	>1	140	na	140
Total xylenes	160	40	4	>21	100	na	120
Pesticides and PCBs (ug/kg or ppb)							
Aldrin	9.5	na	21	9.5	na	>21	na
Chlordane	2.8	na	na	>4.5	na	na	na
Dieldrin	3.5	na	34	1.9	na	34	na
Heptachlor	1.5	na	0.3	2	na	na	na
p,p'-DDD (TDE)	63	16	44	28	na	68	na
p,p'-DDE	62	9	na	9.3	na	na	na
p,p'-DDT	>270	34	46	12	na	19	>6
Total PCBs	3,100	1,000	4,900	450	130	>4,900	1,100
Ionizable organic compounds (up/kg or ppb)							
Phenols							
2-Methylphenol	77	72	6	55	>72	na	63
2,4-Dimethylphenol	77	210	na	55	29	na	29
4-Methylphenol	3,600	1,800	100	110	670	880	670
Phenol	1,200	1,200	160	>220	1,200	180	420
Pentachlorophenol	400	690	12	>150	>140	na	>140
Miscellaneous Extractables (ug/kg or ppb)							
Benzoic acid	760	650	180	>31	650	na	650
Benzyl alcohol	73	870	16	>12	57	na	73

* This table was obtained from the State of Washington Dept. of Ecology and contains several SOCs not addressed in the remainder of this report.

** This concentration was not used because there is some question about its accuracy.

[1] AETs associated with these bioassays are derived from the following sources: Amphipod and Echinoderms from the State of Washington Department of Ecology (WADOE) 1996; Oysters, Microtox and Benthic Community Analysis from the Puget Sound Estuary Program 1988; and Neathes and Bivalve AETS via personal communication with Brett Betts, WADOE, October, 2001.

For our analyses, the MSQS for HCB (22 parts per billion or ppb) is defined as our threshold for injury. Within the State's database, all tested sediment samples containing more than 22 ppb of HCB show an adverse effect on benthic communities; that is, all analyses indicate a significant reduction in invertebrate population abundance and/or species diversity when compared to a reference location. We choose to define this initial injury level as relatively insignificant; i.e., 5% of the ecological service value for any form of marine habitat[2]. For the purposes of our analyses, as more AETs are exceeded we assume a greater injury and assess a greater amount of service loss. We choose three additional injury levels for HCB (and other SOCs where only Washington SMS data are available): a 10% reduction in ecological service value or service loss when at least half of the AETs are exceeded, a 15% service loss when three-quarters of the AETs are exceeded, and a 20% service loss when all invertebrate AETs are exceeded. These additional injury levels are associated with HCB concentrations of 70, 130, and 230 ppb (Table 2).

A 20% service loss is the maximum injury level assigned to HCB because no data were obtained to indicate effects on biota other than invertebrates. Some may argue that assigning this level of service loss to marine sediments is too low if all tested invertebrate groups are adversely affected. This criticism results from several factors, such as:

- By definition, AETs reflect the *MINIMUM* concentration at which an effect is observed in *ALL* tests. AETs are continually evolving; with associated concentrations usually moving higher simply because all you need to change the AET is to identify a test that shows no effect at that concentration.
- Marine AETs are often mortality endpoints and frequently focus on a 10-day acute test that usually does not reflect sublethal effects on tested organisms, and they are gross evaluations of an organism's ability to survive in some contaminated sediment state.
- Some AETs only focus on the adult life stage, a period in the life cycle that is not necessarily the most sensitive to chemical effects.
- None are life cycle tests that focus on whether an animal can live, grow, reproduce and maintain their population.
- Additionally, an invertebrate may not metabolize ingested chemicals, and although not injurious to the invertebrate, the concentration of contaminant may be harmful to a higher trophic-level organism that eats the contaminant-laced invertebrate—an example is a fish injury at a concentration lower than an invertebrate AET (see PCBs
- Finally, although no documented information is available on HCB effects on phyla other than invertebrates, mortality to invertebrates and diminution of either benthic community abundance or diversity should have a measurable effect on higher trophic-level organisms. If the quantity or quality of food is diminished, foraging organisms will be required to expend greater amounts of their finite energy budget to replace the diminished food resources (see Page 3).

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[2] Habitats used in our HEA include: marsh, intertidal areas (13 feet (ft) above to 4 ft below Mean Lower Low Water (MLLW)); shallow subtidal areas (4 ft below to 14 ft below MLLW); and deep subtidal areas (depths more than 14 ft below MLLW).

Table 2. Concentrations of chlorobenzenes estimated to cause injuries to natural resources in Hylebos Waterway. Injuries are based on State of Washington SQS and AET values, expressed in dry weight.

SOC	BIOASSAY	CONCENTRATION	INJURY
Hexachlorobenzene (HCB)			
	"Bivalve" AET	6	(Not used)[1]
	Echinoderm AET	--	
	Benthic Community	22 ◄	5% Service Loss
	Microtox AET	70 ◄	10% Service Loss
	Amphipod AET	130 ◄	15% Service Loss
	Neanthes AET	>120	
	Oyster AET	230 ◄	20% Service Loss
1,2-dichlorobenzene (oDCB)			
	"Bivalve" AET	6	(Not used)[1]
	Echinoderm AET	na	
	Neanthes AET	na	
	Microtox AET	35 ◄	5% Service Loss
	Benthic Community	50	
	Oyster AET	50 ◄	20% Service Loss
	Amphipod AET	>110	
1,3-dichlorobenzene (mDCB)			
	Echinoderm AET	>4.4	
	"Bivalve" AET	21 ◄	5% Service Loss
	Neanthes AET	--	
	Oyster AET	>170	
	Microtox AET	>170	
	Amphipod AET	>170	
	Benthic Community	>170	
1,4-dichlorobenzene (pDCB)			
	"Bivalve" AET	97	(Not used)[1]
	Benthic Community	110 ◄	10% Service Loss
	Microtox AET	110	
	Echinoderm AET	--	
	Oyster AET	120 ◄	20% Service Loss
	Neanthes AET	--	
	Amphipod AET	120	
1,2,4-trichlorobenzene (TCB)			
	Echinoderm AET	>4.8	
	"Bivalve" AET	10	(Not used)[1]
	Benthic Community	--	
	Microtox AET	31 ◄	5% Service Loss
	Amphipod AET	51 ◄	10% Service Loss
	Oyster AET	64 ◄	20% Service Loss
	Neanthes AET	--	

[1] The "bivalve" bioassay AET is not used if values are present for the more-accepted Oyster bioassay.

Conversely, an AET represents a concentration of a substance where an effect always occurs, but this substance is found in combination with a variety of other contaminants (of different concentrations) in tested sediments. Typically no single contaminant is present in sediment. AETs, then, do not reflect the singular effect from an individual contaminant.

Although there are arguments to suggest a 20% Service Loss is either insufficient or too excessive, we believe that the service losses assigned to invertebrate bioassay data do not overstate injury.

Injuries for Other SOCs Based Solely on Washington SMS Information—There are several other SOCs for which only benthic and bioassay data from the Washington Department of Ecology are readily available. In most instances, *four or more AETs* are present in our analytical database. In those cases, service loss is established in a manner identical to that used for HCB. SOCs in this category include:

Metals	***Phenols***	***Phthalates***	***Other SOCs***
Antimony	2-methyl phenol	Butylbenzyl phthalate	Hexachlorobutadiene
Arsenic	4-methyl phenol		
Cadmium	2,4-dimethyl phenol		
Copper			
Lead			
Mercury			
Silver			
Zinc			

In some instances, however, fewer than four AETs are available, and we identify service loss per SOC based on the following scheme. When *only three AETs* are available, the lowest is identified as a 5% Service Loss, the second lowest is defined as a 10% Service Loss, and the third is defined as 20% Service Loss (i.e., all available AETs are exceeded). SOCs in this category include the following.

Metals	***Phenols***	***Phthalates***	***Other SOCs***
Chromium	Pentachlorophenol	bis (2-ethylhexyl) phthalate	1,2,4-trichlorobenzene
	Phenol	Dimethylphthalate	
		Di-n-butyl phthalate [3]	

When *only two AETs* are available, the lowest is identified as a 5% Service Loss, and the highest as a 20% Service Loss. SOCs in this category include the following

Metals	***Chlorobenzenes***	***Phthalates***	***Other SOCs***
Nickel	1,2-dichlorobenzene	Diethylphthalate	none
	1,4-dichlorobenzene	Di-n-octyl phthalate	

In one instance, *only one AET* is identified for a SOC (i.e., 1-3 dichlorobenzene) and is assigned a 5% Service Loss.

Service losses associated with various concentrations of metals are presented in Table 3, and service losses associated with concentrations of phenols, phthalates, and Hexachlorobutadiene are presented in Tables 4, 5, and 6, respectively.

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[3] For Di-n-butyl phthalate, all three AETs have the same concentration: 1,400 ppb dw. In this instance, only one service loss level (20%) is assigned (Table 5).

Table 3. Concentrations of metals estimated to cause injuries to natural resources in Hylebos Waterway Injuries are based on State of Washington Sediment Quality Standards and AET values, expressed in parts per million dry weight.

SOC	BIOASSAY	CONCENTRATION (ppm)	INJURY
Antimony (Sb)			
	"Bivalve" AET	5.9	5% Service Loss
	Echinoderm AET	9.3	
	Neanthes AET	21	10% Service Loss
	Microtox AET	--	
	Benthic Community	150	15% Service Loss
	Oyster AET	--	
	Amphipod AET	200	20% Service Loss
Arsenic (As)			
	"Bivalve" AET	35	(Not used)[1]
	Benthic Community	57	5% Service Loss
	Neanthes AET	63	
	Echinoderm AET	130	10% Service Loss
	Amphipod AET	450	15% Service Loss
	Microtox AET	700	
	Oyster AET	700	20% Service Loss
Cadmium (Cd)			
	"Bivalve" AET	3.6	(Not used)[1]
	Echinoderm AET	2.7	5% Service Loss
	Neanthes AET	3.0	
	Benthic Community	5.1	10% Service Loss
	Microtox AET	9.6	
	Oyster AET	9.6	15% Service Loss
	Amphipod AET	14	20% Service Loss
Chromium (Cr)			
	"Bivalve" AET	63.5	5% Service Loss
	Neanthes AET	94	10% Service Loss
	Microtox AET	--	
	Echinoderm AET	>96	
	Benthic Community	260	20% Service Loss
	Oyster AET	--	
	Amphipod AET	>1100	
Copper (Cu)			
	Neanthes AET	270	5% Service Loss
	"Bivalve" AET	298	(Not used)[1]
	Oyster AET	390	
	Echinoderm AET	390	10% Service Loss
	Microtox AET	390	
	Benthic Community	530	15% Service Loss
	Amphipod AET	1,300	20% Service Loss

[1] The "bivalve" bioassay AET is not used if values are present for the more-accepted Oyster bioassay.

Table 3. Continued.

SOC	BIOASSAY	CONCENTRATION (ppm)	INJURY
Lead (Pb)			
	"Bivalve" AET	336	(Not used)[1]
	Neanthes AET	360 ◄	5% Service Loss
	Echinoderm AET	430	
	Benthic Community	450 ◄	10% Service Loss
	Microtox AET	530 ◄	15% Service Loss
	Oyster AET	660	
	Amphipod AET	1,200 ◄	20% Service Loss
Mercury (Hg)			
	Microtox AET	0.41 ◄	5% Service Loss
	Oyster AET	0.59	
	Neanthes AET	1.3 ◄	10% Service Loss
	Echinoderm AET	1.4 ◄	15% Service Loss
	"Bivalve" AET	1.7	(Not used)[1]
	Benthic Community	2.1	
	Amphipod AET	2.3 ◄	20% Service Loss
Nickel (Ni)			
	"Bivalve" AET	>82	
	Microtox AET	--	
	Oyster AET	--	
	Echinoderm AET	110 ◄	5% Service Loss
	Benthic Community	>140	
	Neanthes AET	150 ◄	20% Service Loss
	Amphipod AET	>370	
Silver (Ag)			
	"Bivalve" AET	3.0 ◄	5% Service Loss
	Microtox AET	>0.56	
	Oyster AET	>0.56	
	Neanthes AET	3.3 ◄	10% Service Loss
	Amphipod AET	6.1 ◄	15% Service Loss
	Benthic Community	>6.1	
	Echinoderm AET	8.4 ◄	20% Service Loss
Zinc (Zn)			
	Benthic Community	410 ◄	5% Service Loss
	Echinoderm AET	460	
	Neanthes AET	530 ◄	10% Service Loss
	"Bivalve" AET	839	(Not used)[1]
	Microtox AET	1,600 ◄	15% Service Loss
	Oyster AET	1,600	
	Amphipod AET	3,800 ◄	20% Service Loss

[1] The "bivalve" bioassay AET is not used if values are present for the more-accepted Oyster bioassay.

Table 4. Concentrations of phenols estimated to cause injuries to natural resources in Hylebos Waterway Injuries are based on State of Washington Sediment Quality Standards and AET values, expressed in parts per billion dry weight.

SOC	BIOASSAY	CONCENTRATION (ppb)	INJURY
2-methyl phenol (MP2)			
	"Bivalve" AET	6	(Not used)[1]
	Neanthes AET	--	
	Echinoderm AET	55	5% Service Loss
	Oyster AET	63	10% Service Loss
	Benthic Community	72	15% Service Loss
	Microtox AET	>72	
	Amphipod AET	77	20% Service Loss
4-methyl phenol (MP4)			
	"Bivalve" AET	100	(Not used)[1]
	Echinoderm AET	110	5% Service Loss
	Oyster AET	670	
	Microtox AET	670	10% Service Loss
	Neanthes AET	880	
	Benthic Community	1,800	15% Service Loss
	Amphipod AET	3,600	20% Service Loss
2,4-dimethyl phenol (DMP)			
	"Bivalve" AET	--	(Not used)[1]
	Oyster AET	29	5% Service Loss
	Microtox AET	29	
	Echinoderm AET	55	10% Service Loss
	Neanthes AET	--	
	Amphipod AET	77	15% Service Loss
	Benthic Community	210	20% Service Loss
Pentachlorophenol (PCP)			
	"Bivalve" AET	12	5% Service Loss
	Oyster AET	>140	
	Microtox AET	>140	
	Echinoderm AET	>150	
	Neanthes AET	--	
	Amphipod AET	400	10% Service Loss
	Benthic Community	690	20% Service Loss
Phenol			
	"Bivalve" AET	160	(Not used)[1]
	Neanthes AET	180	5% Service Loss
	Echinoderm AET	>220	
	Oyster AET	420	10% Service Loss
	Microtox AET	1,200	
	Amphipod AET	1,200	
	Benthic Community	1,200	20% Service Loss

[1] The "bivalve" bioassay AET is not used if values are present for the more-accepted Oyster bioassay.

Table 5. Concentrations of phthalates estimated to cause injuries to natural resources in Hylebos Waterway Injuries are based on State of Washington Sediment Quality Standards and AET values, expressed in parts per billion dry weight.

SOC	BIOASSAY	CONCENTRATION (ppb)	INJURY
bis [2-Ethylhexyl] phthalate (bEPH)			
	Benthic Community	1,300 ◄	5% Service Loss
	Echinoderm AET	1,700	
	Microtox AET	1,900 ◄	10% Service Loss
	Oyster AET	1,900	
	Neanthes AET	2,000 ◄	20% Service Loss
	"Bivalve" AET	2,200	(Not used)[1]
	Amphipod AET	>8,300	
Butylbenzyl phthalate (BBPH)			
	Microtox AET	63 ◄	5% Service Loss
	"Bivalve" AET	100	
	Echinoderm AET	200 ◄	10% Service Loss
	Oyster AET	>470	
	Neanthes AET	>580	
	Benthic Community	900 ◄	15% Service Loss
	Amphipod AET	970 ◄	20% Service Loss
Di-n-butyl-phthalate (DnBPH)			
	"Bivalve" AET	58	(Not used)[1]
	Microtox AET	1,400 ◄	20% Service Loss
	Neanthes AET	na	
	Echinoderm AET	>31	
	Oyster AET	1,400	
	Amphipod AET	1,400	
	Benthic Community	>5,100	
Di-n-octyl phthalate (DOPH)			
	"Bivalve" AET	61 ◄	5% Service Loss
	Microtox AET	na	
	Neanthes AET	na	
	Echinoderm AET	>98	
	Oyster AET	>420	
	Amphipod AET	>2,100	
	Benthic Community	6,200 ◄	20% Service Loss
diethylphthalate (DEPH)			
	"Bivalve" AET	6 ◄	5% Service Loss
	Echinoderm AET	>62	
	Neanthes AET	--	
	Microtox AET	>48	
	Oyster AET	>73	
	Benthic Community	200 ◄	20% Service Loss
	Amphipod AET	>1,200	
dimethylphthalate (DMPH)			
	"Bivalve" AET	6	(Not used)[1]
	Microtox AET	71 ◄	5% Service Loss
	Echinoderm AET	85 ◄	10% Service Loss
	Neanthes AET	--	
	Oyster AET	160 ◄	20% Service Loss
	Benthic Community	>1,400	
	Amphipod AET	>1,400	

[1] The "bivalve" bioassay AET is not used if values are present for the more-accepted Oyster bioassay.

Table 6. Concentrations of hexachlorobudadiene estimated to cause injuries to natural resources in Hylebos Waterway. Injuries are based on State of Washington Sediment Quality Standards and AET values expressed in parts per billion dry weight.

SOC	BIOASSAY	CONCENTRATION (ppb)	INJURY
Hexachlorobutadiene (HCBD)			
	"Bivalve" AET	6	(Not used)[1]
	Benthic Community	11	5% Service Loss
	Microtox AET	120	10% Service Loss
	Amphipod AET	180	15% Service Loss
	Echinoderm AET	1.3 [2]	
	Oyster AET	270	20% Service Loss
	Neanthes AET	--	

[1] The "bivalve" bioassay AET is not used if values are present for the more-accepted Oyster bioassay.

[2] There is some question about the validity of this number; therefore, it was not used.

PAH INJURIES TO VERTEBRATES AND INVERTEBRATES

The extent of information on injuries from PAH contamination is more extensive than that for HCB. Effects data are available for both invertebrates and fishes. We focus our analysis on two types of information. First is a discussion of some biological effects of PAHs on English sole *Pleuronectes vetulus.* This information is compiled from studies performed by the NOAA Northwest Fisheries Science Center, and summarized by Johnson (2000). It is expressed in terms of Total PAHs, a combination of numerous high and low molecular weight PAHs that are listed in Table 7. The second source of material on PAH effects is AET information on invertebrates from the Washington Sediment Management Standards database. Unfortunately, a Total PAHs concentration is not provided in the AET data, only total high molecular weight PAHs (Total HPAHs) and total low molecular weight PAHs (Total LPAHs). The Total HPAHs concentrations are used in this is report for effects comparisons between the flatfish and invertebrates. These concentrations are chosen because they represent higher AETs than for LPAHs, and consequently are considered not to overstate injuries.

Table 7. Polycyclic aromatic hyrdocarbons (PAHs) combined to represent Total PAHs in NWFSC studies of PAH effects on English Sole.

Low Molecular Weight PAHs	***High Molecular Weight PAHs***
2-Methylnaphthalene	Benz[a]anthracene
Acenaphthene	Benzo[a]pyrene
Acenaphthylene	Benzo[ghi]perylene
Anthracene	benzofluoranthenes (b+k)
Fluorene	Chrysene
Naphthalene	Dibenzo[a,h]anthracene
Phenanthrene	Fluoranthene
	Indeno[1,2,3-cd]pyrene
	Pyrene

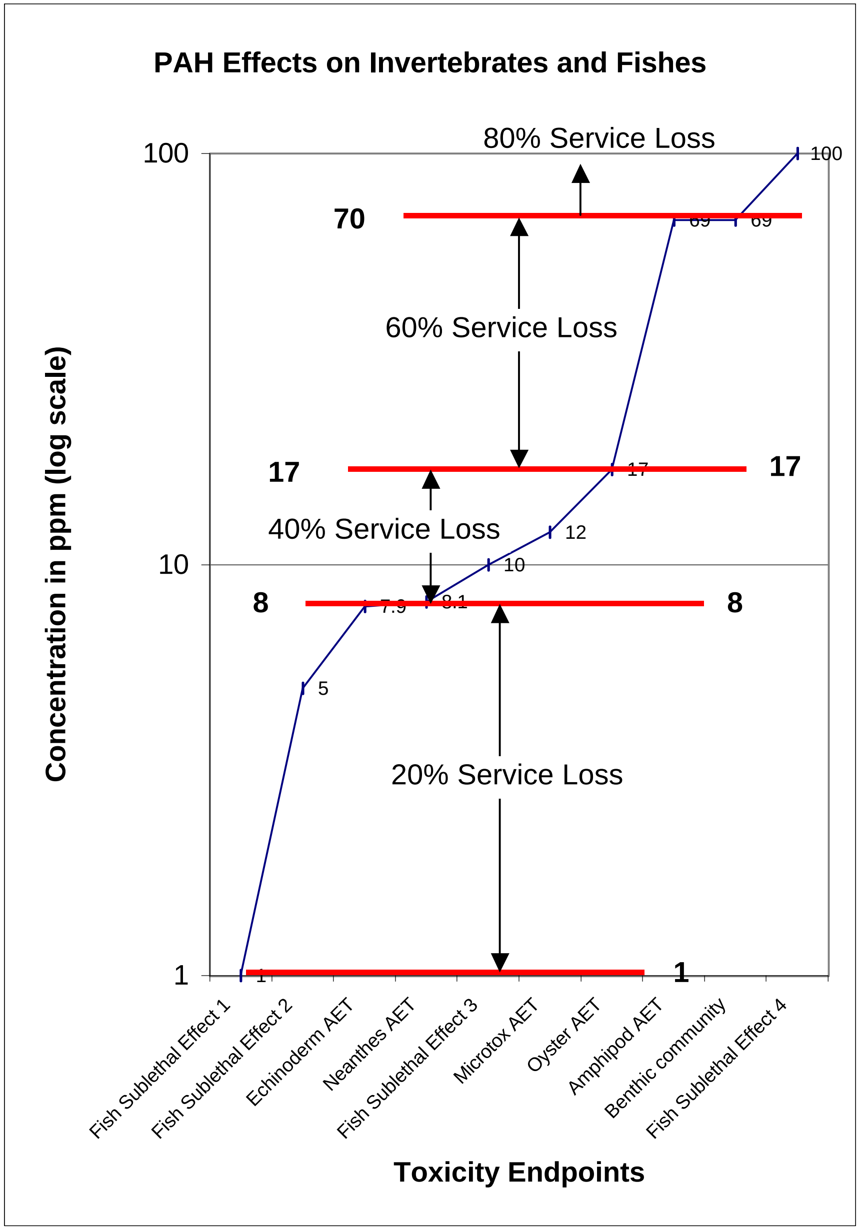
English sole continues to be one of the most extensively studied fish species in pollution monitoring research. Because it is a relatively shallow-water bottom-dwelling flatfish and occurs in urban and non-urban environments along the Pacific Coast of North America, it is particularly likely to take up sediment-associated contaminants, both through direct contact with sediments and through its diet. Since this species is relatively sedentary and shows high fidelity to sites where it resides, biological effects in English sole are generally an accurate reflection of PAH exposure at sites where they are collected. Numerous studies show that English sole from PAH-contaminated embayments are highly susceptible to the development of liver cancer and related lesions, and also appear to be prone to several other adverse health effects, such as reproductive abnor-malities, immune dysfunction, and alterations in growth and development (Myers *et al.* 1994, 1998; Arkoosh *et al.* 1966; and Johnson *et al.* 1998).

Based on a review of the English sole studies and the State of Washington SMS information, the following can be stated about PAH injuries to natural resources.

- The first sign of reproductive effects of PAHs on English sole in Puget Sound occurs at about 1 ppm dry weight, and cancerous/pre-cancerous lesions are also fairly frequently encountered. At that concentration, nearly 10% of English sole studied contain 1 or more of a variety of toxicopathic lesions in soft body tissue and nearly 5% of adult females are infertile when compared to female populations in relatively uncontaminated areas.
- By 5 ppm, the number of individuals with lesions has increased three-fold and female infertility has increased to 17% above baseline. By 7.9 ppm, invertebrates also begin to be affected (Table 1).
- By 10 ppm, over 40% of all English sole studied have one or more lesions, nearly 25% of adult females are infertile, and between 10 and 69 ppm, more than half of the invertebrate bioassays show adverse effects.
- By 100 ppm, over 70% of all English sole studied in Puget Sound have some form of toxicopathic lesions, and for adult females, over half have inhibited gonadal growth, over two-thirds do not participate in spawning, and more than three-quarters are infertile. All invertebrate AETs are exceeded.

Information in the previous paragraph describes a significant range of injuries to natural resources from exposure to PAHs in marine sediments. This suggests that a significant range of service losses is associated with these varied injuries. Initial impacts to vertebrates are reported by Johnson (2000) to begin at sediment concentrations as low as 54 ppb, and the variety and extent of injuries to English sole increase markedly with rising PAH concentrations. While this information is from studies focusing on direct exposure to contaminated sediments, these fish indirectly suffer substantial additional exposure through ingestion of invertebrate prey residing in contaminated sediment (Rice *et al.* 1999). In turn, the prey are directly affected by sediment concentrations of PAHs, as low as 7.9 ppm.

To map PAH injuries and identify a range of service losses, impacts on English sole and invertebrate AETs are graphed against PAH concentrations in sediments (Figure 1). The PAH concentrations are represented on the y-axis in (base 10) logarithmic form to permit observing effects details at low concentrations as well as evaluate effects at very high concentrations on the same scale. A 20% threshold service loss is assigned at 1ppm dry weight, with a general grouping of additional flatfish injuries and an invertebrate AET between 1 ppm and 8.1 ppm (Figure 1). This initial service loss is much higher than that assigned for HCB (discussed on page 3) because more trophic levels of the biological community are



References	Toxicity Endpoint	Concentration ppm dw
4	Fish Sublethal Effect 1	1
4	Fish Sublethal Effect 2	5
1	Echinoderm AET	7.9
1	Neanthes AET	8.1
4	Fish Sublethal Effect 3	10
1	Microtox AET	12
1	Oyster AET	17
1	Amphipod AET	69
1	Benthic community	69
4	Fish Sublethal Effect 4	100

References

1 Washington Department of Ecology
4 Johnson 2000

Flatfish Injuries

Sublethal 1 --- initial effects on fecundity and occurrence of lesions in soft tissue
Sublethal 2 -- significant increases lesion occurrence (>30% and reduced fecundity (>15%)
Sublethal 3 -- >40% of all individuals with lesions and fecundity reduced by ~25%
Sublethal 4 -- ~75% occurrence of lesions and fecundity reduced by ~50%.

80% Service Loss > 70 ppm dry weight	All tested invertebrates affected; flatfish injuries include ~50% reduction in fecundity and ~75% occurrence of at least one lesion/fish
60% Service Loss 17 to 70 ppmt	All tested invertebrates affected.
40% Service Loss 8 to 17 ppm	One-half of tested invertebrates affected; sig-nificant injuries to flatfish include ~25% reduc-tion in fecundity & 31% occurrence of lesions
20% Service Loss 1 to 8 ppm	Begin to see effects on invertebrates and fishes. Flatfish fecundity reduced by ~5% and up to 10% of all fish with some form of lesion.

Figure 1. Information used to determine injury threshold concentrations for Total PAHs and their associated Percent Service Losses. November 14, 2001

involved. A 40% service loss is assigned to the range of 8 to 17 ppm because both the extent of biological effects on fish and the number of invertebrate AETs that are exceeded at these concentrations. A 60% service loss is assigned to the range of 17 to 70 ppm because of continued substantial increases of biological effects and the incorporation of all invertebrate AETs. Finally, an 80% service loss is assigned to PAH sediment concentrations above 70 ppm.

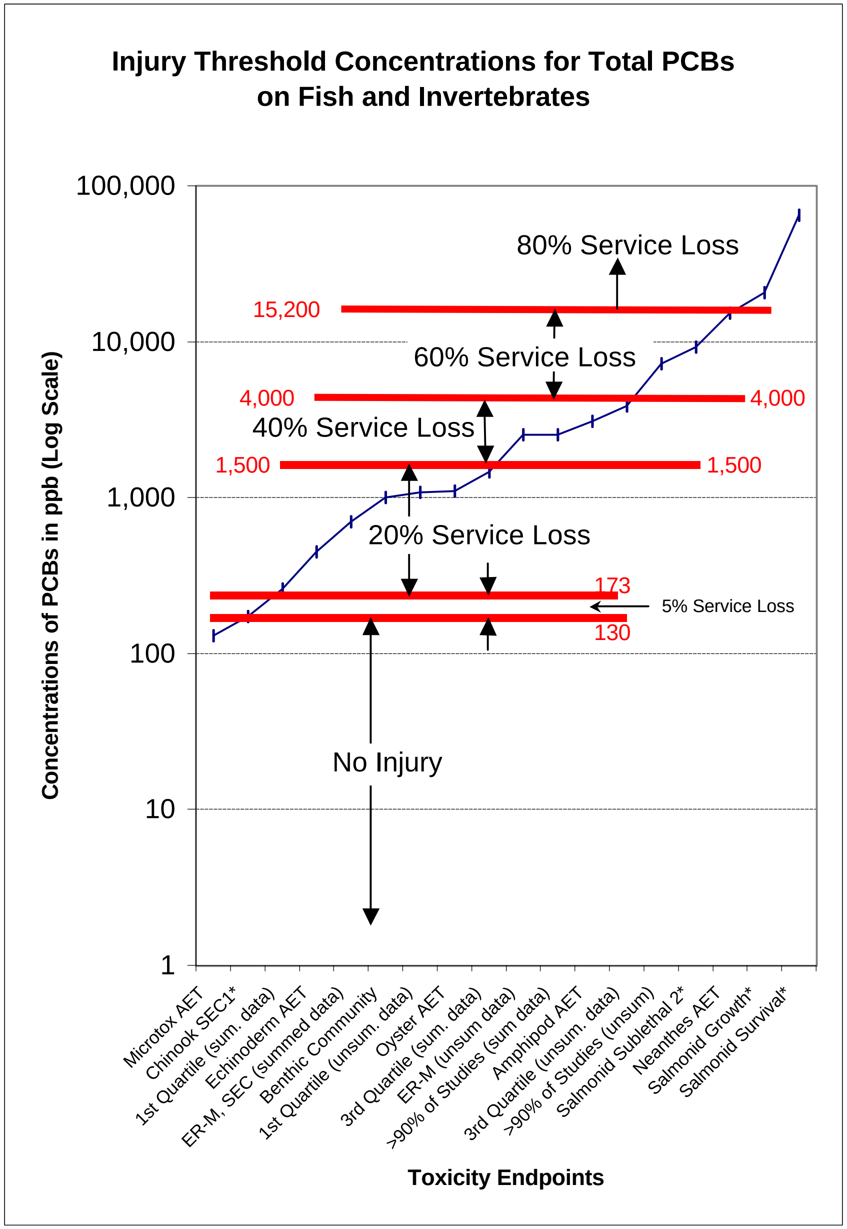
Other SOCs Handled Similarly to PAHs--An evaluation of the extent of vertebrate and invertebrate injuries are similar for four other SOCs: total polychlorinated biphenyls (PCBs), p,p'DDT, p,p'DDE, and p,p'DDD. Significant information was reviewed in addition to the Washington SMS data, including extensive reviews of numerous effects studies by MacDonald (1994) for PCBs and the various DDT congeners, and other effects data for PCBs on several salmonid species (Meador et al. In press [b], and others). As with PAHs, a graph of toxic endpoints and associated concentrations has been developed for each SOC. An attempt is made to assign higher service losses at points on the graph where there is a notable increase in effect concentration between adjacent listed endpoints

PCBs--Although the range of effects on fishes and invertebrates are similar for PAHs and PCBs, an additional service loss level is assigned to PCBs to accommodate the occurrence of an invertebrate AET at a concentration lower than the initial fish injury. A 5% Service Loss, similar to initial invertebrate effects on many previously discussed SOCs, is incorporated for the Microtox™ AET. This results in the presence of five injury levels: 5-20-40-60-and 80% Service Losses for five concentration ranges of 130-173 ppb (5% Service Loss), 173-1,500 ppb (20% Service Loss), 1,500-4,000 ppb (40% Service Loss), 4,000-15,200 ppb (60% Service Loss), and greater than 15,200 ppb (80% Service Loss (Figure 2).

DDT Congeners--DDTs are assigned 10-20-30-40% service losses. These SOCs are assigned a lower service loss range than PAHs and PCBs due to the scope of biological effects identified in available scientific literature; most studies only describe injuries to invertebrates. A summary graph and table of service loss information for p,p'DDT is found in Figure 3. It identifies injury ranges as 12-45 ppb (10% Service Loss), 45-456 ppb (20% Service Loss), 456-2,100 ppb (30% Service Loss) and concentrations greater than 2,100 ppb (40% Service Loss).

Figure 4 summarizes toxic endpoint information for p,p'DDE. It identifies injury ranges as 9-65 ppb (10% Service Loss), 65-7,000 ppb (20% Service Loss), 7,000-21,500 ppb (30% Service Loss) and concentrations greater than 21,500 ppb (40% Service Loss).

Figure 5 summarizes toxic endpoint information for p,p'DDD. It identifies injury ranges as 16-70 ppb (10% Service Loss), 70-1,500 ppb (20% Service Loss), 1,500-3,600 ppb (30% Service Loss) and concentrations greater than 3,600 ppb (40% Service Loss).



Reference	Toxicity Endpoint	Concentration ppb dw
1	Microtox AET	130
3	Chinook SEC[1*]	173
2	1st Quartile (sum. data)	259
1	Echinoderm AET	450
2	ER-M, SEC (summed data)	699
1	Benthic Community	1,000
2	1st Quartile (unsum. data)	1,080
1	Oyster AET	1,100
2	3rd Quartile (sum. data)	1,467
2	ER-M (unsum data)	2,530
2	>90% of Studies (sum data)	2,530
1	Amphipod AET	3,100
2	3rd Quartile (unsum. data)	3,864
2	>90% of Studies (unsum)	7,199
4	Salmonid Sublethal 2*	9,200
1	Neanthes AET	15,190
4	Salmonid Growth*	20,600
4	Salmonid Survival*	64,900

[1]--SEC = Sediments Effects Concentration

[2]--ER-M = Effects Range-Median

* concentrations based on 2.3% TOC

References

1 Washington Department of Ecology

2 MacDonald 1994

3 Meador et al. In press (b)

4 Stratus Consulting, 2000

80% Service Loss > 15.2 ppm dry weight	All tested invertebrates affected and salmonid growth and survival are affected
60% Service Loss 4 to 15.2 ppm	All invertebrates affected and sublethal effects on salmonids such as changes in immuno-suppression and P450 induction
40% Service Loss 1.5 to 4 ppm	most tested invertebrates affected
20% Service Loss 0.173 to 1.5 ppm	First signs of cellular compromise in chinook salmon; several invertebrates affected
5% Service Loss 130 to 172 ppb	Begin to see effects on invertebrates

Figure 2.--Information used to determine injury threshold concentrations for Total PCBs and their associated Percent Service Losses.

November 14, 2001

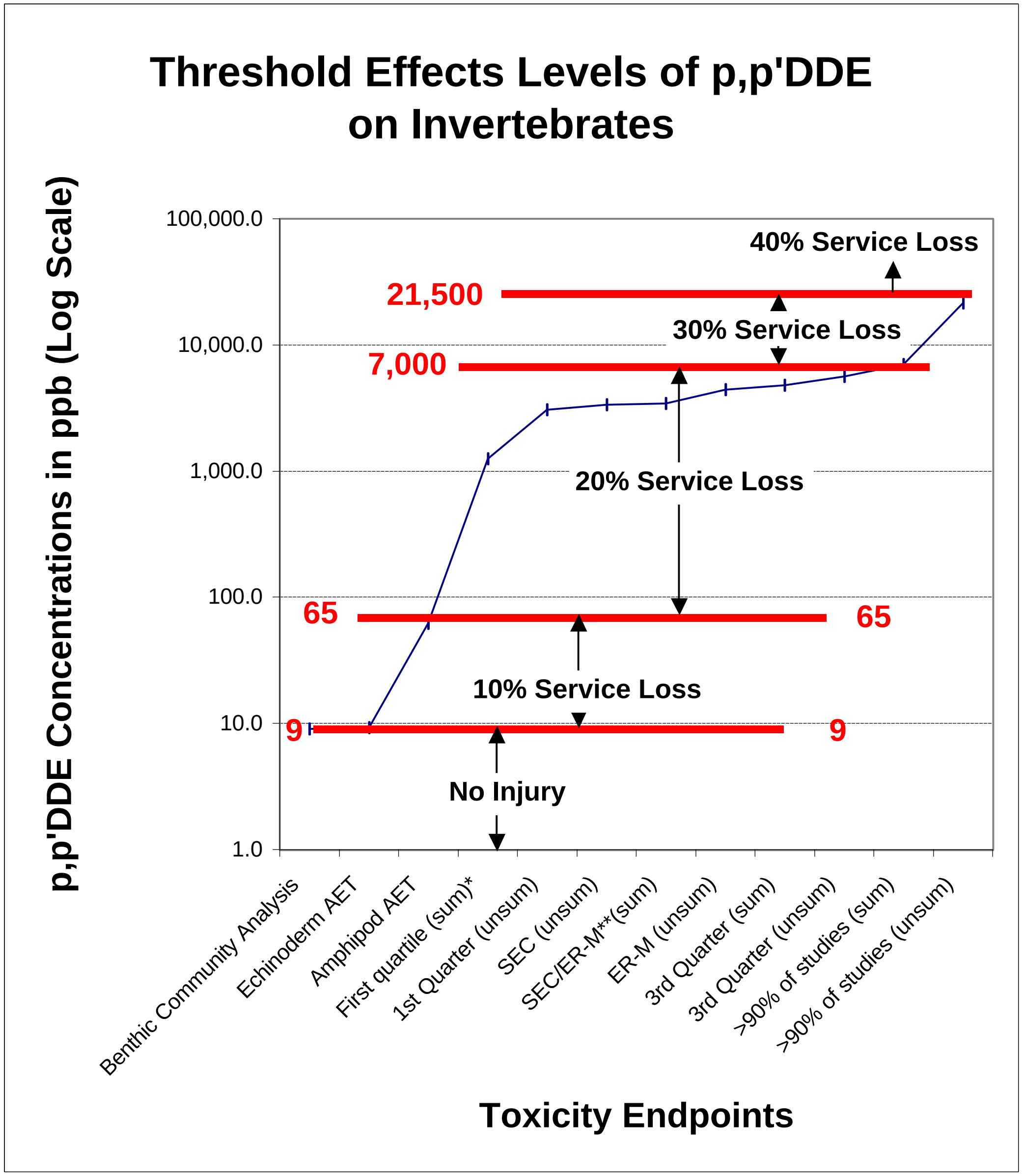
Ref.	Toxicity Endpoint	Concentration (ppb dw)*
1	Echinoderm AET	12
1	Neanthes AET	19
3	1st Quartile (summed data)	28
1	Benthic community	34
3	ER-M (summed data)	35
3	1st Quartile (unsummed data)	45
3	ER-M (unsummed data)	185
3	SEC (summed data)	228
3	3rd Quartile (summed data)	456
3	3rd Quartile (unsummed data)	1,182
3	>90% of studies (summed data)	1,643
3	>90% of studies (unsummed data)	2,012

* concentrations based on 2.3% TOC

References
1 Washington Department of Ecology
3 MacDonald, 1994

40% Service Loss >2,100 ppb dw	All bioassays show effects.
30% Service Loss 456 to 2,100 ppb	over 90% of studies reported by MacDonald 1994 show effects by this range.
20% Service Loss 45 to 456 ppb	All State of Washington AETs exceeded; Many tudies reported by MacDonald 1994 report effects in this range
10% Service Loss 12 to 45 ppb	Initial level of adverse effects observed in State of Washington AETs.

Figure 3. Information used to determine threshold injury concentrations for p,p'DDT and associated Percent Service Losses



Reference	Toxicity Endpoint	Concentration (ppb dw)
1	Benthic Community Analysis	9.0
1	Echinoderm AET	9.3
1	Amphipod AET	62.0
3	First quartile (sum)*	1,259.0
3	1st Quarter (unsum)	3,067.0
3	SEC (unsum)	3,346.0
3	SEC/ER-M**(sum)	3,424.0
3	ER-M (unsum)	4,402.0
3	3rd Quarter (sum)	4,809.0
3	3rd Quarter (unsum)	5,637.0
3	>90% of studies (sum)	7,030.0
3	>90% of studies (unsum)	21,511.0

* Concentrations for MacDonald (1994) data based on 2.3% TOC

** Effects Range-Median Sediment Effects Concentration

References

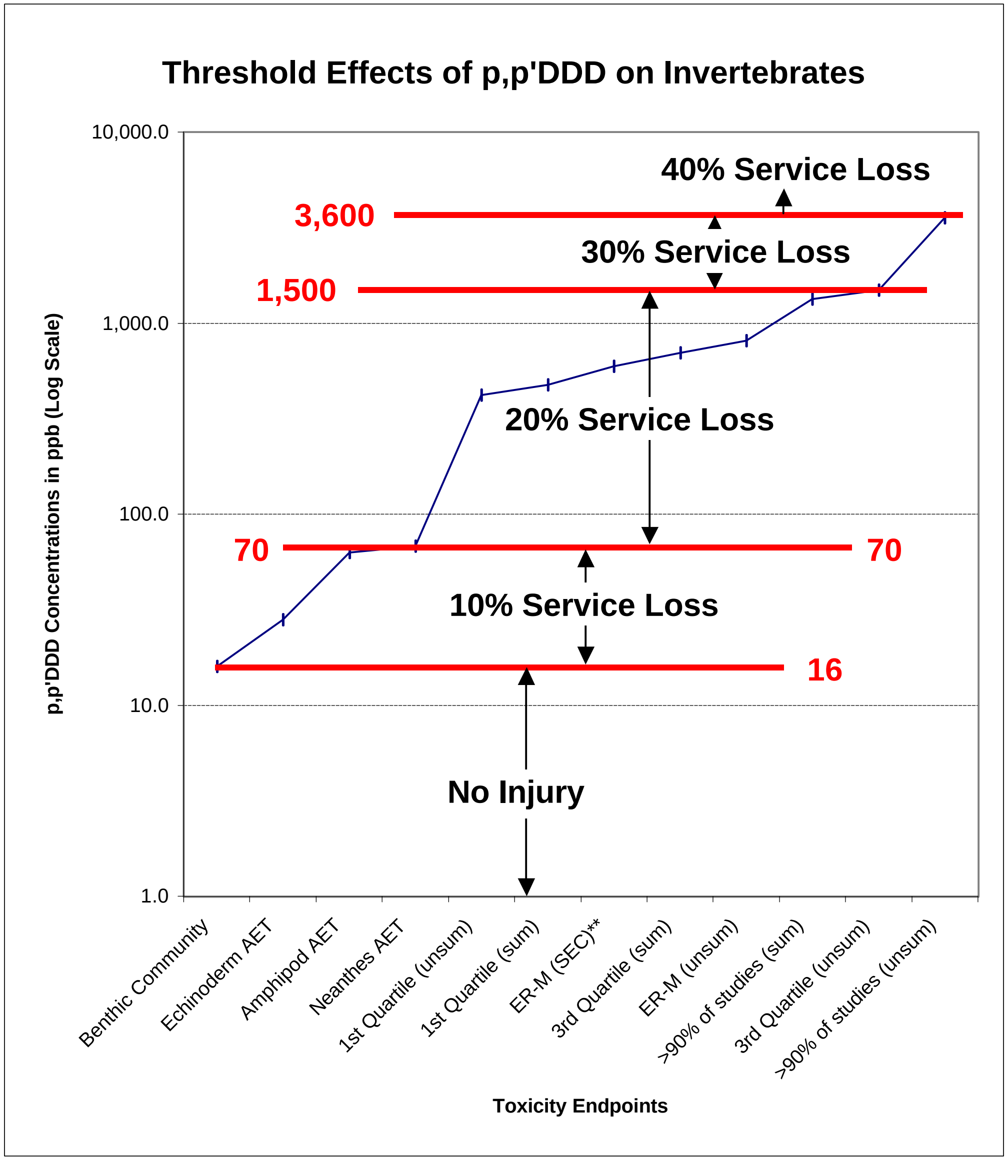
1 Washington Department of Ecology

3 MacDonald, 1994

40% Service Loss >21,500 ppb dw	All bioassays show effects.
30% Service Loss 7,000 to 21,500	Over 90% of studies examined by MacDonald 1994 show effects by this range.
20% Service Loss 65 to 7,000	All State of Washington AETs exceeded; Many tudies reported by MacDonald 1994 report effects in this range.
10% Service Loss 9 to 65 ppb	Initial level of adverse effects observed in State of Washington AETs.

Figure 4.--Information used to determine threshold injury concentrations for p,p'DDD and associated Percent Service Losses

November 14, 2001



Reference	Toxicity Endpoint	Concentration (ppb dw)*
1	Benthic Community	16.0
1	Echinoderm AET	28.0
1	Amphipod AET	63.0
1	Neanthes AET	68.0
3	1st Quartile (unsum)	420.0
3	1st Quartile (sum)	474.0
3	ER-M (SEC)**	593.0
3	3rd Quartile (sum)	700.0
3	ER-M (unsum)	808.0
3	>90% of studies (sum)	1,334.0
3	3rd Quartile (unsum)	1,493.0
3	>90% of studies (unsum)	3,553.0

* Concentrations for MacDonald (1994) data based on 2.3% TOC

** Effects Range-Median Sediment Effects Concentration

References

1 Washington Department of Ecology

3 MacDonald, 1994

40% Service Loss >3,600 ppb dw	All bioassays show effects.
30% Service Loss 1,500 to 3,600 ppb	over 90% of studies reported by MacDonald 1994 show effects by this range.
20% Service Loss 70 to 1,500 ppb	All State of Washington AETs exceeded; Many tudies reported by MacDonald 1994 report effects in this range
10% Service Loss 16 to 70 ppb	Initial level of adverse effects observed in State of Washington AETs.

Figure 5. Information used to determine injury threshold concentratons for p,p'DDD and associated Percent Service Losses.

November 14, 2001

Tributyltin Service Losses—There is no MSQS associated with tributyltin (TBT), and more recent AET information is scanty. Any information from the Pacific Northwest is relatively slight. Consequently, we rely on information compiled from studies elsewhere and the analysis by Meador et al. (In press [a]) that supports a sediment quality threshold for TBT in Puget Sound to protect prey species for juvenile salmonids that are listed by the Endangered Species Act. In this document he calculates an effects level of 6,000 ppb OC; a median concentration for all sublethal effects studies (mostly growth impairment). With a calculated average TOC for Hylebos Waterway of 2.3%t[4], the previously stated carbon normalized number would translate to 138 ppb dry weight.

For specific studies, TBT concentrations associated with adverse effects range from 100 ppb to 1000 ppb dry weight. Meador (1997) indicates that 329 ppb is a concentration in a range needed to produce a lethal response for a sensitive invertebrate such as the amphipod, *Eohaustorius washingtonianus*. Bryan and Langston (1992) and Langston and Burt (1991) (from Meador et al., In press [a]) have suggested that some populations of bivalves in United Kingdom waters have disappeared in locations with TBT sediment concentrations over 700 ppb, while Fent and Hunn (1995) (from ibid) noted that clams have disappeared in other areas where sediment TBT exceeds 800 ppb. Lastly, Meador and Rice (2001) report severe reductions in growth of the polychaete, *Armandia brevis,* for sediment concentrations in the range of 100 – 1,000 ppb.

Based on the above TBT information, the threshold value for injuries from TBT is assigned at 138 ppb dry weight (Table 8). A 10% service loss is associated with this threshold value. A 20% service loss is assigned to concentrations greater than 1,000 ppb.

Table 8. Concentrations of Tributyltin considered to cause injuries to natural resources in Hylebos Waterway

SOC	BIOASSAY	Concentration in ppb	INJURY
Tributyltin (TBT)			
	NMFS threshold[1]	138	5% Service Loss
	Bivalve abundance[2]	>800	
	Bivalve abundance[3]	>700	
	Polycheate growth[4]	1,000	20% Service Loss

[1] Meador et al.(In press [a]) proposes that a concentration of 6,000 ppb per gram Carbon would be protective for many, but not all prey species. For the average TOC for Hylebos stations, this would translate as 138 ppb DW (average TOC = 2.3%).

[2] Disappearance of clams in areas were TBT exceeded 800 ppb DW, reported by Fent and Hunn 1995.

[3] Populations of bivalves *Macoma balthica* and *Scrobicularia plana* have disappeared in locations with concentrations over 700 ppb--reported by Bryan and Langston (1992) and Langston and Burt (1991).

[4] Severe reductions in growth of *Armandia brevis* for sediment concentrations in the range of 100 - 1,000 ng/g (ppb), reported by Meador and Rice (2001)

[4] Average TOC (Total Organic Carbon) is based on the mean value for 23 of 28 sediment sampling stations occupied during the 1994 NOAA survey, conducted on behalf of the Commencement Bay Natural Resource Co-Trustees. Selection of 23 stations based on a review of station locations and areas of extensive wood waste.

SUMMARY

This report assigns threshold sediment concentrations for injuries to natural resources from various SOCs. The ecological service losses associated with the thresholds (and higher concentrations) are associated with the variety and extent of injuries pertaining to each SOC. If only invertebrate AET information is used to estimate injury thresholds, the maximum service loss value is determined to be 20%. If information from reports on injuries to fishes is incorporated into our analysis along with the AETs, both initial and maximum service losses are higher, with thresholds at 10-20% and maximum values up to 80%. A summary table (Table 9) lists injury thresholds and service losses for all SOCs discussed in this report.

ASSOCIATED INFORMATON FOR HYLEBOS WATERWAY

Threshold injury values for SOCs are needed to map injury footprints associated with each SOC in Hylebos Waterway. Attached to this report is a series of tables that list injury level information for each SOC at every sediment sampling station occupied in the various surveys incorporated into our analyses. For each SOC, calculated chemical concentrations per station are presented in descending order. All stations having injury threshold concentrations are highlighted.

Also attached to this appendix is an addendum that describes the steps and techniques for sediment data preparation.

Table 9. A summary of assigned percent service losses for all Substances of Concern incorporated into the HEA for Hylebos Waterway.

Substance of Concern	symbol	units	Levels of Service Loss 5%	10%	15%	20%	25%	30%	40%	50%	60%	75%	80%	99%
Total PAHs		ppm dw				1			8		17		70	
Total PCBs		ppm dw	0.130			0.173			1.5		4		15.2	
Metals														
Antimony	Sb	ppm dw	5.9	21	150	200								
Arsenic	As	ppm dw	57	130	450	700								
Cadmium	Cd	ppm dw	2.7	5.1	9.6	14								
Chromium	Cr	ppm dw	63.5	94	--	260								
Copper	Cu	ppm dw	270	390	530	1,300								
Lead	Pb	ppm dw	360	450	530	1,200								
Mercury	Hg	ppm dw	0.41	1.3	1.4	2.3								
Nickel	Ni	ppm dw	110	--	--	150								
Silver	Ag	ppm dw	3.0	3.3	6.1	8.4								
Zinc	Zn	ppm dw	410	530	1,600	3,800								
Tributyltin	TBT	ppm dw	0.138	--	--	1								
Chlorobenzenes														
1,2-dichlorobenzene	oDCB	ppb dw	35	--	--	50								
1,3-dichlorobenzene	mDCB	ppb dw	21	--	--	--								
1,4-dichlorobenzene	pDCB	ppb dw	--	110	--	120								
1,2,4-trichlorobenzene	TCB	ppb dw	31	51	--	62								
Hexachlorobenzene	HCB	ppb dw	22	70	130	230								
Phthalates														
bis (2-Ethylhexyl) phthalate	bEPH	ppb dw	1,300	1,900	--	2,000								
Butylbenzyl phthalate	BBPH	ppb dw	63	200	900	970								
Di-n-bytyl phthalate	DnBPH	ppb dw	--	--	--	1,400								
Di-n-octyl phthalate	DOPH	ppb dw	61	--	--	6,200								
diethylphthalate	DEPH	ppb dw	6	--	--	200								
dimethylphthalate	DMPH	ppb dw	71	85	--	160								
Phenols														
2-methyl phenol	MP2	ppb dw	55	63	72	77								
4-methyl phenol	MP4	ppb dw	110	670	1,800	3,600								
2,4-dimethyl phenol	DMP	ppb dw	29	55	77	210								
Pentachlorophenol	PCP	ppb dw	12	400	--	690								
Phenol	Phenol	ppb dw	180	420	--	1,200								
Hexachlorobutadiene	HCBD	ppm oc	11	120	180	270								
DDTs														
Dichlorodiphenyldichloroethane	p,p'DDD	ppb dw		16		70		1,500	3,600					
Dichlorodiphenyldichloroethylene	p,p'DDE	ppb dw		9		65		7,000	21,500					
Dichlorodiphenyltrichloroethane	p,p'DDT	ppb dw		12		45		456	2,100					

March 28, 2002

REFERENCES

369 Arkoosh, M. A., E. Clemons, P. Huffman. H. R. Sandborn, E. Casillas, and J. E. Stein. 1996. Leukoproliferative response of splenic leukocytes from English sole (*Pleuronectes vetulus*), exposed to chemical contaminants. Environ. Toxicol. Chem., 15:1154-1162.

370 Bryan, G. W. and W. J. Langston. 1992. Bioavailability, accumulation, and effects of heavy metals in sediments with special reference to United Kingdom estuaries: a review. Environ. Pollut. 76:89-131.

371 Cacela, D., J. Lipton, D. Beltman, and J. Hansen. 2001. Associating Ecosystem Service Loss with Indicators of Toxicity in Habitat Equivalency Analysis. Prepared for the NOAA Damage Assessment Center by Stratus Consulting, Inc., Boulder, CO 80306-4059. 18 pp.

372 Holmlund, C. M., and M. Hammer. 1999. Ecosystem services generated by fish populations. Ecological Economics 29:253-268.

373 Johnson, L. 2000. An analysis in support of sediment thresholds for polycyclic aromatic hydrocarbons (PAHs) to protect estuarine fish. USDOC, NOAA, NMFS, Northwest Fisheries Science Center, 2725 Montlake Blvd. E., Seattle, WA. 32 pp.

374 Johnson, L. L., J. T. Landahl, L. A. Kubin, B. H. Horness, M. S. Myers, T. K. Collier, and J. E. Stein. 1998. Assessing the effects of anthropogenic stressors on Puget Sound flatfish populations. Neth. J. Sea Res. 39:125-137.

375 Langston, W. J. and G. R. Burt. 1991. Bioavailability and effects of sediment-bound TBT in deposit feeding clams, *Scrobicularia plana.* Mar. Environ. Res. 32:61-77.

378 MacDonald, D. D. 1994. Sediment Injury in the Southern California Bight: Review of the Toxic Effects of DDTs and PCBs in Sediments. Prepared for NOAA, August, 1994, by MacDonald Environmental Sciences Ltd. Ladysmith, B.C., V0R 2E0. 96 pp. + Appendices.

376 Meador, J. P. 1997. Comparative toxicokinetics of tributyltin in five marine species and its utility in predicting bioaccumulation and toxicity. Aquatic Toxicol. 37:307-326.

379 Meador, J.P, T.K. Collier, and J.E. Stein. In press (a). Determination of a tissue and sediment threshold for tributyltin to protect prey species of juvenile salmonids listed under the Endangered Species Act. Submitted 22 February, 2001 for publication to Aquatic Conservation: Marine and Freshwater Ecosystems. London, U.K. 24 pp.

380 Meador, J.P. 2000. Predicting the fate and effects of tributyltin in marine systems. Rev. Environ. Contam. Toxicol. 166:1-48.

381 Meador, J.P, T.K. Collier, and J.E. Stein. In press (b). Use of tissue and sediment based threshold concentrations of polychlorinated biphenyls (PCBs) to protect juvenile salmonids listed under the U.S. Endangered Species Act. Submitted for publication to Aquatic Conservation: Marine and Freshwater Ecosystems. London, U.K. 37 pp. plus figures and tables.

382 Meador, J.P. and C.A.Rice. 2001. Impaired growth in the polychaete *Armandia brevis* exposed to tributyltin in sediment. Mar. Environ. Res. 51:113-129.

390 Michelsen, T.C. 1992. Organic Carbon Normalization of Sediment Data. Washington Department of Ecology, Sediment Management Unit, Technical Information Memorandum. 10 pp.

383 Myers, M. S., C. M. Stehr, O. P. Olson, L. L. Johnson, B. B. McCain, S-L. Chan, and U. Varanasi. 1994. Relationships between toxicopathic hepatic lesions and exposure to chemical contaminants in English sole (*Pleuronectes vetulus*), starry flounder (*Platichthys stellatus*), and white croaker (*Genyonemus lineatus*) from selected marine sites on the Pacific Coast, U.S.A. Environ. Health Perspect. 102: 200-215.

384 Myers, M. S., L. L. Johnson, T. Hom, T. K. Collier, J. E. Stein, and U. Varanasi. 1998. Toxicopathic hepatic lesions in subadult English sole (*Pleuronectes vetulus*), from Puget Sound Washington, U.S.A.: Relationships with other biomarkers of contaminant exposure. Mar. Environ. Res. 45:47-67.

388 Puget Sound Estuary Program. 1988. Sediment Quality Values Refinement: Volume 1, 1988 Update and Evaluation of Puget Sound AET. Prepared for the U.S. EPA, Region 10 B Office of Puget Sound, Seattle, WA.

385 Rowe, C. L., O. M. Kinney, R. D. Nagle, and J. D. Congdon. 1998. Elevated Maintenance costs in the Anuran (*Rana catesbeiana*) Exposed to a Mixture of Trace Elements during the Embryonic and Early Larval Periods. Physiol. Zool. 71(1):27-35.

386 Strange, E., H. Galbraith, S. Bickel, D. Mills, D. Beltman, and J. Lipton. In prep. Determining ecological equivalence in service-to-service scaling of salt marsh restoration. Submitted: Environmental Management. Stratus Consulting, Inc., P.O.Box 4059, Boulder, CO 80306. Revised April 19, 2001. 47pp.

387 U.S. Environmental Protection Agency (EPA). 1989. Commencement Bay Nearshore/Tideflats Record of Decision. Environmental Protection Agency, Region 10, Seattle, WA.

389 Washington Department of Ecology. 1996. Progress Re-evaluating Puget Sound Apparent Effects Thresholds (AESs). Volume 1: 1994 Amphipod and Echinoderm Larval AETs. Prepared for the Puget Sound Dredged Disposal Analysis (PSDDA) Agencies.

December 17 2001

MEMORANDUM FOR: Commencement Bay Natural Resource Co-Trustees

FROM: Robert Wolotira, NOAA Damage Assessment and Restoration Center NW.

SUBJECT: Calculating and separating the effects of multiple contaminants.

The purpose of this memo is to explain the rationale behind how injuries are allocated in areas where multiple Substances of Concern (SOCs) co-occur. The following is a discussion of the principles used to (1) identify total service loss when multiple SOCs co-occur and (2) to divide the total service loss among the SOCs.

Determining Total Service Loss from Multiple Co-Occurring SOCs. Cumulative loss from multiple contaminants was initially considered as a simple summation of individual service losses. However, this calculation is often inappropriate, since summing several contaminant injuries could result in a total service loss that exceeds 100%. Consequently, a proportionally-weighted total service loss is calculated as follows. The basic rule used is that the effect from each SOC is based on whatever residual habitat service value exists subsequent to any previous service loss calculation. If no previous service loss has been calculated, then the initial calculation is performed on the initial (uninjured) habitat value, and all subsequent SOC injury calculations are perfomed sequentially.

Total injury (**I.**) to a habitat is equal to the sum of injuries from all SOCs co-occurring in that habitat:

$$I. = \sum_{i=1}^{n} I_i$$

Where I_i = habitat injury from SOC_i and
n = the total number of co-occurring SOCs

The habitat injury from an SOC is equal to the product of the Percent Service Loss (PSL) of that SOC multiplied by the current Habitat Service Value (HSV) of that habitat. The current HSV of a habitat is equal to its service value less any prior injury. Total habitat injury can then be expressed by the equation:

$$I. = \sum_{i=1}^{n} (PSL_i \times HSV_{i-1})$$

Where PSL_i = Percent Service Loss from SOC_i
HSV_{i-1}= that habitat service value prior to injury by SOC_i, and
n = the total number of co-occurring SOCs

It follows that HSV_0 = the habitat service value (HSV) prior to any injury, or simply, the initial HSV. Consequently, the HSV subsequent to HSV_0 (i.e., $HSV_{i>0}$) is called the Residual Service Value (RSV). An example follows for determining total injury from three SOCs with identical toxicities (i.e., identical PSLs)

Substance of Concern	Percent Service Loss	Habitat Service Value	Injury from SOC_i		Residual Service Value
SOC_i	**PSL_i**	**HSV**	**I_i**		**RSV**
SOC_1	0.30	1.0 (i.e.,HSV_0)	= 0.300	(RSV_0 - I_i)	1.0 - 0.30 = 0.70
SOC_2	0.30	0.70 (or RSV_1)	= 0.210	(RSV_1 – I_2)	0.70 – 0.21 = 0.49
SOC_3	0.30	0.49 (i.e.,RSV_2)	= 0.147	(RSV_2 – I_3)	0.49 – 0.147=0.343
		Total Injury	**= 0.657**		

The PSL for SOC_1 is multiplied times the initial HSV to determine the injury from SOC_1, i.e., 0.30 x 1.0 = 0.30. The HSV for calculating injuries from SOC_2 is obtained by subtracting the injury from $SOC_1$from the initial HSV, i.e.,0.70; this now is RSV_1. Injury from SOC_2 is determined by multiplying its PSL (0.30) times the RSV remaining after calculating and subtracting the injury from SOC_1, or 0.30 x 0.70 = 0.21. Injury from SOC_3 is determined by multiplying its PSL (0.30) times the RSV remaining after calculating and subtracting the injury from SOC_2, or 0.30 x 0.49 = 0.147. Total injury from the effects of the three SOCs = 0.657.

Calculation of total injury would be similar for any number of SOCs and any combination of PSLs, and the same calculations would hold true if all PSLs were not identical. The following are two examples of the calculations, with the PSLs rearranged to show that regardless of the sequence the results are identical.

Dissimilar PSL listed first

Substance of Concern	Percent Service Loss	Habitat Service Value	Injury from SOC_i		Residual Service Value
SOC_i	**PSL_i**	**HSV**	**I_i**		**RSV**
SOC_1	0.10	1.0 (i.e.,HSV_0)	= 0.100	(RSV_0 - I_i)	1.0 - 0.10 = 0.90
SOC_2	0.30	0.90 (or RSV_1)	= 0.270	(RSV_1 – I_2)	0.90 – 0.27 = 0.63
SOC_3	0.30	0.63 (i.e.,RSV_2)	= 0.189	(RSV_2 – I_3)	0.63 – 0.189=0.441
		Total Injury	**= 0.559**		

Dissimilar PSL listed last

Substance of Concern	Percent Service Loss	Habitat Service Value	Injury from SOC_i		Residual Service Value
SOC_i	**PSL_i**	**HSV**	**I_i**		**RSV**
SOC_2	0.30	1.0 (i.e.,HSV_0)	= 0.300	(RSV_0 - I_i)	1.0 - 0.30 = 0.70
SOC_3	0.30	0.70 (or RSV_1)	= 0.210	(RSV_1 – I_2)	0.70 – 0.21 = 0.49
SOC_1	0.10	0.49 (i.e.,RSV_2)	= 0.049	(RSV_2 – I_3)	0.49 – 0.049=0.441
		Total Injury	**= 0.559**		

Dividing total injury among the co-occuring SOCs. Calculating total injury is a sequential process. Using this method, the initial SOC injury will be calculated on the initial HSV, or the highest value habitat. Any subsequent SOC injury calculation would involve a residual habitat service value that is less than the initial value. This situation would result in a service loss smaller than the initial service loss, even if the SOCs had identical PSLs. To rectify this inequity, the final injury value assigned to each SOC is based on the proportion of each SOC's PSL to the sum of all PSLs (not the sum of injuries). This proportion is then multiplied times the total injury to determine the final proportional injury associated with an SOC. It is expressed by the following equation:

$$\text{Final } I_i = PSL_i / \sum_{i=1}^{n} PSL_i \times \sum_{i=1}^{n} I_i$$

Where PSL_i is the Percent Service Loss for SOC_i,

ΣPSL is the total of all individual PSLs, combined, and

ΣI_i is the total (initial) injury estimate derived on the previous page

Examples follow.

Identical PSLs

Substance of Concern	Percent Service Loss		Injury from SOC_i	Percent Service Loss Proportions	Reallocated Proportional Injury
SOC_i	**PSL_i**		**I_i**	**$PSL_{i/}$ PSL.**	
SOC_1	0.30		= 0.300	.30/.90 =0.333	0.33 x 0.657 = 0.219
SOC_2	0.30		= 0.210	.30/.90 =0.333	0.33 x 0.657 = 0.219
SOC_3	0.30		= 0.147	.30/.90 =0.333	0.33 x 0.657 = 0.219
Total PSL.	**= 0.90**	**Total Injury**	**= 0.657**		**0.657**

In the preceeding example for three SOCs with identical PSLs (0.30, each), the initial injury attributed to SOC_2 is less than the injury from SOC_1, and the injury from SOC_3 is less than that for either SOC_1 or SOC_2. The final reallocated proportional injury for each SOC is calculated as 0.30/0.90 x 0.657, or 0.333 x 0.657, or 0.219; i.e., each is attributed 1/3 of the initially determined total injury.

Similarly, determining the proportional final injury to each co-occurring SOC is identical, regardless of the sequence SOCs or differences in initial PSLs

Dissimilar PSL listed first

Substance of Concern	Percent Service Loss	Initial Injury Calculation	Injury from SOC_i	Percent Service Loss Proportions	Reallocated Proportional Injury
SOC_i	**PSL_i**		**I_i**	**$PSL_{i/}$ PSL.**	
SOC_1	0.10	0.10 x 1.0	= 0.100	.10/.70 =0.1429	.1429 x 0.559 = .0799
SOC_2	0.30	0.30 x 0.90	= 0.270	.30/.70 =0.4286	.4286 x 0.559 = .2396
SOC_3	0.30	0.30 x 0.63	= 0.189	.30/.70 =0.4286	.4286 x 0.559 = .2396
Total PSL.	**= 0.70**	**Total Injury**	**= 0.559**		**0.5991**

Dissimilar PSL listed last

Substance of Concern	Percent Service Loss	Initial Injury Calculation	Injury from SOC_i	Percent Service Loss Proportions	Reallocated Proportional Injury
SOC_i	**PSL_i**		**I_i**	**$PSL_{i/}$ PSL.**	
SOC_2	0.30	0.30 x 0.90	= 0.270	.30/.70 =0.4286	.4286 x 0.559 = .2396
SOC_3	0.30	0.30 x 0.90	= 0.270	.30/.70 =0.4286	.4286 x 0.559 = .2396
SOC_1	0.10	0.10 x 1.0	= 0.100	.10/.70 =0.1429	.1429 x 0.559 = .0799
Total PSL.	**= 0.70**	**Total Injury**	**= 0.559**		**0.5991**

Stratus Consulting

Associating Ecosystem Service Loss with Indicators of Toxicity in Habitat Equivalency Analysis

Draft

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1. Introduction

This report addresses applications of Habitat Equivalency Analysis (HEA) in which toxicological information on effects of contaminants on biota is used to define changes in ecological services provided by contaminated habitats. We provide a brief overview of the major components of a HEA and discuss the concept of ecological services. We then describe a framework for developing service loss assignments that incorporates knowledge about the impacts of hazardous substances on biota. Our emphasis is on integrating multiple lines of evidence about the toxicity of hazardous substances as the basis for a mapping from toxicological conditions to ecological services. Finally, we provide an example of how our framework might be used to inform a HEA that addresses ecological services provided by estuarine sediments contaminated with polycyclic aromatic hydrocarbons (PAH).

1.1 Habitat Equivalency Analysis

HEA is a methodology that is used to quantify the relative value of different habitats with respect to the services that they provide and as a framework to scale habitat-based mitigation or restoration plans (NOAA, 1999b). Restoration scaling using HEA involves quantifying the amount of a restoration action so that the benefits of the restoration are equivalent to the losses associated with some action or harm (e.g., contamination), and where both gains and losses are measured in terms of ecological or human use "services" (NOAA, 1999b). Equivalency may be considered with respect to a single species or habitat function of concern, or may involve an integration of services provided to many species. Thus, the basic equivalency model used in HEA, with consideration of discounting due to temporal offsets, is represented as:

$$\sum_{t=t_0}^{t_l} L_t (1+i)^{(P-t)} = \sum_{s=s_0}^{s_l} R_s (1+i)^{(P-s)}$$

(Eqn. 1)

where:

L_t = lost services at time t
R_s = replacement services at time s
t_0 = time when lost services are first suffered
t_l = time when lost services are last suffered
s_0 = time when replacement services are first provided
s_l = time when replacement services are last provided

P = present time when the natural resource damage claim is presented
i = periodic discount rate.

More comprehensive descriptions of HEA are provided by Chapman et al. (1998), NOAA (1999a, 199b), and Strange et al. (2001).

1.2 Service Losses

The concept of "service" is central to HEA, necessitating clarification of the modeled "service" and how reductions in that service are determined. Habitats typically provide many and various types of ecological services (Strange et al., 2001), including consumptive and nonconsumptive uses to humans, and promotion of ecological sustainability through complex biotic and abiotic interactions (e.g., Holmlund and Hammer, 1999). In some contexts it may be possible to evaluate services in economic or human use terms, for example, when considering recreational beach use. However, restricting the concept of service to human uses will typically overlook many important ecological considerations, including ecological functions, structure, or responses of lesser-known ("low-profile") species.

Moreover, "service," per se, is typically not a measurable quantity. Indeed, ecological service is a term of convenience that may embrace any number of different structural or functional attributes of an ecosystem (Daily, 1997; Limburg and Folke, 1999; Norberg, 1999). Therefore, specific measures must be defined to enable quantification. In a HEA context, selection of the ecological services to be modeled effectively serves to establish an "exchange rate" used in determining the amount of habitat that must be restored to compensate for some harm to the environment. Thus, equivalency between the lost and restored habitat is gauged through the level of services provided by the habitats, which can include human use services, nonconsumptive use (passive use) services, and ecological services. In HEA, the focus often is on ecological services, and by restoring ecological services the other services are also restored.

Depending on the context, services may be quantified directly or indirectly. Determining the number of acres of a particular habitat type and determining percent vegetative cover are examples of direct quantification. In contrast, indirect quantification is used when a habitat is known to be contaminated with hazardous substances that are toxic to one or more species residing in the habitat (Cairns and Neiderlehner, 1994). In this report we focus on the latter, indirect quantification of service loss and propose an approach to associating service losses with toxicological benchmark data.

2. Proposed Framework

2.1 Relating Toxic Effects to Service Loss

We suggest a general framework for addressing the problem of ecological service losses, with particular attention on the problems of integrating information about multiple types of toxicity endpoints. For ease of discussion, we use the term "residual services" to represent the amount of services provided by a habitat after suffering service losses due to one or more causes. Thus,

$$\text{Residual Service (\%)} = 100\% - \text{Service Losses (\%)}. \qquad \text{(Eqn. 2)}$$

In evaluating effects of contaminants of biota, we believe that HEA models should accommodate the possibility of multiple degrees of service loss and that the degree of loss should be linked to conditions (e.g., contaminant concentrations) that are associated with various types of physiological responses in individual organisms. Therefore, responses that are patently detrimental to individuals, such as mortality or gross deformities, should be associated with higher degrees of service loss, while intermediate degrees of service loss may be associated with sublethal physiological responses (Figure 1). For example, stimulation of enzyme pathways that is known to occur in response to contaminant exposure may represent a relatively minor service loss. However, the fact that an organism was induced to expend energy to engage in a detoxification process is an indicator that the habitat is not providing full service; we need not assume that stimulation of an enzyme pathway is a precursor to death. Note that the independent variable in this relationship is expressed as physiological outcome, which is presumed to arise from adverse habitat condition(s), and may or may not have a simple correlation with a single stressor. Thus, contaminants (or other stressors) cause service losses through their adverse effects on physiological processes.

Many contaminants express different types of toxic responses (e.g., mortality, growth, behavioral, biochemical effects) which, typically, are manifested in a nonlinear dose-dependent fashion. Conceptually, therefore, for each of these distinct responses there is some dose-response relationship that associates the degree of that effect with an exposure concentration (Figure 2).

Furthermore, organisms can suffer different types of toxicological effects at different exposure concentrations. Therefore, a cumulative response profile for an organism should consider both the nature and the severity of responses for each exposure. Finally, because organisms may be exposed to more than a single contaminant, evaluation of toxicity requires consideration of those response profiles across multiple compounds where responses can be considered to be completely independent, additive, synergistic, or antagonistic.

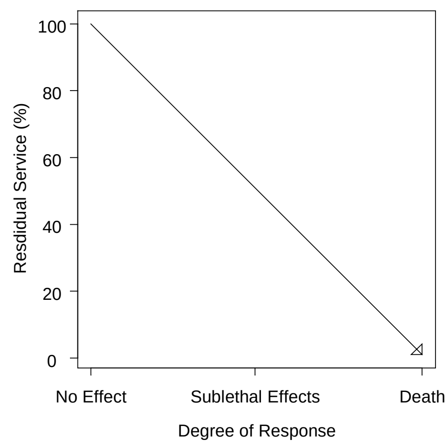

Figure 1. Conceptual relationship between ecological services provided by contaminated habitats and various degrees of toxic responses.

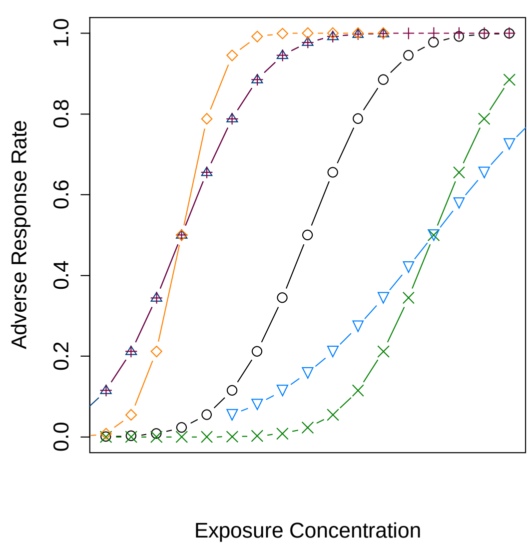

Figure 2. Examples of dose-response curves for different hypothetical responses, each modeled as sigmoidal functions with differing slope and location.

For each of the distinct concentration-response relationships illustrated in Figure 2, a parallel concentration-service loss relationship can be constructed, in which increasing exposure concentration is associated with decreasing residual service (Figure 3). We use the term "mapping" to describe the translation of information about the action of a particular stressor, e.g., elevated concentrations of a hazardous substance, to an index of percent service loss. Because a variety of toxicity endpoints and ecosystem services could be evaluated — and the nature and severity of adverse responses can differ across contaminants and ecological receptors — a variety of general relationships between physiological response and service losses are conceivable (Figure 4).

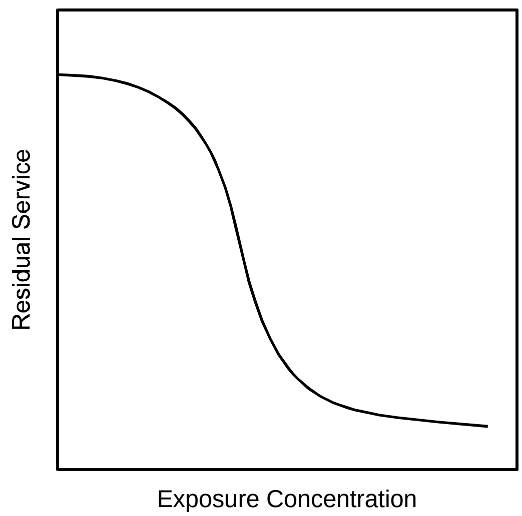

Figure 3. Theoretical "dose-response" relationships between exposure concentration and residual ecological service provided by a contaminated habitat. Relationship is based on mapping responses for a single toxicity endpoint from Figure 2 into conceptual relationship presented in Figure 1.

We suggest that an appropriate definition of percent service loss from an adverse impact to an ecosystem should include reflection of various features associated with the nature of adverse toxicological responses, including:

- the type and severity of the effect(s)
- the degree of the effect
- the extent of the effect(s)
- the organizational level(s) at which the adverse effect occurs (subcellular to ecosystem).

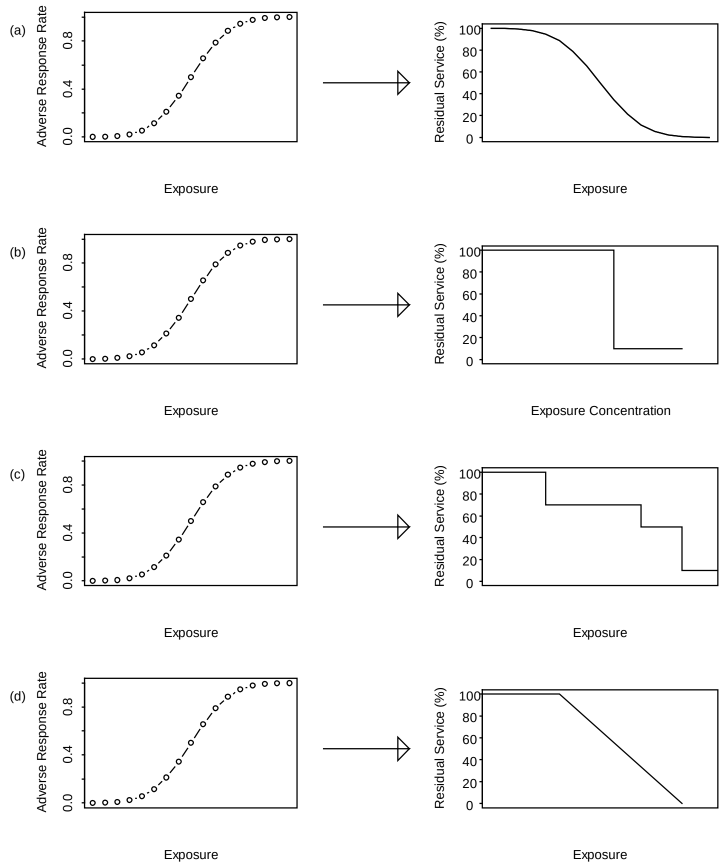

Figure 4. Schematic of mappings from a single hypothetical dose-response curve to alternative service functions: a) sigmoidal; b) single threshold step-function; c) multiple threshold step-function; d) single threshold with linear decline.

Types of effects include lethality, as well as various sublethal effects such as growth impairment or increased rates of histological abnormalities (Table 1). "Severity of effects" is determined by the likelihood that the effect is an indicator of important physiological disruption such as acute mortality, narcosis, carcinogenesis, or reproductive failure. For example, certain types of tumors may be considered severe if they are known to be precancerous, or if they are known to impact the function of the host tissue. In contrast, an effect might be considered "not severe" if the likelihood of physiological impairment is minimal. "Degree of effect" relates to the extent of physiological impairment across a concentration-response relationship. For example, a fish with extensive gill deformation has a suffered a greater impact than a fish with limited gill deformation. "Extent of effect" addresses the percentage of individuals potentially impaired. Certain effects may be considered less consequential if they occur rarely or in a limited part of a population, but could be more serious (and associated with greater service loss) if they are widespread in a population or occur in greater frequency within an organism.

Table 1. Differences among types and degrees of adverse effects.

Feature	Consideration	Examples of typical hierarchy
Type of effect	What physical or behavioral properties are associated with the stressor?	Lethality > skeletal deformities > enzyme pathway induction
Severity of effect	Are known effects a serious threat?	Lethality > deformed mouth parts > hepatic tumors
Extent of effect	Are effects widespread, either within individuals or in the population?	High probability of effect > low probability of effect Greater extent of effect > limited extent of effect
Organizational level affected	What are the ramifications of the effect?	Ecosystem shifts > population reductions > individual mortality

Finally, the level of organization that is affected could be used to reflect service loss, with effects at the habitat/community level being associated with a higher degree of service loss than effects at the cellular level (Figure 5). However, effects that impair the function of higher levels of biological organization are typically more difficult to identify and are likely to be specific to the particular community or ecosystem in question. There are many possible community effects of severe injuries to individuals, especially as a result of broken food webs or changes in competitive relationships. For example, increased mortality among zooplankton may be an example of an effect that impacts higher levels organization in systems, particularly in aquatic systems where zooplankton represent the major link between primary producers and higher trophic levels (Carpenter and Kitchell, 1993).

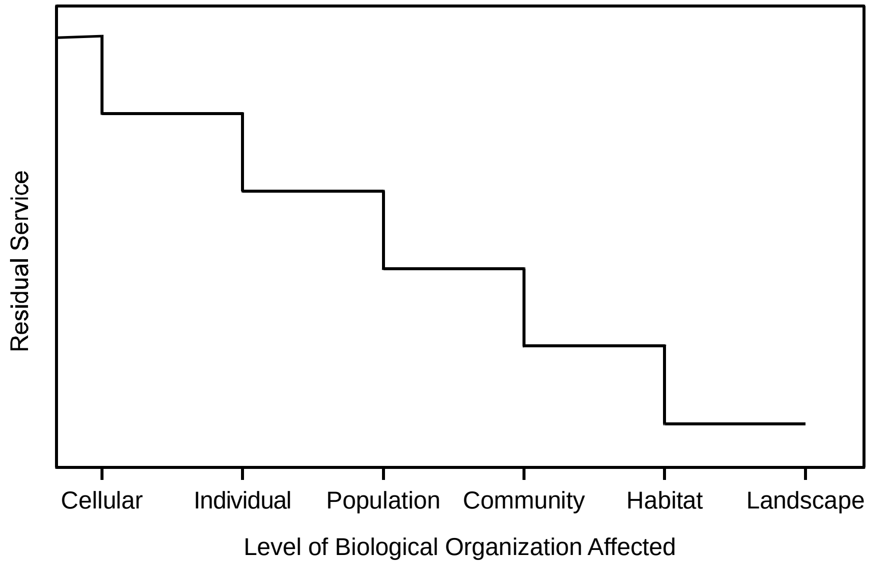

Figure 5. Conceptual relationship between service loss and organizational level of effect.

Some investigators have suggested that ecological services may not be reduced unless there is a measurable reduction in population size (Martin and Richardson, 1995; Attrill and Depledge, 1997). We do not believe that this approach should be used as a "litmus test" in assigning service losses to contaminated habitats. It is often very difficult to detect changes in the size of wild populations (e.g., Wedemeyer et al., 1984; Adams et al., 1993). Changes typically go unnoticed unless the population is monitored because of its commercial importance, or if the change is quite radical, as in the case where the population of a pest species experiences an "explosion" or if a local population of a highly visible species crashes or becomes extinct. Populations fluctuate for many reasons that are independent of hazardous substance concentrations or other qualities of the habitat. Even measurable changes in population size may not be attributable to hazardous substances because very few habitats are in an "equilibrium condition." Indeed, it may be as problematic to demonstrate that an ecosystem was in equilibrium condition as it is to demonstrate that deviations from equilibrium are a result of hazardous substances. Even in situations where hazardous substances were categorically known to cause lethality and subsequent population reductions, it may be practically impossible to detect population changes on a meaningful time scale, especially in long-lived species or in species that have variable sensitivities in different portions of their life history. These features also complicate efforts to assess the success of restoration efforts (Simenstad and Thom, 1996; Miller and Simenstad, 1997).

Therefore, although demonstrable population reductions may suggest higher degrees of service loss assignment, we suggest that less drastic changes also be considered in developing service loss "maps." Because organisms live on a finite energy budget, any stressor that occurs in addition to "natural" (i.e., nonanthropogenic) stressors is detrimental to that organism because the organism must redirect energy to mitigate the effects of the stressor (Rowe et al., 1998). Examples of stress-induced energy redirection include physiological detoxification (e.g., metallothionein induction) and additional caloric expenditures for motility (e.g., due to habitat avoidance behaviors or enlarged home range). Directing energy to these activities must come at the expense of other biological processes, such as growth, reproduction, and avoiding predation. If a particular habitat is more stressful relative to a reference site, it provides less service.

2.2 Using Toxicity Data to Map Service Loss Functions

Because mapping from dose-response curves to service loss functions is not inherently an empirical process, using a predetermined framework provides a rational, reproducible, and transparent way to synthesize available information and use it to define a mapping from empirical data to a service index.

Because of the functional role that ecological service levels play in a HEA model, service reductions are expressed on a percentage basis: the ecological service index of a unit of habitat is reduced by a certain fraction that reflects the difference in habitat quality relative to a reference type. It is relatively simple to identify the extreme conditions on a percent service scale: if a region is rendered uninhabitable it provides 0% service, while an uncontaminated, pristine habitat (intuitively) provides 100% ecological service. In contrast, there is no obvious, intuitive way to quantify the spectrum of conditions that are intermediate to the ideal or the wasteland.

We describe the mapping procedure as a two phase process. The first phase is to associate knowledge about sensitivity of individual toxic response endpoints with a service loss function (e.g., Figure 3). The second step is to integrate service loss due to multiple, possibly independent, stressors into a single value indicating the residual service provided by a unit of habitat (e.g., Figure 5).

An initial attempt to define Phase 1 of the mapping might involve selecting a dose-response curve with a response rate that ranges from 0 to 100% and performing a direct translation of response rate to service loss: a 10% response rate is mapped to 10% service loss, 85% response rate is mapped to 85% service loss, and so on (Figure 4a). This type of mapping function, however, does not consider the nature of the response in question, nor the ramifications of the effect (if any) beyond the individual organism.

Several general classes of mapping are plausible, and different types may be used to describe different stressors within one HEA model (Figure 4). A mapping scheme that is appropriate for lethality responses might not be appropriate for a sublethal response, such as CYP1A induction. In addition to the simple dose-response inverse described above (Figure 4a), some types of endpoints might be best described by a step function, perhaps because of the nature of the supporting evidence or the endpoint itself. A step function may be most appropriate when there is evidence of one or more clear toxicity thresholds and the response endpoint is relatively static otherwise (Figure 4b, 4c). A threshold model can be modified to reflect a graded response for exposures above the threshold (Figure 4d), for example in a case where the dose-response is well described by a hockey stick model.

Phase 2 of the mapping process involves integration of multiple service loss indices (i.e., across different response endpoints) into a single value of residual service for assignment to a particular unit of habitat (Figure 6). This second phase enables consideration of type/severity of effect, degree of effect, extent of effects, and multiple levels of organizational responses. There are several plausible alternative algorithms for integration. We consider two alternatives to be the most appealing, which we term the "minimum" and "multiplicative" models. The minimum model simply assigns to a unit of habitat the smallest residual service value associated with any of the stressors and endpoints considered. Under the multiplicative model, residual service is defined as the product of the residual service values associated with any of the stressors and endpoints considered (Figure 6). The multiplicative model will typically yield lower values of residual service because each additional stressor considered may cause a reduction in residual service, whereas under the minimum model consideration of additional stressors does not necessarily change the resulting residual service values. We believe that the multiplicative model may be more appropriate because it considers all types of evidence of adverse impacts.

The end product of this mapping process is a single relationship between contaminant exposure and residual service (graph in bottom-right of Figure 6). This relationship is used to quantify services in the HEA model (Eqn. 1).

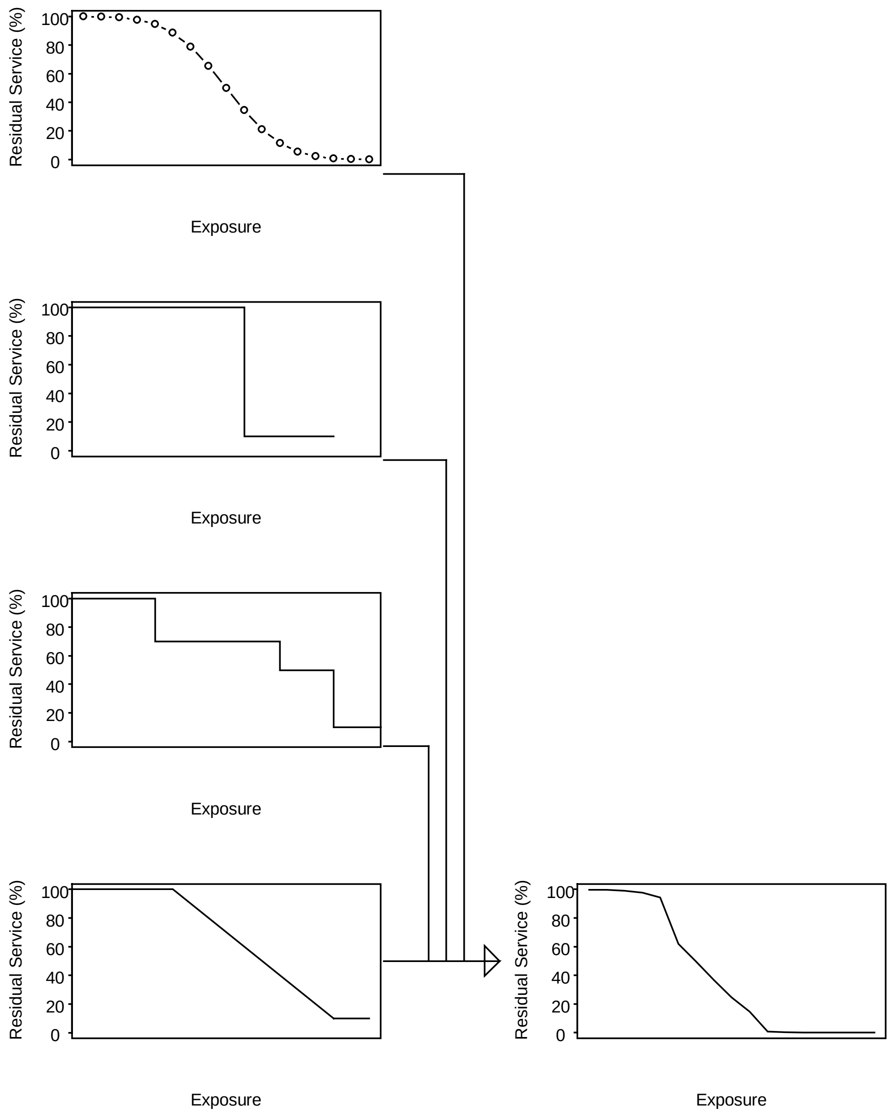

Figure 6. Schematic of hypothetical service loss functions associated with multiple independent adverse response endpoints (depicted in Figure 4) integrated into a single service loss function (multiplicative example; see text for details).

3. Case Study: An Estuary Contaminated with PAH

To illustrate this proposed framework, we provide a brief case-study example of a hypothetical HEA that considers the effects of hazardous substances on the ecological services provided by the Hylebos Waterway, a shallow estuarine embayment of Puget Sound. Sediments in the waterway are contaminated with varying concentrations of PAH.[1]

We begin by determining the community composition of the waterway and focusing on species that are predominant, are considered to be either particularly at risk of PAH toxicity, or could serve as indicator species.

We focus on flatfish and invertebrates in this example. Flatfish are resident in the Hylebos Waterway and may be exposed to PAHs by various routes or modalities, including:

- dietary ingestion of contaminated prey
- incidental ingestion of contaminated sediments
- transdermal exposure due to prolonged direct contact with contaminated sediments.

The invertebrate community is also of particular concern because they are exposed to sediment contaminants, including PAH, and because they function as an important part of the estuarine food web. As such, if invertebrates are impacted the community as a whole may be at risk and the ecological services provided by the habitat are reduced.

The objective in the assessment of service loss is to interpret the pertinent information about the kinds of toxic effects associated with various concentrations of PAH and to use that information to develop a reasonable basis for relating ambient PAH concentrations to reductions in ecological services.

Information about the toxicity of PAH to estuarine species is available from various sources. Each source provides the results of experiments in which various organisms were exposed by various modalities, and the researchers considered a variety of lethal and sublethal endpoints. Casillas et al. (1991) found evidence that elevated sediment PAHs (and/or PCBs) were associated with reproductive physiology in English sole, and other studies (e.g., Malins et al., 1984, 1988) found associations between PAH exposure and hepatic lesions in English sole.

1. We consider only one contaminant as an example of how we might assess service losses. In fact, the Hylebos Waterway is contaminated by numerous hazardous substances in addition to PAHs, and flatfish are not the only species of concern that attempt to reside there.

Here we consider two particular sources of information about PAH toxicity. In principle, all relevant studies could be considered, but a complete treatment is outside the scope of this report. The first data we consider are experiments conducted by NOAA (Johnson, 1999) that examined the relationship between sediment PAH exposure, the prevalence of various kinds of hepatic lesions, and indicators of reproductive success. The second source is a suite of apparent effects thresholds (AETs) associated with various adverse outcomes in marine invertebrates including echinoderms, oysters, *neanthes,* and amphipods. The English sole studies (Johnson, 1999) identified 1 ppm PAH as an important threshold above which the prevalence of hepatic lesions rises. The studies indicated that 1 ppm PAH is also the threshold above which the rates of various types of reproductive effects increased. AETs have been recognized by the State of Washington as valid sediment quality guidelines, and therefore are considered to be particularly relevant for the vicinity. These guidelines are particularly relevant to our analysis because they are specific to benthic invertebrates.

We assembled the various critical thresholds identified by these sources and ranked them according to the principles described above, namely, the type, severity, and extent of impact that could be expected in regions of the waterway where PAH concentrations exceed the published thresholds (Table 2). In this case, our ranking of adverse outcomes consistently corresponds to increasing sediment PAH concentrations. We believe it is appropriate in this case because a) the responses in sole are clearly of increasing severity, and b) the various invertebrate AET thresholds suggest the possibility of important food web disruptions.

The data in Table 2 then were used to develop a mapping of the toxicity data to a service loss function (Figure 7). We associate the highest PAH concentrations with 20% residual services. We elected not to associate high PAH concentrations with 0% residual services for several reasons. None of the data sources described either acute or chronic mortality of English sole, so we propose that highly contaminated sediments continue to provide residual services despite the likelihood of substantially elevated stress. The types of residual services provided by habitats contaminated by 100 ppm PAH include production of resistant invertebrates (which serve as forage) and as spawning habitat of marginal quality. A 60% residual service level was used to describe PAH concentrations associated with sole infertility (23% incidence), sole histological lesions (40% incidence), and exceedence of AETs for oysters and microtox. An 80% residual service level was used to describe lower incidences of impacts to English sole and exceedence of the echinoderm AET. Thus, the service loss mapping illustrated in Figure 5 associates increasing service losses in the case study area with higher PAH concentrations, which in turn, are associated with greater incidence of toxicity and with exceedence of more toxic endpoints. Therefore, we believe that this service loss mapping represents a reasonable approach to relating toxicity data on PAH with the residual ecological services provided by contaminated habitats.

Table 2. Selected evidence of toxic effects of sediment PAH on benthic organisms.

Receptor	Effect	Sediment PAH concentration (ppm, dry weight)	Reference
Echinoderm	AET	4.65	Washington State Sediment Quality Guidelines
Oyster	AET	29.8	Washington State Sediment Quality Guidelines
Microtox	AET	46.5	Washington State Sediment Quality Guidelines
Neanthes	AET	74.4	Washington State Sediment Quality Guidelines
Amphipod	AET	164.3	Washington State Sediment Quality Guidelines
Benthic Community	AET	235.6	Washington State Sediment Quality Guidelines
English sole	9% with 1 or more lesions	1	Johnson (1999)
English sole	4% infertile above baseline	1	Johnson (1999)
English sole	18% with 1 or more lesions	2	Johnson (1999)
English sole	10% infertile above baseline	2	Johnson (1999)
English sole	24% with 1 or more lesions	3	Johnson (1999)
English sole	13% infertile above baseline	3	Johnson (1999)
English sole	31% with 1 or more lesions	5	Johnson (1999)
English sole	17% infertile above baseline	5	Johnson (1999)
English sole	40% with 1 or more lesions	10	Johnson (1999)
English sole	23% infertile above baseline	10	Johnson (1999)
English sole	71% with 1 or more lesions	100	Johnson (1999)
English sole	42% infertile above baseline	100	Johnson (1999)

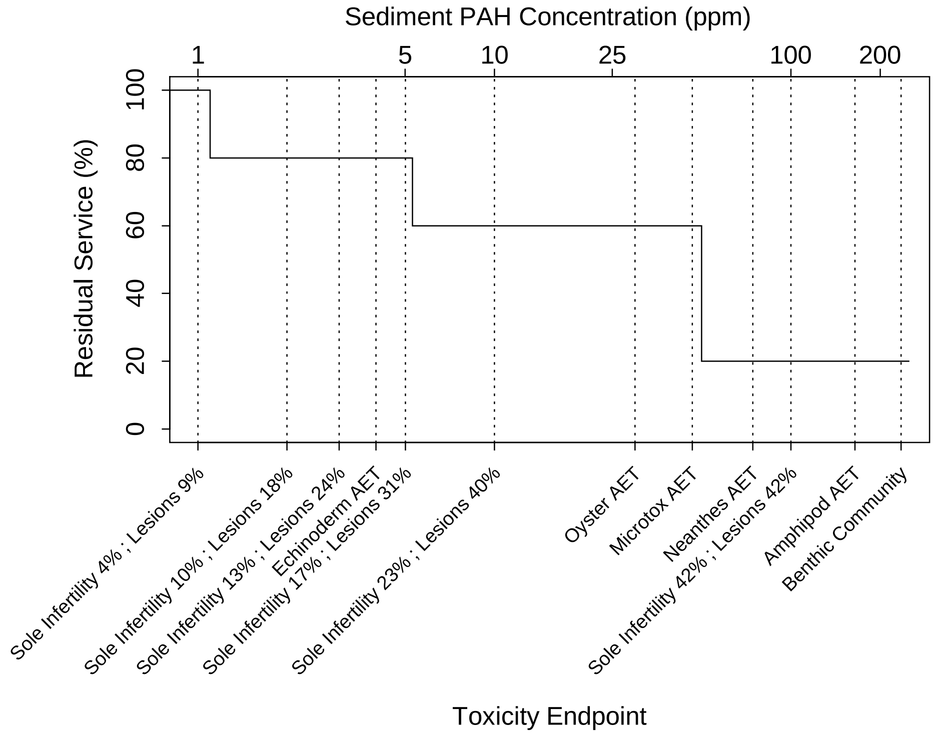

Figure 7. Hypothetical service loss function relating levels of toxic effects of PAH in various organisms to services provided by contaminated estuarine sediments.

4. Conclusion

The framework described herein is intended for use in situations in which HEA methodologies are applied at sites where contamination by hazardous substances is believed to be toxic to biota and therefore injurious to the ecosystem. We recommend development of transparent relationships between service loss that incorporates information on the nature and extent of toxicological endpoints. Although the general framework that we describe requires professional judgment with respect to features relevant to a particular analysis, by laying out important principles that should be followed we believe it serves as a useful guide for analysts who wish to apply toxicological considerations in a HEA.

References

Adams, S.M., G.F. Cada, M.S. Greeley, Jr., and L.R. Shugart. 1993. Evaluating effects of environmental stress using multiresponse indicators. In *Environmental Analysis: The NEPA Experience*, S.G. Hildebrand and J.B. Cannon (eds.). Lewis Publishers, Chelsea, MI, pp. 170-177.

Attrill, M.J. and M.H. Depledge. 1997. Community and population indicators of ecosystem health: targeting links between levels of biological organization. *Aquatic Toxicology* 38:183-197.

Bishop, R.C., W.S. Breffle, J.K. Lazo, R.D. Rowe, and S.M. Wytinck. 2000. Restoration Scaling Based on Total Value Equivalency: Green Bay Natural Resource Damage Assessment. Prepared for the U.S. Fish & Wildlife Service by Stratus Consulting, Boulder, CO. October 25.

Breffle, W.S., E.R. Morey, R.D. Rowe, and D.M. Waldman. Forthcoming. Combining stated-choice questions with observed behavior to value NRDA compensable damages: A case study of recreational fishing in Green Bay and the Lower Fox River. In *The Handbook of Contingent Valuation*, D. Bjornstad, J. Kahn, and A. Alberini (eds.). Edward Elgar Publishing, Northampton, MA.

Cairns, J. and B.R. Neiderlehner. 1994. Estimating the effects of toxicants on ecosystem services. *Environmental Health Perspectives* 102(11):936-939.

Carpenter, S.R. and J.F. Kitchell (eds.) 1993. *The Trophic Cascade in Lakes*. Cambridge Univ. Press, Cambridge, UK.

Casillas, E., D. Misitano, L.L. Johnson, L.D. Rhodes, T.K. Collier, J.E. Stein, B.B. McCain, and U. Varanasi. 1991 Inducibility of spawning and reproductive success of female English sole *(Parphyrs vetulus)* from urban and nonurban areas of Puget Sound, Washington. *Marine Environmental Research* 31:99-122.

Chapman, D., N. Iadanza, and T. Penn. 1998. Calculating Resource Compensation: An Application of the Service-to-Service Approach to the Blackbird Mine Hazardous Waste Site. NOAA Damage Assessment and Restoration Program Technical Paper 97-1.

Clements, W.H. and P.M. Kiffney. 1994. Assessing contaminant effects at higher levels of biological organisation. *Environmental Toxicology and Chemistry* 13:357-359.

Daily, G.C. 1997. *Nature's Services: Societal Dependence on Natural Ecosystems*. Island Press, Washington, DC.

Holmlund, C.M. and M. Hammer 1999. Ecosystem services generated by fish populations. *Ecological Economics* 29:253-268.

Johnson, L. 1999. Biological Effects of Polycyclic Aromatic Hydrocarbons (PAHs) on English Sole: An Analysis in Support of Sediment Quality Guidelines to Protect Marine Biota. NOAA Technical Report.

Knuth, B.A., N.A. Connelly, and M.A. Shapiro. 1993. Angler Attitudes and Behavior Associated with Ohio River Health Advisories. Prepared by the Human Dimensions Research Unit of the Department of Natural Resources of the New York State College of Agriculture and Life Sciences. HDRU Series No. 93-6.

Limburg, K.E. and C. Folke. 1999. The ecology of ecosystem services: Introduction to the special issue. *Ecological Economics* 29:179-182.

Malins, D.C., B.B. McCain, J.T. Landahl, M.S. Meyers, M.M. Krahn, D.W. Brown, S.L. Chan, and W.T. Roubal. 1988. Neoplastic and other diseases in fish in relation to toxic chemicals — an overview. *Aquatic Toxicology* 11:43-67.

Malins, D.C. , B.B. McCain, D.W. Brown, S.L. Chan, M.S. Meyers, J.T. Landahl, P.G. Prohaska, A.J. Friedman, L.D. Rhodes, D.G. Burrows, W.D. Gronlund, and H.O. Hodgins. 1984. Chemical-pollutants in sediments and diseases of bottom-dwelling fish in Puget Sound, Washington. *Environmental Science and Technology* 18:705-713.

Martin, M. and B.J. Richardson. 1995. A paradigm for integrated marine toxicity research? Further views from the Pacific Rim. *Marine Pollution Bulletin* 30:8-13.

Miller, J.A. and C.A. Simenstad. 1997. A comparative assessment of a natural and created estuarine slough as rearing habitat for juvenile chinook and coho salmon. *Estuaries* 20:792-806.

NOAA. 1999a. Discounting and the Treatment of Uncertainty in Natural Resource Damage Assessment. NOAA Damage Assessment and Restoration Program Technical Paper 99-1.

NOAA. 1999b. Habitat Equivalency Analysis: An Overview. NOAA Damage Assessment and Restoration Program (unpublished).

Norberg, J. 1999. Linking nature's services to ecosystems: Some general ecological concepts. *Ecological Economics* 29:183-202.

Rowe, C.L. and J. Freda. 2000. Effects of acidification on amphibians at multiple levels of biological organization. In *Ecotoxicology of Amphibians and Reptiles*, D.W. Sparling, G. Linder, and C.A. Bishop (eds.). SETAC Press, Pensacola, FL.

Rowe, C.L., O.M. Kinney, R.D. Nagle, and J.D. Congdon. 1998. Elevated maintenance costs in an Anuran (*Rana catesbeiana*) exposed to a mixture of trace elements during the embryonic and early larval periods. *Physiological Zoology* 71(1):27-35.

Rowe, R.D., W.D. Schulze, W.D. Shaw, D. Schenk, and L.G. Chestnut. 1991. Contingent Valuation of Natural Resource Damage due to the Nestucca Oil Spill, Final Report. Prepared for Department of Wildlife, State of Washington, Olympia WA; British Columbia Ministry of Environment, Victoria, BC; Environment Canada, Vancouver, BC. June 15.

Simenstad, C.A. and R.M. Thom. 1996. Functional equivalency trajectories of the restored Gog-Le-Hi-Te estuarine wetland. *Ecological Applications* 6:38-56.

Strange, E., H. Galbraith, S. Bickel, D. Mills, D. Beltman, and J. Lipton. 2001. Determining ecological equivalence in service-to-service scaling of salt marsh restoration. Submitted: *Environmental Management*.

Wedemeyer, G.A., D.A. McLeay, and C.P. Goodyear 1984. Assessing the tolerance of fish and fish populations to environmental stress: the problems and methods of monitoring. In *Contaminant Effects on Fisheries*, V.W. Cairns, P.V. Hodson, and J.O. Nriagu (eds.). John Wiley & Sons, New York, pp. 163-196.