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Pollutants of Concern in Puget Sound

PTI Environmental Services, Bellevue, WA

Prepared for:
Environmental Protection Agency, Seattle, WA

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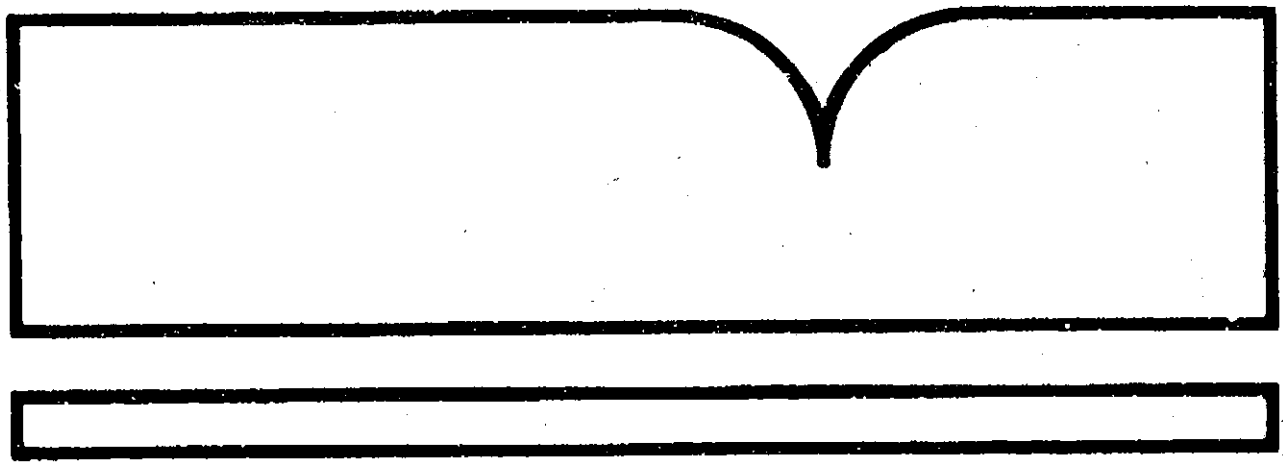

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POLLUTANTS OF CONCERN IN PUGET SOUND

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This report replaces and updates information originally compiled for the 1986 User's Manual for the Pollutant of Concern Matrix. The information in this report is available only in hard copy, not in a spreadsheet format as in 1986.

The report contains the following information for 64 pollutants:

Table I	Regulatory status and analytical considerations
Table II	Criteria, guidelines and regulatory action levels
Table III	Sources of pollutants
Table IV	Concentrations of pollutants in Puget Sound

The report also contains a narrative section on each pollutant, describing exposure routes and risks, sources and fate in the environment, and other information.

The information contained in this report is useful to permit writers and reviewers, resource managers, writers and reviewers of environmental impact statements and others.

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POLLUTANTS OF CONCERN IN PUGET SOUND

Prepared for

U.S. Environmental Protection Agency
Office of Puget Sound
Region 10
Seattle, Washington

EPA Contract 68D80085
PTI Contract C744-15

April 1991

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LIST OF ACRONYMS

AET	apparent effects threshold
CFR	Code of Federal Regulations
CLP	Contract Laboratory Program
Corps	U.S. Army Corps of Engineers
CSO	combined sewer overflow
DW	dangerous waste
Ecology	Washington Department of Ecology
EHW	extremely hazardous waste
EPA	U.S. Environmental Protection Agency
FDA	U.S. Food and Drug Administration
HCB	hexachlorobenzene
HCBD	hexachlorobutadiene
HCH	hexachlorocyclohexane
HPAH	high molecular weight polycyclic aromatic hydrocarbons
LPAH	low molecular weight polycyclic aromatic hydrocarbons
MCLGs	maximum contaminant level goals
MCLS	maximum contaminant levels
MEK	Methylethyl ketone
Metro	Municipality of Metropolitan Seattle
NOAA	National Oceanic and Atmospheric Administration
NURP	National Urban Runoff Program
PCBs	polychlorinated biphenyls
PCDDs	dibenzo-*p*-dioxins
PCDFs	polychlorinated dibenzofurans
PCP	pentachlorophenol
PNELs	probable no-effect levels
PPM	parts per million
PPB	parts per billion
PSDDA	Puget Sound Dredged Disposal Analysis
PSEP	Puget Sound Estuary Program
RfD	reference dose
QA/QC	quality assurance and quality control
SEDQUAL	sediment quality database
SLCs	screening level concentrations
TBT	tributyltin
2,3,7,8-TCDD	2,3,7,8-tetrachlorodibenzo-*p*-dioxin
TPPS	Toxicant Pretreatment Planning Study

ACKNOWLEDGMENTS

This document replaces and provides updated information originally compiled for the 1986 *Pollutant of Concern Matrix*, which was prepared by Tetra Tech, Inc. for the U.S. Environmental Protection Agency (EPA) in partial fulfillment of Contract No. 68-03-1977. The current document was prepared by PTI Environmental Services in partial fulfillment of EPA Contract No. 68D80085. Ms. Sally Hanft of EPA was the Project Officer and Dr. John Armstrong of EPA was the Project Monitor for this document.

Primary authors of this report were Ms. Kimberly Henson, Ms. Teresa Michelsen, Mr. Wayne Clark, Ms. Lorraine Read, and Ms. Jennifer Sampson.

EXECUTIVE SUMMARY

Recent studies by federal, state, and local agencies have identified adverse biological conditions associated with contaminants in some areas of Puget Sound. Permit writers, resource managers, reviewers of environmental impact statements, and others involved in environmental decision-making are faced with the task of assessing a wide variety of information to deal with the pollution problems. To address the needs of this diverse group of data users, information on each of 64 pollutants is summarized in the following tables:

Table I:	Regulatory Status and Analytical Considerations for Pollutants of Concern
Table II:	Criteria, Guidelines, and Regulatory Action Levels for Pollutants of Concern
Table III:	Sources of Pollutants
Table IV:	Concentrations of Pollutants in Puget Sound.

In addition, characteristics of these pollutants are summarized in the text of the report. A general description of the pollutants is provided as well as a brief comment on exposure routes and risks and sources and fate in the environment (including soil, submerged sediment, air, and water).

1. INTRODUCTION

Background information on the development of this report and criteria used to select pollutants of concern are described in the following sections. Explanations of column headings for each of four tables of information on these pollutants of concern and examples of how to interpret the data for one chemical [i.e., benzo(a)pyrene] are provided in Chapter 2 (*Pollutants of Concern Tables*). More detailed text descriptions of each pollutant of concern are provided in Chapter 3 (*Description of Pollutants of Concern in Puget Sound*).

BACKGROUND

Recent studies by federal, state, and local agencies have found that significant adverse biological conditions are associated with contaminated sediments in some areas of Puget Sound (i.e., Commencement Bay, Elliott Bay, Eagle Harbor, Sinclair Inlet, Everett Harbor, and the Duwamish River). These studies have been performed by or for the U.S. Environmental Protection Agency (EPA), Washington Department of Ecology (Ecology), National Oceanic and Atmospheric Administration (NOAA), U.S. Army Corps of Engineers (Corps) Seattle District, and Municipality of Metropolitan Seattle (Metro). A limited number of pollutants of concern were listed as part of an EPA Region 10 project to quantify chemical loadings into Puget Sound (Jones & Stokes 1983). This list was circulated for review to the Technical Advisory Committee and the Management Committee of the Puget Sound Estuary Program (PSEP). Review comments from Ecology suggested development of a broader list of pollutants. This broader list was developed into the *Users Manual for the Pollutant of Concern Matrix* (PSEP 1986). The objective of this report is to update, expand, and reorganize the 1986 manual based on the results of a survey of users conducted in January 1990. Instructions on the use of electronic spreadsheet tables and diskettes containing these tables were provided with the 1986 manual but have been deleted for this report because most users preferred using written copies of the tables (see Chapter 2, *Pollutants of Concern Tables*).

The information summarized in the pollutant tables and the subsequent descriptive text in Chapter 3 can be used as a reference for permit writers, reviewers, and inspectors when evaluating discharges from new or existing industrial and municipal facilities; an aid for the design and execution of field investigations and monitoring efforts; and a resource for agency personnel in evaluating environmental conditions and potential impacts of pollutants on Puget Sound. Examples of recent Puget Sound studies that have been used to update

this report include urban bay investigations completed by EPA in 1988 for Elliott Bay and Everett Harbor, sediment data from the 1989 and 1990 Puget Sound Ambient Monitoring Program, and class II inspection surveys of industrial and municipal effluents conducted by Ecology between 1987-1990.

SELECTION OF THE POLLUTANTS OF CONCERN

In 1986, an initial list of over 100 inorganic and organic contaminants of potential concern in Puget Sound (Tables 1 and 2) was compiled for possible inclusion in the pollutants of concern list. These contaminants were chosen from 1) EPA's list of priority pollutants, 2) lists compiled specifically for Puget Sound (e.g., Konasewich et al. 1982; Quinlan et al. 1985; Jones & Stokes 1983), 3) PSEP and Puget Sound Dredged Disposal Analysis (PSDDA) workshops held to establish procedures for environmental analysis of inorganic and organic contaminants (PSEP 1989a,b), and 4) from field investigations in Puget Sound (e.g., Gahler et al. 1982; Malins et al. 1980; Romberg et al. 1984; Tetra Tech 1985a). Experts in specific fields of chemical research also provided advice during preparation of the initial list.

In addition to the individual compounds initially considered, three groups of compounds were recommended: high molecular weight polycyclic aromatic hydrocarbons (HPAH), low molecular weight polycyclic aromatic hydrocarbons (LPAH), and total polychlorinated biphenyls (PCBs) (rather than individual Aroclors or PCB congeners). The 1986 pollutants of concern list included 52 contaminants or groups of contaminants selected from this initial list of 100 chemicals.

Twelve additional contaminants or groups of contaminants have been added to the text and tables to accommodate responses from the recent survey of the 1986 manual users. These new contaminants were recommended based on worker safety, treatment plant operation, toxicity, and water quality considerations. The following four EPA priority pollutants were added: bis(2-ethyl)-hexylphthalate, benzene, toluene, and total xylenes. In addition, the following eight groups of pollutants that are not EPA priority pollutants were added:

- Mono- and di-chlorodehydroabietic acids (resin acids found in pulp mill discharges)

TABLE 1. INORGANIC CONTAMINANTS OF POTENTIAL CONCERN IN PUGET SOUND

Antimony	Copper	Silver
Arsenic	Lead	Zinc
Cadmium	Mercury	Cyanide
Chromium	Nickel	Organotins

TABLE 2. ORGANIC CONTAMINANTS OF POTENTIAL CONCERN IN PUGET SOUND

Phenols					
65*	Phenol			HSL	4-Methylphenol
HSL*	2-Methylphenol			34	2,4-Dimethylphenol
Substituted Phenols					
24	2-Chlorophenol	21	2,4,6-Trichlorophenol	57	2-Nitrophenol
31	2,4-Dichlorophenol	HSL	2,4,5-Trichlorophenol	59	2,4-Dinitrophenol
22	4-Chloro-3-methylphenol	64	Pentachlorophenol	60	4,6-Dinitro-o-cresol
Miscellaneous Organic Acids (guaiacols/resin acids)					
2-Methoxyphenol (guaiacol)		4,5,6-Trichloroguaiacol		Monochlorodehydroabietic acids	
3,4,5-Trichloroguaiacol		Tetrachloroguaiacol		Dichlorodehydroabietic acids	
LPAH Compounds					
55	Naphthalene	1	Acenaphthene	81	Phenanthrene
77	Acenaphthylene	80	Fluorene	78	Anthracene
Alkylated LPAH Compounds					
2-Methylnaphthalene		1-Methylnaphthalene		1,2,3-Methylphenanthrenes	
HPAH Compounds					
39	Fluoranthene	74	Benzo(b)fluoranthene	83	Indeno(1,2,3-cd)pyrene
84	Pyrene	75	Benzo(k)fluoranthene	82	Dibenzo(a,h)anthracene
72	Benzo(a)anthracene	73	Benzo(a)pyrene	79	Benzo(g,h,i)perylene
76	Chrysene				
Chlorinated Aromatic Hydrocarbons					
26	1,3-Dichlorobenzene			8	1,2,4-Trichlorobenzene
27	1,4-Dichlorobenzene			20	2-Chloronaphthalene
25	1,2-Dichlorobenzene			9	Hexachlorobenzene (HCB)

Table 2. (Continued)

Chlorinated Aliphatic Hydrocarbons					
12	Hexachloroethane			52	Hexachlorobutadiene
Phthalates					
71	Dimethylphthalate	68	Di-n-butylphthalate	69	Di-n-octylphthlate
70	Diethylphthalate	67	Butylbenzylphthalate		
Miscellaneous Oxygenated Compounds					
54	Isophorone	HSL	Dibenzofuran		Polychlorinated Dibenzodioxins
HSL	Benzyl alcohol		Polychlorinated dibenzofurans		
HSL	Benzoic acid				
Organonitrogen Compounds					
82	N-Nitrosodiphenylamine		9(H) - Carbozole		
Pesticides					
93	p,p'-DDE	90	Dieldrin	102	α-HCH
94	p,p'-DDD	91	α-chlordane	103	β-HCH
92	p,p'-DDT	98	Endrin	104	Δ-HCH
89	Aldrin	100	Heptachlor	105	γ-HCH (lindane)
PCBs					
Total PCBs[a]					
Volatile Halogenated Alkanes					
45	Chloromethane	23	Chloroform	32	1,2-Dichloropropane
46	Bromomethane	10	1,2-Dichloroethane	51	Chlorodibromomethane
16	Chloroethane	11	1,1,1-Trichloroethane	14	1,1,2-Trichloroethane
44	Dichloromethane	6	Carbon tetrachloride	47	Bromoform
13	1,1'-Dichloroethane	48	Bromodichloromethane	15	1,1,2,2-Tetrachloroethane

Table 2. (Continued)

		Volatile Halogenated Alkenes			
88	Vinyl chloride	33	Cis-1,3-dichloropropene	87	Trichloroethene
29	1,1'-Dichloroethene		Trans-1,3-dichloropropene	85	Tetrachloroethene
30	Trans-1,2-dichloroethene				
		Volatile Aromatic and Chlorinated Aromatic Hydrocarbons			
4	Benzene	38	Ethylbenzene	HSL	Total xylenes
86	Toluene	HSL	Styrene	7	Chlorobenzene

[a] Indicates EPA priority pollutant number.

[b] EPA hazardous substance list (HSL) compound.

[c] Total PCBs includes monochlorobiphenyls through decachlorobiphenyls.

- Chlorinated guaiacols (associated with bleached discharges of pulp mills)
- Three of the most toxic PCB congeners that elicit adverse biological effects similar to those of 2,3,7,8-tetrachlorodibenzodioxin
- Polychlorinated dibenzofurans
- Polychlorinated dibenzodioxins (in addition to 2,3,7,8-tetrachlorodibenzodioxin)
- Methylethyl ketone (MEK)
- Methylnaphthalenes (1-methyl and 2-methyl isomers)
- Methylated phenanthrenes.

Pollutants that are not included in this report but have been recommended for routine monitoring based on a recently completed pesticide reconnaissance survey (PSEP 1991) include: diazinon in water and sediment (an organophosphate insecticide often used for control of fruit, vegetable, and ornamental foliage pests), diuron in water (a uracil herbicide used for sterilizing soils), and endosulfan I in sediment (a chlorinated pesticide used for control of foliar feeding insects on a wide variety of plants). These three pesticides were detected in samples of sediment or waters in drainage basins of Puget Sound at levels that could cause biological effects (PSEP 1991). The reconnaissance survey was completed after the pollutant tables had been completed. Eight other pesticides were detected in samples collected during this reconnaissance survey but at concentrations that are of less concern. The detected pesticides are part of a broad group of organophosphate, chlorinated, polar phosphorous, carbamate, and urea pesticides, and chlorinated and triazine herbicides that were tested for in these samples. These compounds had been identified as of potential concern in Puget Sound based on an earlier review of contemporary pesticide usage (PSEP 1988).

Contaminants selected as pollutants of concern for this report meet all of the following general criteria: 1) high toxicity (measured in laboratory studies), 2) high persistence in the environment, 3) high bioaccumulation potential. In addition, pollutants of concern meet one or more of the following specific criteria for Puget Sound: 4) high measured water column or effluent concentration, 5) existence of known sources, 6) high concentration relative to sediments from Puget Sound reference areas, and/or 7) widespread distribution in Puget Sound. The last two criteria were evaluated using data from over 1,000 sediment samples that have been incorporated into EPA's sediment quality database (SEDQUAL) for Puget Sound. Some extremely toxic chemicals that are supported by few environmental data in Puget Sound are nevertheless included as pollutants of

concern because of sufficient public or agency concern over their potential impacts. For example, available data on chlorinated dibenzodioxins, chlorinated dibenzofurans, and chlorinated guaiacols are summarized even though information is available on their distribution in only a few areas of Puget Sound. Inclusion of these chemicals with few data indicates gaps in present knowledge of toxic pollutants in Puget Sound.

2. POLLUTANTS OF CONCERN TABLES

The following four pollutants of concern tables are provided in this chapter:

Table I:	Regulatory Status and Analytical Considerations for Pollutants of Concern
Table II:	Criteria, Guidelines, and Regulatory Action Levels for Pollutants of Concern
Table III:	Sources of Pollutants
Table IV:	Concentrations of Pollutants in Puget Sound.

The information provided in each column of the tables, in addition to associated references, is also described.

TABLE I. REGULATORY STATUS AND ANALYTICAL CONSIDERATIONS FOR POLLUTANTS OF CONCERN

The regulatory status of each contaminant and general analytical considerations are reviewed in Table I.

Column 1—EPA identified 65 categories of priority pollutants (including 126 specific chemical substances to be the focus for regulation under the Clean Water Act). Because of their status as priority pollutants (identified by P, T, and N in column 1), these chemicals are most frequently analyzed by contract laboratories participating in EPA Superfund work.

The list of pollutants is found in the Code of Federal Regulations (CFR) Title 40, Part 401.15. The complete list of inorganic and organic chemicals analyzed by the EPA Contract Laboratory Program (CLP) is found in U.S. EPA (1990a,b).

Columns 2, 3, and 4—Analytical methods for water, sediment, and tissue samples are listed in columns 2, 3, and 4. PSEP has recommended guidelines for analysis of many toxic pollutants, including most EPA-designated priority pollutants. For some pollutants, existing EPA methods were adopted by PSEP

(e.g., metals in water). At least one class of pollutants on the pollutants of concern list (i.e., organotin complexes) involves analytical procedures that are not routinely available.

Sources of information for columns 2, 3, and 4 include PSEP (1989a,b) and U.S. EPA (1983a, 1984a, 1990a,b).

Columns 5, 6, 7, 8, 9, and 10—Limits of detection and practical quantification limits for analysis of pollutants will vary depending on the method used and the level of interferences present. Columns 5—10 provide both limits of detection and practical quantification limits for water, sediment, and tissue. If PSEP protocols are available for these values, the PSEP-recommended limits are provided in the table and are enclosed in boxes. Protocols dedicated to analysis of individual contaminants or groups of contaminants may yield lower limits of detection. Individual project goals must be considered when choosing target limits of detection.

Limits of detection for water samples are listed in column 5 and are from PSEP (1989a) for inorganic chemicals and Metro (1981) for organic chemicals. Practical quantification limits for water samples are listed in column 6. These were obtained from EPA CLP guidelines for multimedia analysis of inorganic pollutants (U.S. EPA 1990a) and organic pollutants (U.S. EPA 1990b).

Limits of detection for sediments are provided in column 7 and are in accordance with those recommended by PSEP (1989a,b). Practical quantification limits for sediments are listed in column 8 and are also consistent with PSEP recommendations (PSEP 1989a,b). Although quantification limits for metals are not specifically identified in PSEP (1989a), the values in column 8 are consistent with a PSEP recommendation that metals quantification limits be approximately 3.3 times the limit of detection.

Limits of detection are provided for tissue samples in column 9. Column 10 provides practical quantification limits for tissue samples. These levels are in accordance with those recommended by PSEP (1989a,b).

Example—Benzo(a)pyrene is an EPA priority pollutant that has analytical methods available for water, sediment, and tissue samples. The water method for benzo(a)pyrene is a standard EPA procedure used in the analysis of water and wastewater samples. A PSEP protocol for water has not yet been developed for

benzo(a)pyrene; however, sediment and tissue guidelines for analysis are available for benzo(a)pyrene through PSEP (1989a,b). These guidelines allow the use of various analytical techniques according to a consistent set of quality assurance and quality control (QA/QC) procedures.

In water, the practical quantification limit for benzo(a)pyrene is 10 μg/L (i.e., 10 ppb) for routine analyses of 1-liter samples by EPA CLP methods. Limits of detection (i.e., 1 μg/L) can be obtained through special analytical service requests. Practical quantification limits and limits of detection are also available for benzo(a)pyrene in sediment samples. The limit of detection of 10 μg/kg dry weight for sediments shown in Table I for benzo(a)pyrene is within the limit of detection range of <1 to 50 μg/kg possible by different analytical procedures. The use of limits of detection rather than quantification limits are recommended for analyses of benzo(a)pyrene in tissue samples (i.e., 20 μg/kg wet weight) because of the potential use of tissue data in risk assessment.

TABLE L REGULATORY STATUS AND ANALYTICAL CONSIDERATIONS FOR POLLUTANTS OF CONCERN

	1	2	3	4	5	6	7	8	9	10
		Analytical Methods			Detection and Quantification Limits					
					Water		Sediment		Tissue	
Pollutant of Concern	U.S. EPA Pollutant Status	Water	Sediment	Tissue	DL (µg/L)	QL (µg/L)	DL (µg/kg)	QL (µg/kg)	DL (µg/kg)	QL (µg/kg)
Inorganic Chemicals					(boxes indicate Puget Sound Estuary Program guidelines)					
Antimony	P	200.7/204.2	200.7/204.2	200.7/204.2	0.05	0.17	100	330	20	66
Arsenic	P	200.7/206.2	200.7/206.2	200.7/206.2	0.10	0.30	100	330	20	66
Cadmium	P	200.7/213.2	200.7/213.2	200.7/213.2	0.02	0.07	100	330	10	33
Chromium	P	200.7/218.2	200.7/218.2	200.7/218.2	1	5	NA	NA	NA	NA
Copper	P	200.7/220.2	200.7/220.2	200.7/220.2	0.05	0.17	100	330	10	33
Cyanides	P	335.2	335.2	335.2	NA	20	NA	NA	NA	NA
Lead	P	200.7/239.2	200.7/239.2	200.7/239.2	0.02	0.07	100	330	30	99
Mercury	P	245.1	245.5	245.5	0.0005	0.0017	10	33	10	33
Nickel	P	200.7/249.2	200.7/249.2	200.7/249.2	0.05	0.17	100	330	20	66
Silver	P	200.7/272.2	200.7/272.2	200.7/272.2	0.001	0.003	100	330	10	33
Zinc	P	200.7/289.2	200.7/289.2	200.7/289.2	0.2	0.7	200	660	200	660
Organotin	N	(NCASI 1986)	(NCASI 1986)	(NCASI 1986)	0.001	NA	10	NA	50	NA
Nonionic Organic Compounds										
LPAH Compounds										
Naphthalene	P	8270/1625C	8270/1625C	8270/1625C	10	660	10	200	20	330
Acenaphthylene	P	"	"	"	10	660	10	200	20	330
Acenaphthene	P	"	"	"	10	660	10	200	20	330
Fluorene	P	"	"	"	10	660	10	200	20	330
Phenanthrene	P	"	"	"	10	660	10	200	20	330
Anthracene	P	"	"	"	10	660	10	200	20	330
1-Methylnaphthalene	N	"	"	"	NA	NA	10	200	20	330
2-Methylnaphthalene	T	"	"	"	10	660	10	200	20	330
HPAH Compounds							10	200	20	330
Fluoranthene	P	8270/1625C	8270/1625C	8270/1625C	10	660	10	200	20	330
Pyrene	P	"	"	"	10	660	10	200	20	330
Benzo(a)anthracene	P	"	"	"	10	660	10	200	20	330
Chrysene	P	"	"	"	10	660	10	200	20	330
Benzofluoranthenes (b and k)	N	"	"	"	10	660	10	200	20	330

TABLE I. (Continued)

	1	2	3	4	5	6	7	8	9	10
					Detection and Quantification Limits[a]					
		Analytical Methods			Water		Sediment		Tissue	
Pollutant of Concern	U.S. EPA[b] Pollutant Status	Water	Sediment	Tissue	DL (µg/L)	QL (µg/L)	DL (µg/kg)	QL (µg/kg)	DL (µg/kg)	QL (µg/kg)
Benzo(a)pyrene	P	8270/1625C	8270/1625C	8270/1625C	10	660	10	200	20	330
Indeno(1,2,3-c,d)pyrene	P	"	"	"	10	660	10	200	20	330
Dibenz(a,h)anthracene	P	"	"	"	10	660	10	200	20	330
Benzo(g,h,i)perylene	P	"	"	"	10	660	10	200	20	330
Methylphenanthrene	N	"	"	"	NA	NA	10	200	20	330
Chlorinated Benzenes										
1,3-Dichlorobenzene	P	"	"	"	10	660	10	200	20	330
1,4-Dichlorobenzene	P	"	"	"	10	660	10	200	20	330
Hexachlorobenzene	P	"	"	"	10	660	10	200	20	330
Miscellaneous Extractable Compounds										
N-Nitrosodiphenylamine	T	"	"	"	10	660	10	200	20	330
Methylethyl ketone	N	"	"	"	NA	NA	10	200	20	330
Bis(2-ethylhexyl)phthalate	P	"	"	"	10	660	10	200	20	330
Hexachlorobutadiene	P	"	"	"	10	660	10	200	20	330
Polychlorinated Dibenzofurans										
Tetrachlorinated furans	T	8280/8290	8280/8290	8280/8290	NA	0.0000010	NA	0.0010	NA	0.0010
Pentachlorinated dioxins	T	"	"	"	NA	0.0000010	NA	0.0010	NA	0.0010
Hexachlorinated furans	T	"	"	"	NA	0.0000025	NA	0.0025	NA	0.0025
Heptachlorinated furans	T	"	"	"	NA	0.0000025	NA	0.0025	NA	0.0025
Octachlorinated furans	T	"	"	"	NA	0.0000050	NA	0.0050	NA	0.0050
Polychlorinated Dibenzo-p-dioxins										
2,3,7,8-Tetrachlorodioxin (TCDD)	P	8280/8290	8280/8290	8280/8290	NA	0.0000010	NA	0.0010	NA	0.0010
Other tetrachlorinated dioxins	T	"	"	"	NA	0.0000010	NA	0.0010	NA	0.0010
Pentachlorinated dioxins	T	"	"	"	NA	0.0000010	NA	0.0010	NA	0.0010
Hexachlorinated dioxins	T	"	"	"	NA	0.0000025	NA	0.0025	NA	0.0025
Heptachlorinated dioxins	T	"	"	"	NA	0.0000025	NA	0.0025	NA	0.0025
Octachlorinated dioxins	T	"	"	"	NA	0.0000050	NA	0.0050	NA	0.0050

TABLE I. (Continued)

	1	2	3	4	5	6	7	8	9	10
					Detection and Quantification Limits[c]					
	U.S. EPA[b]	Analytical Methods			Water		Sediment		Tissue	
Pollutant of Concern	Pollutant Status	Water	Sediment	Tissue	DL (µg/L)	QL (µg/L)	DL (µg/kg)	QL (µg/kg)	DL (µg/kg)	QL (µg/kg)
PCBs										
Total PCBs	P	8080/8270	8080/8270	8080/8270	NA	NA	1	10	1	20
3,3',4,4'-Tetrachloro biphenyl	N	"	"	"	NA	NA	NA	NA	NA	NA
3,3',4,4',5-Pentachloro biphenyl	N	"	"	"	NA	NA	NA	NA	NA	NA
3,3',4,4',5,5'-Hexachloro biphenyl	N	"	"	"	NA	NA	NA	NA	NA	NA
Pesticides										
Aldrin	P	"	"	"	0.004	0.04	1	10	2	20
Dieldrin	P	"	"	"	0.002	0.02	1	10	2	20
4,4'-DDD	P	"	"	"	0.011	0.11	1	10	2	20
4,4'-DDE	P	"	"	"	0.004	0.04	1	10	2	20
4,4'-DDT	P	"	"	"	0.012	0.12	1	10	2	20
gamma-Hexachlorocyclohexane	P	"	"	"	0.009	0.09	1	10	2	20
Volatile Organic Compounds										
Benzene	P	8240/1624C	8240/1624C	8240/1624C	NA	5	2	10	5	20
Chloroform	P	"	"	"	NA	5	2	10	5	20
Ethylbenzene	P	"	"	"	NA	5	2	10	5	20
Toluene	P	"	"	"	NA	5	2	10	5	20
Trichloroethene	P	"	"	"	NA	5	2	10	5	20
Tetrachloroethene	P	"	"	"	NA	5	2	10	5	20
Total xylenes	T	"	"	"	NA	5	2	10	5	20
Ionizable Organic Compounds										
Phenols										
Phenol	P	8270/1625C	8270/1625C	8270/1625C	10	660	10	200	20	330
4-Methylphenol	T	"	"	"	10	660	10	200	20	330
Pentachlorophenol	P	"	"	"	50	3300	50	1000	100	3300

TABLE I. (Continued)

	1	2	3	4	5	6	7	8	9	10
					Detection and Quantification Limits[c]					
	U.S. EPA[b]	Analytical Methods			Water		Sediment		Tissue	
Pollutant of Concern	Pollutant Status	Water	Sediment	Tissue	DL (µg/L)	QL (µg/L)	DL (µg/kg)	QL (µg/kg)	DL (µg/kg)	QL (µg/kg)
Resin Acids and Guaiacols										
2-Methoxyphenol (guaiacol)	N	NA	(NCASI 1986)	(NCASI 1986)	NA	NA	NA	NA	NA	NA
Chlorinated guaiacols	N	NA	"	"	NA	NA	NA	NA	NA	NA
Chlorinated dehydroabietic acids	N	NA	"	"	NA	NA	NA	NA	NA	NA

[a] Information contained in each column is explained by column number in the text.

[b] P = Chemical is currently on the EPA Priority Pollutant List
T = Chemical is on the EPA Target Compound List or is a target analyte
N = Chemical is not an EPA priority pollutant or other routinely analyzed hazardous substance list compound.

[c] Limits enclosed in boxes are Puget Sound Estuary Program guidelines; all other limits are from the methods referenced in columns 2,3, and 4
DL = Detection limit (the lowest concentration that can be discerned above the random response of a blank sample
QL = Quantification limit (the lowest concentration that can be accurately measured and reported without qualification as an estimated quantity
G = Limits of detection for LPAH and HPAH are based on the limits established for a single compound (e.g., if PCBs are measured as Aroclors, the detection limit is that for a single representative Aroclor)
NA = Data are not available.

TABLE II: CRITERIA, GUIDELINES, AND REGULATORY ACTION LEVELS FOR POLLUTANTS OF CONCERN

Criteria, guidelines, and regulatory action levels in drinking water, ambient water, shellfish and fish tissue, and sediment are summarized for pollutants of concern in Table II.

Column 1—Available goals, standards, or proposed standards for drinking water are included in column 1 and defined in footnote b of Table II. These values are provided for comparison with ambient water quality criteria provided in other columns of the table. Under the Safe Drinking Water Act, EPA promulgates maximum contaminant level goals (MCLGs) for drinking water. These MCLGs are nonenforceable health goals. Primary MCLGs are set at a level at which no known or anticipated adverse effects on human health occur, allowing an adequate margin of safety. Maximum contaminant levels (MCLs) are enforceable standards that are set as close to MCLGs as feasible. MCLs may be set higher than MCLGs after considering factors such as available treatment technologies and cost effectiveness. If MCLG or MCL values are not available for a pollutant, secondary MCLGs are provided as available. These secondary values address aesthetic qualities such as taste and odor. Finally, other values in Table II that are proposed or are not yet available in final form are qualified by a "P" code.

Primary and secondary drinking water regulations are found in 40 CFR Parts 141 and 143, respectively. Proposed and recently finalized MCLs and MCLGs were published in the Federal Register (U.S. EPA 1985a) and updated 23 March 1988.

Columns 2, 3, 4, and 5—Ambient water quality criteria documents are published and updated periodically by EPA. These criteria reflect the latest scientific knowledge on identifiable effects of pollutants on public health, freshwater and saltwater aquatic life, and recreation. The available acute and chronic criteria are summarized and presented for freshwater and saltwater aquatic life in columns 2–5. Dashes indicate that no criteria or toxicity thresholds are available in the water quality criteria documents.

The ambient water quality criteria documents are published and updated by EPA. Data in Table II were obtained from the Gold Book (U.S. EPA 1986a) and from the water quality criteria documents announced in the Federal Register (U.S. EPA 1985b).

Columns 6 and 7—Human health effects presented in the ambient water quality documents are summarized in columns 6 and 7. Values in column 6 (cancer risk) reflect estimates of ambient water concentrations of known or suspected carcinogens that represent a one in one million (10^{-6}) incremental cancer risk. The 10^{-6} incremental cancer risk was chosen for use in the table because it represents the middle range of values presented by EPA in the water quality criteria documents. The cancer risk concentration for carcinogens and no-effect (toxicity) concentrations for noncarcinogens were estimated by extrapolation from animal toxicity or human epidemiological studies using the following assumptions: a 70-kg man as the exposed individual and an average daily consumption of freshwater and estuarine fish and shellfish products equal to 6.5 grams per day. Criteria based on these assumptions are estimated to be protective of an adult male who experiences average exposure conditions. The ambient water quality documents provide a wealth of information on contaminants. The values provided in columns 6 and 7 merely summarize the information contained in the water quality documents and are not meant to replace them. Dashes indicate that no human health data are available in the water quality documents.

Columns 8 and 9—The U.S. Food and Drug Administration (FDA) has established action levels for a limited number of contaminants in seafood (i.e., fish and shellfish). These levels are listed in column 8. These administrative guidelines, when exceeded, may trigger FDA to investigate the area where the seafood was raised or caught. A range of legal limits for seafood established by other countries is provided in column 9. Dashes indicate that no values were available.

The FDA action levels have been compiled from FDA documents (U.S. FDA 1982, 1984). Nauen (1983) compiled a summary of legal limits for other countries for the Food and Agricultural Organization of the United Nations.

Columns 10 and 11—EPA has derived a measure of toxicological potency from the dose-response relationship for a chemical of concern using a data set for

the most sensitive species. Noncarcinogens are characterized by a reference dose (RfD) value, which is the highest average daily exposure over a lifetime that would not be expected to cause adverse effects. Carcinogens are characterized by a carcinogenic potency factor, which is a measure of the cancer-causing potential of a substance. Both RfDs and carcinogenic potency factors are provided in Table II, columns 10 and 11, respectively. Dashes indicate that neither an RfD nor a carcinogenic potency factor is available.

Columns 12, 13, and 14—Expected (mean) equilibrium partitioning values and lower and upper 95 percent confidence interval values have been determined for sediments for selected nonionic organic chemicals and are provided in columns 12, 13, and 14. The values were obtained from Zarba (March 1989, personal communication) and are normalized to organic carbon.

Column 15—Some interim guidelines were recently developed for assessing sediment quality. From a national database, the Battelle Marine Research Laboratory and the Criteria and Standards Division of EPA developed what were originally termed probable no-effects levels (PNELs) and are now termed screening level concentrations (SLCs). To develop these guidelines, the presence of a given benthic species is correlated to sediment contaminant concentrations to determine the minimum concentration for a given chemical compound that was not exceeded in 90 percent of the samples containing the species. This process is carried out for numerous species to determine an SLC (Battelle 1985a; 1986). Fourteen SLC values (normalized to organic carbon content) are available for organic compounds in marine sediments and are listed in column 15. Dashes indicate that no SLC values were available.

The source of the SLC values in Table II is a final report to EPA by Battelle (Neff et al. 1987).

Column 16—Chemical criteria for marine sediment quality standards have recently been proposed by Ecology (1990; WAC 173-204-320). Values shown in column 16 for inorganic and ionizable organic chemicals are expressed as µg/kg dry weight. Corresponding chemical criteria for nonionic organic chemicals (in parentheses) are expressed as µg/kg organic carbon. Dashes indicate that no marine sediment quality standards were available.

Columns 17, 18, 19, and 20—Other interim guidelines, called apparent effects threshold (AET) values, have been developed for federal and Washington state agencies (U.S. EPA 1988; Ecology 1990). AET values were derived using chemical and biological data from several Puget Sound investigations. The AET values in columns 17, 18, and 19 are based on toxicity data from the amphipod bioassay, the oyster larvae bioassay, and the Microtox® bioassay, respectively. AET values in column 20 are based on effects as measured by abundances of benthic infauna. The AET values are defined as the concentration above which statistically significant biological effects ($P < 0.05$) always occur in the databases of sediment samples used to create the values. Dashes indicate that AET values have not been established, usually because insufficient data are available.

The data used in developing AET values are from Battelle (1985b), Chan et al. (1985a,b), Osborn et al. (1985), Barrick et al. (1988), Romberg et al. (1984), Tetra Tech (1985b), Beller et al. (1988), Pastorok et al. (1988), and U.S. Navy (1985).

Column 21—Exceedance of criteria established in Ecology's Dangerous Waste Regulations [Ecology 1984 (revised 1990); Chapter 173-303 WAC] generally depends on the volume of waste generated as well as the chemical and physical characteristics of the waste. Although the numeric criteria are not easily adapted to tabular format, WAC 173-303-9903 (Ecology 1984) designates a discarded chemical product as either a dangerous waste (DW) or an extremely hazardous waste (EHW) and also provides the reason for the designation. Reportable quantities for the discarded chemicals are determined in accordance with their toxic constituents, as established in WAC 173-303-081 and 173-303-084. The information from WAC 173-303-9903 has been provided in column 21 where data are available. Chemicals that are not specifically listed or designated may still be classified as EHW or DW by the regulations. Dashes indicate that the chemical is not specifically listed in the regulations.

The information in column 21 was compiled from the state Dangerous Waste Regulations [WAC 173-303-9903, as amended by WSR 90-20-101 (17 October 1990)] (Ecology 1984). These regulations incorporate, by reference, the National Institute for Occupational Safety and Health's *Registry of Toxic Effects of Chemical Substances* and EPA's spill table (40 CFR 302.4). Both the registry and the spill table are references for determining toxic categories for constituents in a waste.

Additional data summaries of lifetime cancer risk vs. chemical concentration provided in Appendix A have been updated from Tetra Tech (1986a,b) using information from U.S. EPA (1990c).

Example—No EPA drinking water standards or ambient water quality criteria have been set for benzo(a)pyrene. Some information is available for HPAH compounds, a group of organic chemicals on EPA's priority pollutant list that includes benzo(a)pyrene. These data include water concentrations described in EPA water quality criteria documents as apparent threshold levels for acute or chronic toxic effects on marine organisms (300 μg/L) and an estimate of the 10^{-6} incremental cancer risk (31.1 ng/L) to humans by consumption of contaminated seafood. The chemical criterion for the benzo(a)pyrene marine sediment standard in Puget Sound is 99,000 μg/kg organic carbon. Additional levels of concern for benzo(a)pyrene in sediments are available for different biological effects indicators based on Puget Sound field investigations; resulting AET values range from 1,600 to 3,600 μg/kg dry weight. The state dangerous waste regulations classify benzo(a)pyrene as a persistent hydrocarbon that has been sufficiently designated as a carcinogen (Category P,+; see Table II) for reportable quantity purposes. Benzo(a)pyrene is a persistent HPAH compound for which there is sufficient animal or human data to establish the compound as a carcinogen. There are no FDA or other legal limits for benzo(a)pyrene in tissue samples.

TABLE II. *(Continued)*

	12	13	14	15	16	17	18	19	20	21
	Sediment									
	Equilibrium Partitioning Coefficient Values (µg/kg)			SLC (µg/kg organic carbon)	Sediment Quality Criteria (µg/kg)	Apparent Effects Threshold (µg/kg dry weight)				Dangerous Waste Designation
Pollutant of Concern	Low	Mean	High			Amphipod Toxicity	Oyster Larvae Toxicity	Microtox Toxicity	Benthic Effects	
Inorganic Chemicals	NOTE: All values in (PARENTHESES) are expressed in terms of µg/kg ORGANIC CARBON; all other values are in terms of µg/kg DRY WEIGHT									
Antimony	—	—	—	—	150,000	200,000	26,000	26,000	150,000	—
Arsenic (III)/(V)	—	—	—	—	57,000	93,000	700,000	700,000	57,000	EHW,B+/B
Cadmium	—	—	—	—	5,100	6,700	9,600	9,600	5,100	—
Chromium (III)/(VI)	—	—	—	—	260,000	270,000	>37,000	27,000	260,000	—
Copper	—	—	—	—	390,000	1,300,000	390,000	390,000	530,000	—
Cyanides	—	—	—	—	—	—	—	—	—	EHW,A
Lead	—	—	—	—	450,000	660,000	660,000	530,000	450,000	—
Mercury	—	—	—	—	410	2,100	590	410	—	EHW,EP
Nickel	—	—	—	—	140,000	>140,000	39,000	28,000	>140,000	—
Silver	—	—	—	—	6,100	6,100	>560	>560	6,100	—
Zinc	—	—	—	—	410,000	960,000	1,600,000	1,600,000	410,000	—
Organotin	—	—	—	—	—	—	—	—	—	—
Nonionic Organic Compounds										
LPAH Compounds	—	—	—	—	(370,000)	24,000	5,200	5,200	13,000	—
Naphthalene	—	—	—	(41,400)	(99,000)	2,400	2,100	2,100	2,700	EHW,B
Acenaphthylene	—	—	—	(4,740)	(66,000)	1,300	>560	>560	1,300	—
Acenaphthene	—	—	—	—	(16,000)	2,000	500	500	730	—
Fluorene	—	—	—	(10,100)	(23,000)	3,600	540	540	1,000	EHW,B
Phenanthrene	(24,000)	(100,000)	(440,000)	(36,800)	(100,000)	6,900	1,500	1,500	5,400	—
Anthracene	—	—	—	(16,300)	(220,000)	13,000	960	960	4,400	—
1-Methylnaphthalene	—	—	—	—	—	—	—	—	—	—
2-Methylnaphthalene	—	—	—	—	(64,000)	1,900	670	670	1,400	—

TABLE II. (Continued)

	12	13	14	15	16	17	18	19	20	21
	Sediment									
Pollutant of Concern	Equilibrium Partitioning Coefficient Values (µg/kg) Low	Mean	High	SLC (µg/kg organic carbon)	Sediment Quality Criteria[h] (µg/kg)	Apparent Effects Threshold[i] (µg/kg dry weight) Amphipod Toxicity	Oyster Larvae Toxicity	Microtox Toxicity	Benthic Effects	Dangerous Waste Designation[j]
HPAH Compounds	—	—	—	—	(980,000)	69,000	17,000	12,000	69,000	—
Fluoranthene	—	—	—	(84,400)	(160,000)	30,000	2,500	1,700	24,000	DW,D
Pyrene	—	—	—	(66,500)	(1,000,000)	16,000	3,300	2,600	16,000	—
Benz[a]anthracene	—	—	—	(26,100)	(110,000)	5,100	1,600	1,300	5,100	EHW,P,+
Chrysene	—	—	—	(38,400)	(110,000)	9,200	2,800	1,400	9,200	EHW,P,+
Total benzofluoranthenes	—	—	—	—	(230,000)	7,600	3,600	3,200	9,900	EHW,P,+
Benzo[a]pyrene	—	—	—	(39,700)	(99,000)	3,000	1,600	1,600	3,600	EHW,P,+
Indeno(1,2,3-c,d)pyrene	—	—	—	—	(33,000)	1,800	600	600	2,600	DW,+
Dibenz[a,h]anthracene	—	—	—	—	(33,000)	540	230	230	970	EHW,A,P,+
Benzo[g,h,i]perylene	—	—	—	—	(31,000)	1,400	720	670	2,600	—
Methylphenanthrenes	—	—	—	—	—	—	—	—	—	—
Chlorinated Benzenes										
1,3-Dichlorobenzene	—	—	—	—	(15,000)	>170	>170	>170	>170	EHW,B,H
1,4-Dichlorobenzene	—	—	—	—	(3,100)	120	120	110	110	EHW,B,H
Hexachlorobenzene	—	—	—	—	(380)	130	230	70	22	EHW,H
Miscellaneous Extractable Compound										
N-Nitrosodiphenylamine	—	—	—	—	(11,000)	48	130	40	28	EHW,B,+
Methylethyl ketone	—	—	—	—	—	—	—	—	—	DW,D,I
Bis(2-ethylhexyl)phthalate	—	—	—	(50,000)	(47,000)	>3,100	1,900	1,900	1,300	—
Hexachlorobutadiene	—	—	—	—	(3,900)	180	270	120	11	EHW,C,H
Dibenzofuran	—	—	—	—	(15,000)	1,700	540	540	700	—
Polychlorinated Dibenzofurans										
Tetrachlorinated furans	—	—	—	—	—	—	—	—	—	—
Pentachlorinated furans	—	—	—	—	—	—	—	—	—	—
Hexachlorinated furans	—	—	—	—	—	—	—	—	—	—
Heptachlorinated furans	—	—	—	—	—	—	—	—	—	—
Octachlorinated furans	—	—	—	—	—	—	—	—	—	—

NOTE: All values in (PARENTHESIS) are expressed in terms of µg/kg ORGANIC CARBON, all other values are in terms of µg/kg DRY WEIGHT

TABLE II. (Continued)

	12	13	14	15	16	17	18	19	20	21
	Sediment									
	Equilibrium Partitioning Coefficient Values (µg/kg)			SLC (µg/kg organic carbon)	Sediment Quality Criteria[b] (µg/kg)	Apparent Effects Threshold[c] (µg/kg dry weight)				Dangerous Waste Designation[d]
Pollutant of Concern	Low	Mean	High			Amphipod Toxicity	Oyster Larvae Toxicity	Microtox Toxicity	Benthic Effects	
Polychlorinated Dibenzo-p-dioxins	NOTE: All values in (PARENTHESES) are expressed in terms of µg/kg ORGANIC CARBON; all other values are in terms of µg/kg DRY WEIGHT									
2,3,7,8-Tetrachlorodioxin (TCDD)	—	—	—	—	—	—	—	—	—	—
Other tetrachlorinated dioxins	—	—	—	—	—	—	—	—	—	—
Pentachlorinated dioxins	—	—	—	—	—	—	—	—	—	—
Hexachlorinated dioxins	—	—	—	—	—	—	—	—	—	—
Heptachlorinated dioxins	—	—	—	—	—	—	—	—	—	—
Octachlorinated dioxins	—	—	—	—	—	—	—	—	—	—
PCBs										
Total PCBs	(8,300)	(41,000)	(210,000)	(3,660)	(12,000)	3,100	1,100	130	1,100	—
3,3',4,4'-Tetrachloro biphenyl	—	—	—	—	—	—	—	—	—	—
2,3',4,4',5-Pentachloro biphenyl	—	—	—	—	—	—	—	—	—	—
2,3',4,4',5,5'-Hexachloro biphenyl	—	—	—	—	—	—	—	—	—	
Pesticides										
Aldrin	—	—	—	—	—	—	—	—	—	EHW,X,H
Dieldrin	—	—	—	—	—	—	—	—	—	EHW,X,H,+
4,4'-DDD	—	—	—	—	—	43	—	—	16	EHW,C,H,+
4,4'-DDE	—	—	—	—	—	15	—	—	9	—
4,4'-DDT	(180)	(830)	(3,900)	(50,500)	—	>270	>6	—	34	EHW,X,H,+
gamma-Hexachlorocyclohexane	(430)	(5,800)	(76,000)	(62,600)	—	—	—	—	—	EHW,H,+
Volatile Organic Compounds										
Benzene	—	—	—	—	—	—	—	—	—	EHW,C,+
Chloroform	(13,000)	(8,000)	(500,000)	—	—	—	—	—	—	EHW,C,H,+
Ethylbenzene	(2,400)	(17,000)	(120,000)	—	(3,800)	>50	37	33	10	—
Toluene	—	—	—	—	—	—	—	—	—	EHW,C,I
Trichloroethene	—	—	—	—	—	—	—	—	—	EHW,C,H,+
Tetrachloroethene	(5,100)	(35,000)	(250,000)	—	(22,000)	>210	140	140	57	EHW,C,H,+
Total xylenes	—	—	—	—	(12,000)	>160	120	100	40	EHW,C,I

TABLE II. (Continued)

	12	13	14	15	16	17	18	19	20	21
	Sediment									
Pollutant of Concern	Equilibrium Partitioning Coefficient Values (µg/kg) Low	Mean	High	SLC (µg/kg organic carbon)	Sediment Quality Criteria (µg/kg)	Apparent Effects Threshold (µg/kg dry weight) Amphipod Toxicity	Oyster Larvae Toxicity	Microtox Toxicity	Benthic Effects	Dangerous Waste Designation
Ionizable Organic Compounds	NOTE: All values in (PARENTHESES) are expressed in terms of µg/kg ORGANIC CARBON; all other values are in terms of µg/kg DRY WEIGHT									
Phenols										
Phenol	—	—	—	—	420	1,200	420	1,200	1,200	EHW,C
4-Methylphenol	—	—	—	—	670	3,600	670	670	1,800	EHW,C
Pentachlorophenol	—	—	—	—	360	>360	>140	>140	>690	EHW,A,H
Resin Acids and Guaiacols										
2-Methoxyphenol (guaiacol)	—	—	—	—	—	—	—	—	—	—
Chlorinated guaiacols	—	—	—	—	—	—	—	—	—	—
Chlorinated dehydroabietic acids	—	—	—	—	—	—	—	—	—	—

a Information contained in each column is explained by column number in the text.

b
G = Maximum contaminant level goals (MCLGs), nonenforceable health goals
M = Maximum contaminant levels (MCLs), enforceable standards
P = Proposed value
( ) = Secondary MCLGs
— = No MCL or MCLG values are proposed or exist.

c
( ) = There are insufficient data available to develop criteria; values presented are lowest observed effect levels (LOELs).
H = Freshwater quality criteria for some chemicals are a function of hardness. The relationship is not linear and the equations specific to each chemical are found in the criteria documents. For this table, a criteria concentration based on a hardness value of 50 mg/L calcium carbonate is provided. Exact criteria values must be calculated from the equations.
P = Toxicity is pH dependent. This value is calculated based on a pH of 7.8.
* = Where two values are provided for arsenic, the first is for trivalent arsenic (III) and the second is for pentavalent arsenic (V). Where two values are provided for chromium, the first is for trivalent chromium (III) and the second is for hexavalent chromium (VI).
— = No criteria or toxicity thresholds are presented in the water quality criteria documents.

TABLE II. (Continued)

[d] EPA water quality criteria documents contain criteria based on human health effects associated with exposure from consumption of fish and shellfish that are assumed to have bioconcentrated pollutants from the water in which they live. The criteria concentrations were estimated using the following assumptions: one 70-kg man as the exposed individual, and the average daily consumption of freshwater and estuarine fish and shellfish products equal to 6.5 grams per day. The calculated concentrations presented in the cancer risk column are associated with an estimated 1 in 1 million incremental cancer risk. For noncarcinogens, the toxicity column displays the calculated concentration expected to protect humans from adverse effects.

= Analysis of the toxic effects data resulted in a calculated value comparable to the existing drinking water standards. *No criteria were calculated based solely on the consumption of seafood.* The drinking water standard is shown.

* = Where two values are provided for chromium, the first is for trivalent chromium (III) and the second is for hexavalent chromium (VI).

--- = No human health data are available in the water quality criteria documents.

[e] The ranges shown represent the legal limits established by other countries for edible marine organisms. Dioxin (TCDD equivalency) values are from British Columbia (.0114 ppb) and Canada (0.020). Other values were compiled by the Food and Agricultural Organization (FAO) of the United Nations (Nauen 1983).

[f] The reference dose (RfD) is the highest average daily exposure over a lifetime that would not be expected to cause adverse effects.
R = A plot of the noncarcinogenic risk index for mercury vs. concentration in seafood is found in Appendix A.

* = Where two values are provided for chromium, the first is for trivalent chromium (III) and the second is for hexavalent chromium (VI).

— = No RfD is available.

[g] Carcinogens are characterized by a carcinogenic potency factor, a measure of the cancer-causing potential for the substance $(mg-kg/day)^{-1}$.
C = Carcinogenic PAH compounds; potency factors for carcinogenic PAH other than benzo(a)pyrene have not been published. However, relative potency estimates for these compounds [relative to benzo(a)pyrene] are under consideration by EPA.
R = A plot of lifetime cancer risk for arsenic, carcinogenic PAH, and PCBs vs. concentration in seafood is found in Appendix A.
N = Not considered a carcinogen via dietary exposure.
--- = No carcinogenic potency factor is available.

[h] The sediment quality criteria values are expressed EITHER as µg/kg DRY WEIGHT (metals and ionizable organic compounds) or µg/kg ORGANIC CARBON (nonpolar organic compounds), as provided in WAC 173-204-320. All criteria values expressed in terms of ORGANIC CARBON are shown in parenthesis ( ).

[i] > = An apparent effects threshold has not been established (i.e., the value shown is the highest concentration found at a nonimpacted station and no higher concentrations have been documented).

[j] Washington State Dangerous Waste Regulations (Chapter 173-303 WAC, as amended by WSR 90-20-101), provide the reasons for designation of a chemical as an extremely hazardous waste or dangerous waste and the categories of reportable quantities, as set forth below:

EHW = Extremely hazardous waste
DW = Dangerous waste
X = Toxic, Category X
A = Toxic, Category A
B = Toxic, Category B
C = Toxic, Category C
D = Toxic, Category D
? = Toxic, Category not determined
H = Persistent, halogenated hydrocarbon
O = Corrosive
P = Persistent, polycyclic aromatic hydrocarbon
+ = International Agency for Research on Cancer (IARC) animal or human sufficient or limited carcinogen
I = Ignitable
R = Reactive
EP = Toxicity characteristic.

A chemical that is not listed or specifically designated may still be designated EHW or DW by the regulations.

TABLE III: SOURCES OF POLLUTANTS

Major known sources of contamination around Puget Sound are listed in Table III. Municipal and industrial discharges are the two main categories of point sources. Additional information of pollutant sources is summarized in Chapter 3, *Description of Pollutants of Concern in Puget Sound.*

Column 1—For municipal discharges, chemicals are classified according to their frequency of detection (i.e., detected in >25 percent of samples analyzed, detected in ≤25 percent of samples, or not detected). When results for fewer than five samples are available, no estimate of the frequency of detection is given. Dashes indicate that insufficient information (i.e., fewer than five samples) is available to categorize the chemical.

The municipal discharge data used to categorize the pollutants are from Metro's Toxicant Pretreatment Planning Study (TPPS) (Cooley et al. 1984) and Barrick (1982). Supporting data were obtained from the city of Everett wastewater treatment plant (Baird, C.E., 1 August 1985, personal communication) and Class II inspection surveys (Andreasson 1990a,b; Hallinan 1988; Heffner 1988, 1990c; Reif 1988a; Zinner 1991).

Column 2—The types of industries from which release of each chemical has been documented is coded in the industrial (point source) column of Table III (column 2). Each industry is given a separate descriptor code (e.g., ship building and repair is designated by the letter S). Dashes indicate that insufficient data are available to categorize the sources of these pollutants.

Industrial sources of pollutants were determined using data from industrial reports prepared by Ecology's Water Quality Investigation Section (Joy 1987; Norton et al. 1987; Stinson and Norton 1987b), and from Class II industrial surveys, including recent reports by Hallinan (1989; 1990a,b), Hallinan and Ruiz (1990), Heffner (1989a,b; 1990a,b), and Reif (1988b, 1990). Information from older Class II industrial surveys was abstracted from the Commencement Bay remedial investigation (Tetra Tech 1985b) and feasibility study (Tetra Tech 1986c). Additional data were included from Norton (5 February 1986, personal communication), Galvin and Moore (1982), Martin and Pavlou (1985), Palmork et al. (1973), Sittig (1980), Stranks (1976), and Young et al. (1979).

Column 3—Data generated from combined sewer overflow (CSO) sampling are included in column 3. Chemicals found in CSOs are classified according to their frequency of detection (i.e., the same as municipal discharges). Dashes indicate that insufficient information is available to categorize the chemical. The Metro TPPS (Cooley et al. 1984) was the source of information on pollutants in CSOs.

Column 4—Nonpoint sources are difficult to identify and quantify. Nonpoint sources listed in Table III include agricultural, urban, and industrial runoffs and groundwater seeps. The urban runoff designation includes those chemicals detected in >10 percent of the samples analyzed for the National Urban Runoff Program (NURP) (U.S. EPA 1983b). The 10 percent criterion, used in the NURP summary report, was also used in Table III. Dashes indicate that insufficient information is available to categorize the type of source.

Urban runoff data are primarily from NURP (U.S. EPA 1983b) and are considered representative of Puget Sound's urban runoff. The NURP study included analyses of runoff from Bellevue, Washington. Also accommodated in the table are urban and industrial runoff and groundwater data gathered by Ecology and other investigators, as summarized in Tetra Tech (1985b) and in recent reports by Johnson and Norton (1989), Joy (1987), Norton (1988; 1990a,b), Stinson and Norton (1987a,b), and Stinson et al. (1987).

Column 5—Occasionally, product spills (e.g., ore and oil) occur in Puget Sound that release chemicals into the environment. The types of spills that have occurred in Puget Sound where chemicals are expected to be found are indicated in column 5. Dashes indicate that there are insufficient data to categorize the type of spill. Sources of information used to categorize pollutant spills include Norton (1985b), Sittig (1980), and Tetra Tech (1985b).

Example—Benzo(a)pyrene has been detected in >25 percent of available municipal effluent and CSO samples from Puget Sound. There are insufficient data to document the presence of benzo(a)pyrene in discharges from industrial point sources in Puget Sound, although there are a number of industrial processes that are expected to generate benzo(a)pyrene (e.g., combustion of fossil fuel, primary production of ferrous and nonferrous metals, and wood treatment with creosote). There are also insufficient published data from Puget Sound or NURP (U.S. EPA 1983b) to document the presence of benzo(a)pyrene in discharges

from nonpoint sources in Puget Sound, although benzo(a)pyrene has been reported as a component of stormwater runoff in research studies conducted in Lake Washington, southern California, Narragansett Bay, and Europe. Benzo(a)-pyrene is an expected component of most oil spills.

TABLE III. SOURCES OF POLLUTANTS [a]

Pollutant of Concern	1	2 Point Sources	3	4	5
	[b] Municipal	[c] Industrial	[d] CSOs	Nonpoint Sources	[e] Spills
Inorganic Chemicals					
Antimony	>25%	C,CA,(E),LS,M,(OC),OR,PI	>25%	UR,IR	OS
Arsenic	>25%	C,(E),IC,L,LS,M,OC,OR,P,S	>25%	AR,UR,IR,GW	OS
Cadmium	>25%	C,CP,(DC),M,PI	>25%	UR,IR,GW	C
Chromium	>25%	CP,F,IC,(L),OR,P,PI,(S),(SC)	>25%	UR,IR,GW	C,OS
Copper	>25%	C,CA,CP,L,LS,M,OR,P,PD,PI,S	>25%	UR,IR,GW	OS
Cyanides	>25%	C,CP,(F),(M),OR,P,PD,PH	>25%	UR	C
Lead	>25%	C,CA,(DC),LS,M,OC,OR,P,PD,PI,(S)	>25%	UR,IR,GW	OS,O
Mercury	>25%	B,CA,(DC),(IC),M,OC,OR,P,S	>25%	UR,IR,GW	C,OS
Nickel	>25%	C,CA,M,OC,OR,P,PI	>25%	GW,IR,UR	OS
Silver	>25%	(CP),(E),IC,PH,PI	>25%	GW,IR,UR	OS,C
Zinc	>25%	C,CA,(CP),(DC),(E),IC,LS,M, ...,OC,OR,P,PD,R,S,SC	>25%	GW,IR,UR	OS
Organotin	>25%	D,OC,OR,PL,S	>25%	--	--
Nonionic Organic Compounds					
LPAH Compounds	>25%	(CO),D,L,M,(P),(RU),(S)	>25%	GW,IR,UR	O
Naphthalene	>25%	D,L,(OR),(OC),(P)	>25%	GW,IR,UR	O
Acenaphthylene	ND	L	≤25%	GW,IR,UR	O
Acenaphthene	≤25%	L,M,PD	≤25%	GW,IR,UR	O
Fluorene	≤25%	L,M,PD	>25%	GW,IR,UR	O
Phenanthrene	>25%	D,L,M,PD	>25%	GW,IR,UR	O
Anthracene	>25%	D,L	≤25%	GW,IR,UR	O
1-Methylnaphthalene	≤25%	(OR),(OC),(P)	≤25%	GW,IR,UR	O
2-Methylnaphthalene	>25%	(OR),(OC),(P)	>25%	GW,IR,UR	O
HPAH Compounds	>25%	(CO),D,L,M,OC,(P),(R),(S)	>25%	GW,IR,UR	O
Fluoranthene	>25%	D,L,M	>25%	GW,IR,UR	O
Pyrene	>25%	D,L,M	>25%	GW,IR,UR	O
Benz(a)anthracene	>25%	L,M	>25%	GW,IR,UR	O
Chrysene	>25%	L,M	>25%	GW,IR,UR	O

TABLE III. (Continued)

Pollutant of Concern	1	2 Point Sources	3	4	5
	Municipal[b]	Industrial[c]	CSOs[d]	Nonpoint Sources[e]	Spills[f]
Total benzofluoranthenes	>25%	L	>25%	GW,IR,UR	O
Benzo(a)pyrene	>25%	L,M	≤25%	GW,IR,UR	O
Indeno(1,2,3-c,d)pyrene	>25%	L	>25%	GW,IR,UR	O
Dibenz(a,h)anthracene	≤25%	L	≤25%	GW,IR,UR	O
Benzo(g,h,i)perylene	>25%	L	≤25%	GW,IR,UR	O
Methylphenanthrenes	--	(OR),(P)	--	GW,IR,UR	--
Chlorinated Benzenes					
1,3-Dichlorobenzene	≤25%	(E),OC	≤25%	IR	--
1,4-Dichlorobenzene	≤25%	(E),OC	≤25%	IR,UR	--
Hexachlorobenzene	ND	(M),OC	--	IR	--
Miscellaneous Extractable Compounds					
N-Nitrosodiphenylamine	ND	(OC),(PI),(RU)	≤25%	UR	--
Methylethyl ketone (2-butanone)	--	DC,OC,PD,PI,S	--	IR,UR	C
Bis(2-ethylhexyl)phthalate	--	(E),PL,(PI)	--	IR	C
Hexachlorobutadiene	ND	(DC),OC	--	GW	C
Dibenzofuran	--	C,L,LS	--	IR,UR	--
Polychlorinated Dibenzofurans	--	B,L,OC,P	--	IR	--
Tetrachlorinated furans	--	B,L,OC,P	--	--	--
Pentachlorinated furans	--	B,L,OC,P	--	--	--
Hexachlorinated furans	--	B,L,OC,P	--	IR	--
Hexachlorinated furans	--	B,L,OC,P	--	IR	--
Octachlorinated furans	--	B,L,OC,P	--	IR	--
Polychlorinated Dibenzo-p-dioxins	--	B,L,OC,P	--	IR	--
2,3,7,8-Tetrachlorodioxin (TCDD)	--	B,L,OC,P	--	--	--
Other tetrachlorinated dioxins	--	B,L,OC,P	--	--	--
Pentachlorinated dioxins	--	B,L,OC,P	--	IR	--
Hexachlorinated dioxins	--	B,L,OC,P	--	IR	--
Heptachlorinated dioxins	--	B,L,OC,P	--	IR	--
Octachlorinated dioxins	--	B,L,OC,P	--	IR	--

TABLE III. (Continued)

Pollutant of Concern	1	2	3	4	5
	Point Sources				
	[b] Municipal	[c] Industrial	[d] CSOs	[e] Nonpoint Sources	[f] Spills
PCBs					
Total PCBs	≤25%	M,SC	>25%	IR,UR	C,O
3,3',4,4'-Tetrachloro biphenyls	--	--	--	--	--
3,3',4,4',5-Pentachloro biphenyls	--	--	--	--	--
3,3',4,4',5,5'-Hexachloro biphenyls	--	--	--	--	--
Pesticides					
Aldrin	--	--	--	AR	--
Dieldrin	--	--	--	AR	--
4,4'-DDD	--	--	--	AR	C
4,4'-DDE	--	--	--	AR	C
4,4'-DDT	--	--	--	AR	C
gamma-Hexachlorocyclohexane	--	L,LS,OC	--	AR,UR	C
Volatile Organic Compounds					
Benzene	ND	DC,OC,OR,PI,(PL),(RU)	--	IR,UR	O,C
Chloroform	>25%	DC,(E),IC,M,OC,OR,P,PI,(PL)	>25%	GW,UR	C
Ethylbenzene	>25%	(OC),OR,(PI)	>25%	GW,IR,UR	O,C
Toluene	ND	D,OC,OR,PI	--	GW,IR,UR	O,C
Trichloroethene	>25%	CA,(DC),(E),(M),OC,P	>25%	GW,IR,UR	C
Tetrachloroethene	>25%	CA,(DC),(E),IC,(M),P,(PI),PD,OC	>25%	GW,IR	C
Total xylenes	≤25%	OC,(OR),PD,(PI),PL	--	GW,IR,UR	O,C
Ionizable Organic Compounds					
Phenols					
Phenol	>25%	IC,L,LS,M,OC,OR,P,(PL),(RU)	>25%	UR,IR	C
4-Methylphenol	--	(L),(M),(OC),(OR),P,(PL),(RU)	--	UR,IR	O,C
Pentachlorophenol	≤25%	IC,L,OC,P	--	UR,IR,GW	C
Resin Acids and Guaiacols					
2-Methoxyphenol (guaiacol)	--	P	--	IR	--
Chlorinated guaiacols	--	B,P	--	IR	--
Chlorinated dehydroabietic acids	--	B,P	--	IR	--

TABLE III. (Continued)

[a] Information contained in each column is explained by column number in the text.

[b] >25% = Chemical occurs in more than 25 percent of samples from Puget Sound municipal discharges
≤25% = Chemical occurs in 25 percent or fewer samples from Puget Sound municipal discharges
ND = Chemical not detected based on available information
-- = There are insufficient data to categorize.

[c] The following codes are used to describe industries that are point sources of chemicals:

B = Bleach plant
C = Copper smelters
CA = Chloralkali plants
CO = Coal handling
CP = Chrome and silver plating
D = Docks
DC = Dry cleaning
E = Electronics
F = Ferro, silicon, chrome industries
IC = Inorganic chemical manufacturing
L = Log/wood treatment facility, plywood
LS = Log sort yards
M = Primary production of ferrous and nonferrous metals
OC = Organic chemical manufacturing
OR = Oil refining
P = Pulp mills
PD = Petroleum Distributor
PI = Paint and ink
PH = Photography
PL = Plastics
R = Roofing
RU = Rubber manufacturing
S = Ship building/repair
SC = Scrap yards

Codes enclosed in parentheses are potential sources that have not yet been documented in Puget Sound

-- = There are insufficient data to categorize.

[d] >25% = Chemical occurs in more than 25 percent of samples from Puget Sound combined sewer overflows (CSOs)
≤25% = Chemical occurs in 25 percent or fewer samples from Puget Sound CSOs
ND = Chemical not detected based on available information
-- = There are insufficient data to categorize.

[e] The following codes are used to describe nonpoint sources of chemicals:

UR = Urban runoff
AR = Agricultural runoff
IR = Industrial runoff
GW = Groundwater seeps

-- = There are insufficient data to categorize.

[f] Sources of chemicals may be attributed to the following spills:

O = Oil spills, or particularly in the case of PAH, creosote spills
C = Miscellaneous product spills
OS = Ore spills
-- = There are insufficient data to categorize.

TABLE IV: CONCENTRATIONS OF POLLUTANTS IN PUGET SOUND

Concentrations of chemicals in sediment, fish and shellfish tissue, and water samples from Puget Sound are summarized in Table IV. The summary for sediments includes substantial data for reference areas chosen by different investigators as regions removed from known sources of contamination in Puget Sound, as well as for nonreference areas (e.g., urban embayments and the central basin). Data for tissue and water samples are more limited. Additional information on the general environmental distribution of each pollutant is presented in Chapter 3, *Description of Pollutants of Concern in Puget Sound*.

Columns 1 through 15—A computerized database was used to calculate minimum, median, 90th percentile, and maximum concentrations found in sediment samples taken from reference and nonreference areas and urban bays in Puget Sound. Detection frequencies for sediment samples were also calculated. All sediment analyses conducted in Puget Sound have not been incorporated into the database, but the data for some chemicals represent nearly 1,000 samples from nonreference stations and 150 samples from reference stations in Puget Sound. This database is continuing to be expanded. In all cases, the highest detected value is used as the maximum. The minimum is either the lowest detected value or the lowest detection limit for undetected values (whichever is smaller). These criteria were used because detection limits can vary substantially for different samples and studies. Dashes indicate that insufficient data are available to calculate concentrations, or that no data are available for that chemical.

The sediment chemistry database compiled for Puget Sound (U.S. EPA 1988) includes data from Chan et al. (1985a,b), Battelle (1986), Beller et al. (1988), Pastorok et al. (1988), Romberg et al. (1984), PSAMP (1990a,b), PSDDA (1988, 1989) Tetra Tech (1985b, 1986d, 1990), Trial and Michaud (1985), and U.S. Navy (1985), Crecelius et al. (1989), PTI (1990).

Columns 16, 17, 18 and 19—Minimum and maximum concentrations of chemicals found in fish muscle tissue are provided in columns 16 and 17 of Table IV. Minimum and maximum concentrations of chemicals in shellfish tissue are provided in columns 17 and 18. The data are not presently available in a form that readily allows computation of the medians and percentiles provided for the sediments. Liver or hepatopancreas tissues are not included. However, limited chlorinated dioxin and furan data for fish tissues include whole fish

samples reported by Terpening (4 October 1989, personal communication). Dashes indicate insufficient data are available.

The summary ranges of concentrations in Table IV are intended to provide an indication of the magnitude of contamination in fish and shellfish tissues from Puget Sound. More detailed summaries by specific areas of Puget Sound and by species are provided in PSEP (1988b) and Faigenblum (1988). Mean values are also provided in these reports, although not all of the data provided in the ranges in Table IV are included in those references.

Tissue data were compiled from Beller et al. (1988), CH2M Hill (1989), Clark (1983), Crecelius et al. (1989), Faigenblum (1988), Goldberg et al. (1983), Landolt et al. (1987), Malins et al. (1980), Norton (5 February 1986, personal communication), Pastorok et al. (1988), PSEP (1988b), PTI (1990), Sherwood et al. (1980), Tetra Tech (1985b), Yake et al. (1984) and Terpening (4 October 1989, personal communication).

Columns 20 and 21—Information for concentrations of pollutants in waters from reference areas has not yet been included in Table IV. Thus, columns 22 and 23 contain dashes indicating the lack of data.

Columns 22 and 23—Minimum and maximum concentrations found in nonreference Puget Sound waters are provided in columns 20 and 21 of Table IV. The data are not presently in a form that allows computation of medians and percentiles provided for the samples. Blank spaces indicate that insufficient data are available.

Receiving water data were compiled from water quality surveys by Ecology (Bernhardt 1982; Johnson and Prescott 1982a,b,c,d; Norton 1985a,b), EPA (Osborn 1980a,b), and Metro (Romberg et al. 1984).

Example—Benzo(a)pyrene is found in sediments from nonreference and reference areas and from urban bays. Based on the current database, concentrations of benzo(a)pyrene in sediments of Puget Sound range from <1 to 100,000 μg/kg dry weight, although most concentrations (even in nonreference areas) are <240 μg/kg dry weight. Benzo(a)pyrene has been detected in shellfish tissue (maximum 240 μg/kg wet weight), but not in muscle tissue of fish. Water concentrations of benzo(a)pyrene in Puget Sound are typically undetected (i.e., <1 μg/L).

TABLE IV. CONCENTRATIONS OF POLLUTANTS IN PUGET SOUND [a]

	1	2	3	4	5	6	7	8	9	10
	Sediments (μg/kg dry weight) [b]									
	Reference Areas [c]					Nonreference Areas [d]				
Pollutant of Concern	Minimum	Median	90th Percentile	Maximum	Detection Frequency	Minimum	Median	90th Percentile	Maximum	Detection Frequency
Inorganic Chemicals										
Antimony	U40	1,640	5,860	9,470	25/58	U19	1,000	29,900	1,370,000	537/734
Arsenic	340	7,050	17,400	26,200	63/73	U110	11,100	35,100	12,200,000	972/1018
Cadmium	U40	425	1,570	8,100	61/70	20	630	2,800	184,000	939/987
Chromium	9,600	49,000	142,000	888,000	71/71	U3,000	46,300	120,000	1,080,000	856/864
Copper	3,690	26,700	52,900	76,000	70/71	U50	52,000	175,000	14,300,000	974/983
Cyanides	U800	—	—	—	0/3	50	250	500	1,000	34/53
Lead	U100	12,500	27,500	44,000	142/151	U100	37,000	203,000	71,100,000	961/1005
Mercury	6.60	82.0	176	231	138/157	3.00	190	740	52,000	831/958
Nickel	9,600	31,400	66,000	140,000	70/70	U2,000	31,800	56,200	368,000	900/905
Silver	14 0	140	386	2,260	130/150	7.00	340	1,800	8,270	798/888
Zinc	14,700	63,500	99,700	133,000	71/71	15,300	100,000	315,000	6,010,000	963/983
Organotin	U1 2	1.7	—	U6	1/3	U1.6	2.3	—	3	3/6
Nonionic Organic Compounds										
LPAH Compounds	2 0	42	160	3,600	48/70	U1.1	500	5,300	630,000	766/801
Naphthalene	U0 50	7 5	94	200	22/72	U0.10	140	1,500	52,000	580/881
Acenaphthylene	U0.10	14	—	490	4/66	U0.10	44	340	37,000	431/783
Acenaphthene	0.20	18	—	130	6/68	U0.10	61	740	81,000	500/846
Fluorene	U0.10	3.3	170	270	16/71	U0.10	73	970	84,000	576/897
Phenanthrene	2 0	12	150	1,500	41/72	U0.10	270	2,700	330,000	793/923
Anthracene	0 30	7 4	38	1,100	26/72	U0.10	120	1,400	190,000	710/915
1-Methylnaphthalene	1 0	4 5	—	16	8/10	U0.10	29	160	1,200	60/88
2-Methylnaphthalene	0 30	4	21	75	19/42	U0.30	61	450	23,000	435/643
1,3-Dimethylnaphthalene	—	—	—	—	—	U2 0	58	—	79	6/9
2,6-Dimethylnaphthalene	1.0	15	—	20	3/5	U2 0	23	200	1,400	48/68

TABLE IV. (Continued)

	1	2	3	4	5	6	7	8	9	10
	Sediments (ug/kg dry weight)[b]									
	Reference Areas[c]					Nonreference Areas[d]				
Pollutant of Concern	Minimum	Median	90th Percentile	Maximum	Detection Frequency	Minimum	Median	90th Percentile	Maximum	Detection Frequency
HPAH Compounds	17	120	420	28,000	53/69	0.40	1,900	20,100	3,200,000	793/807
Fluoranthene	3.0	19	98	5,000	47/73	U0.20	460	4,200	1,300,000	636/833
Pyrene	3.0	17	93	4,500	48/73	U0.40	510	4,700	740,000	635/833
Benz[a]anthracene	2.0	7.0	100	2,500	38/72	U0.10	230	2,100	300,000	706/909
Chrysene	2.0	11	67	6,000	43/71	U0.30	340	2,700	350,000	781/912
Total benzofluoranthenes	U4.0	23	100	5,200	28/63	U0.20	500	5,100	300,000	677/821
Benzo[e]pyrene	1.0	8.5	70	1,900	33/72	U0.10	240	2,400	100,000	723/904
Indeno[1,2,3-c,d]pyrene	1.0	6.6	420	1,000	15/65	U0.40	140	1,400	40,000	594/831
Dibenz[a,h]anthracene	0.40	3.7	—	390	6/64	U0.40	58	550	12,000	414/810
Benzo[g,h,i]perylene	2.8	5.0	420	1,300	16/63	U0.40	130	1,100	32,000	551/820
1-Methylphenanthrene	U2.0	8.0	—	280	9/12	0.70	68	390	100,000	241/278
2-Methylphenanthrene	5.0	8.0	—	200	5/5	2.2	83	480	110,000	184/187
3-Methylphenanthrene	390	—	—	390	1/1	1.3	66	870	97,000	68/68
Chlorinated Benzenes										
1,3-Dichlorobenzene	U0.05	4.0	—	4.0	4/63	U0.05	25	120	210	63/691
1,4-Dichlorobenzene	B0.5	14	—	23	2/63	U0.05	30	150	31,000	129/702
Hexachlorobenzene	0.01	0.025	—	0.04	2/72	0.02	3.0	130	730	71/764
Polychlorinated Dibenzofurans	—	—	—	—	—	—	—	—	—	—
Polychlorinated Dibenzo-p-dioxins										
2,3,7,8-Tetrachlorodioxin (TCDD)	—	—	—	—	0/0	U0.002	U0.002	0.031	0.080	—/34
PCBs										
Total PCBs	U0.10	7.2	37	41	18/73	U0.01	110	1,100	24,000	604/889
Miscellaneous Extractable Compounds										
N-Nitrosodiphenylamine	U0.50	—	—	—	0/54	0.20	28	370	950	51/494
Methylethyl ketone	—	—	—	—	—	—	—	—	—	—
Bis(2-ethylhexyl)phthalate	B0.50	62	2,000	2,800	13/57	B0.50	160	1,500	21,000	297/589
Hexachlorobutadiene	U0.63	0.20	—	0.20	1/66	0.01	12	290	940	76/712
Dibenzofuran	U1.0	14	—	130	5/40	U0.10	81	610	36,000	336/534

TABLE IV. (Continued)

	1	2	3	4	5	6	7	8	9	10
	Sediments (μg/kg dry weight)[b]									
	Reference Areas[c]					Nonreference Areas[d]				
Pollutant of Concern	Minimum	Median	90th Percentile	Maximum	Detection Frequency	Minimum	Median	90th Percentile	Maximum	Detection Frequency
Pesticides										
Aldrin	U0.01	—	—	—	0/43	U0.01	75	—	99	3/601
Dieldrin	U0.40	—	—	—	0/41	U0.01	10	—	51	3/601
4,4'-DDD	U0.08	—	—	—	0/83	U0.03	8.0	47	180	81/709
4,4'-DDE	U0.03	—	—	—	0/86	U0.01	2.0	16	69	140/783
4,4'-DDT	U0.02	—	—	—	0/84	U0.05	11	79	270	48/711
gamma-Hexachlorocyclohexane	U0.01	—	—	—	0/43	U0.01	10	—	10	5/597
Volatile Organic Compounds										
Benzene	0.094	—	—	0.094	1/8	U0.02	0.006	—	0.15	6/113
Chloroform	U0.07	—	—	0.25	1/24	U0.03	0.14	14	18	14/178
Ethylbenzene	0.03	0.047	—	0.063	2/32	U0.017	10	40	150	27/270
Toluene	U0.17	—	—	0.21	1/8	U0.05	0.23	37	64	14/158
Trichloroethene	U0.03	—	—	—	0/28	0.008	1.2	370	19,000,000	11/263
Tetrachloroethene	0.01	0.048	—	0.06	3/32	0.01	4.2	170	14,000,000	25/237
Total xylenes	0.25	—	—	0.25	1/12	U0.083	7.1	100	160	50/184
Ionizable Organic Compounds										
Phenols										
Phenol	U3.0	53	510	1,800	22/63	0.90	130	850	4,800	346/725
4-Methylphenol	U1.0	120	1,400	1,500	14/41	U0.20	160	1,400	100,000	237/552
Pentachlorophenol	0.10	—	—	0.10	1/59	U0.20	47	380	6,000	93/673
Resin Acids and Guaiacols										
2-Methoxyphenol (guaiacol)	1.3	—	—	1.3	1/5	U0.10	50	370	3,900	90/90
1,2-Chlorodehydroabietic acid	U130	—	—	—	0/3	61	780	4,600	11,000	19/28
1,4-Chlorodehydroabietic acid	U130	—	—	—	0/3	46	610	3,200	3,400	10/28
Dehydroabietic acid	20	30	—	40	2/4	34	1,200	19,000	83,000	32/33
Dichlorodehydroabietic acid	U110	—	—	—	0/4	U57	470	—	710	6/33
Chlorodehydroabietic acid	U110	—	—	—	0/1	U57	—	—	—	0/2

TABLE IV. (Continued)

	11	12	13	14	15	16	17	18	19	20	21	22	23
	Sediment (continued)					Fish and Shellfish Tissue (µg/kg wet weight)				Receiving Water (µg/L)			
	Urban Bays					Fish Tissue (All Areas)		Crab and Bivalve Tissue (All Areas)		Reference Areas		Nonreference Areas	
Pollutant of Concern	Min.	Median	90th Percentile	Max.	Detection Frequency	Min.	Max.	Min.	Max.	Min.	Max.	Min.	Max.
Inorganic Chemicals													
Antimony	U190	1,130	38,500	1,370,000	459/610	U10	1,700	U1,000	14,400	—	—	1.00	17.0
Arsenic	530	13,100	39,400	12,200,000	779/786	160	32,000	400	22,100	—	—	1.00	3,820
Cadmium	U37	700	3,100	184,000	737/768	U0.5	220	U2.0	1,610	—	—	0.10	28.0
Chromium	U3,000	47,000	126,000	1,080,000	649/657	U10	540	U8.0	820	—	—	1.00	320
Copper	1,000	57,000	191,000	14,300,000	758/785	U38	1,250	110	204,000	—	—	1.00	1,240
Cyanides	50	250	500	1,000	34/49	—	—	—	—	—	—	5.0	8.0
Lead	790	45,000	232,000	71,100,000	779/786	U0.5	200	U1	11,200	—	—	U1.0	1,750
Mercury	6.00	206	799	52,000	656/723	U0.5	280	1	1,500	—	—	U0.056	1.40
Nickel	U2,000	32,000	54,400	386,000	688/693	U10	1,100	U75	3,300	—	—	U1.0	435
Silver	7.00	378	1,900	8,270	626/693	U2.0	240	2	340	—	—	0.30	9.00
Zinc	18,400	113,000	359,000	6,010,000	764/764	1,700	11,000	7,200	130,000	—	—	U1.0	11,800
Organotin	—	—	—	—	0/0	—	—	—	—	—	—	—	—
Nonionic Organic Compounds													
LPAH Compounds	1.3	780	6,200	630,000	601/612	—	—	5.6	1,200	—	—	—	—
Naphthalene	1.0	170	1,600	33,000	495/676	U0.10	2,100	U10	1,200	—	—	U0.05	6.4
Acenaphthylene	U0.10	45	390	37,000	385/601	U2.0	U10	U10	U10	—	—	U0.05	U1.0
Acenaphthene	U0.40	62	670	33,000	444/658	U0.08	U10	U10	130	—	—	U10	U10
Fluorene	0.60	86	970	37,000	479/681	U0.11	U10	U10	180	—	—	U0.50	1.4
Phenanthrene	U1.0	340	2,800	330,000	641/707	U0.11	U10	U10	740	—	—	U0.50	U10
Anthracene	U1.0	140	1,300	190,000	588/699	U0.22	U10	U10	130	—	—	U0.50	U10
1-Methylnaphthalene	U5.0	31	200	1,200	47/53	—	—	—	63	—	—	—	—
2-Methylnaphthalene	U1.0	74	460	11,000	382/516	U2.0	U10	U10	160	—	—	—	—
1,3-Dimethylnaphthalene	23	56	—	79	6/6	—	—	—	75	—	—	—	—
2,6-Dimethylnaphthalene	U5.0	25	200	1,400	40/46	—	—	—	79	—	—	—	—

TABLE IV. (Continued)

	11	12	13	14	15	16	17	18	19	20	21	22	23
	Sediment (continued)					Fish and Shellfish Tissue (µg/kg wet weight)				Receiving Water (µg/L)			
	Urban Bays[d]					Fish Tissue (All Areas)		Crab and Bivalve Tissue (All Areas)		Reference Areas		Nonreference Areas	
Pollutant of Concern	Min.	Median	90th Percentile	Max.	Detection Frequency	Min.	Max.	Min.	Max.	Min.	Max.	Min.	Max.
HPAH Compounds	7.5	2,800	21,000	3,200,000	614/617	—	—	—	2,700	—	—	—	—
Fluoranthene	U1.0	610	4,300	1,300,000	667/716	U0.11	U10	U10	970	—	—	U0.50	U1.0
Pyrene	U1.0	630	5,100	740,000	662/716	U0.70	U10	0.97	920	—	—	U0.50	U0.50
Benz(a)anthracene	U1.0	310	1,900	300,000	629/684	U0.22	U10	U2	310	—	—	U10	U10
Chrysene	U1.0	460	2,700	350,000	628/701	U0.22	U10	U2	360	—	—	U0.5	U0.50
Total benzofluoranthenes	2.6	670	5,900	300,000	571/655	U10	U10	U2	330	—	—	U1.0	U1.0
Benzo(e)pyrene	U1.0	330	2,600	100,000	593/689	U0.11	U10	3.2	240	—	—	U1.0	U1.0
Indeno(1,2,3-c,d)pyrene	U0.50	160	1,500	40,000	474/627	U10	U10	U0.9	23	—	—	U10	U10
Dibenz(a,h)anthracene	U0.50	58	540	12,000	367/609	U10	U10	2.6	58	—	—	U1.0	U1.0
Benzo(g,h,i)perylene	U0.50	150	1,200	32,000	468/618	U10	U10	2.5	21	—	—	U1.0	U1.0
1-Methylphenanthrene	U1.0	68	390	100,000	226/248	—	—	—	—	—	—	—	—
2-Methylphenanthrene	2.2	83	460	110,000	182/183	—	—	—	—	—	—	—	—
3-Methylphenanthrene	1.3	66	870	97,000	68/68	—	—	—	—	—	—	—	—
Chlorinated Benzenes													
1,3-Dichlorobenzene	U0.5	25	120	210	63/522	U20	530	U20	110	—	—	—	—
1,4-Dichlorobenzene	U0.5	33	160	31,000	126/527	U20	U40	U20	U20	—	—	—	2.1
Hexachlorobenzene	0.02	5.0	130	730	67/576	U10	29	U0.6	11	—	—	—	—
Polychlorinated Dibenzofurans	—	—	—	—	—	U0.006	<0.199	<0.003	<0.101	—	—	—	—
Polychlorinated Dibenzo-p-dioxins						<0.006	<0.023	U0.009	0.064	—	—	—	—
2,3,7,8-Tetrachlorodioxin (TCDD)	U0.002	U0.002	0.031	0.060	—/34	U0.0004	0.005	U0.0001	0.0054	—	—	U0.010	U0.010
PCBs													
Total PCBs	U1.0	150	1,300	24,000	529/684	US	2,060	1.0	480	—	—	—	—
Miscellaneous Extractable Compound													
N-Nitrosodiphenylamine	0.20	28	370	950	51/357	U10	U10	U10	U10	—	—	U10	U10
Methylethyl ketone	—	—	—	—	—								
Bis(2-ethylhexyl)phthalate	B0.50	210	2,000	21,000	227/448	U10	2,100	U10	9,100	—	—	—	—
Hexachlorobutadiene	0.01	13	300	940	71/539	U40	70	U0.05	2.7	—	—	U20	U20
Dibenzofuran	U1.0	87	580	15,000	3[illegible]/434	U2.0	U10	U10	120	—	—	—	—

TABLE IV. (Continued)

	11	12	13	14	15	16	17	18	19	20	21	22	23
	Sediment (continued)					Fish and Shellfish Tissue (µg/kg wet weight)				Receiving Water (µg/L)			
	Urban Bays[d]					Fish Tissue (All Areas)		Crab and Bivalve Tissue (All Areas)		Reference Areas		Nonreference Areas	
Pollutant of Concern	Min.	Median	90th Percentile	Max.	Detection Frequency	Min.	Max.	Min.	Max.	Min.	Max.	Min.	Max.
Pesticides													
Aldrin	U0.10	75	—	90	3/469	U0.2	U10	U0.2	U2.4	—	—	U0.01	U0.01
Dieldrin	U0.10	10	—	51	3/468	0.28	U20	U0.3	U4.0	—	—	U0.01	U0.01
4,4'-DDD	U0.30	6.0	45	180	86/570	U0.6	U50	U0.1	0.57	—	—	U0.01	U0.01
4,4'-DDE	U0.10	2.0	21	69	127/616	U0.4	410	U0.1	7.2	—	—	U0.01	U0.01
4,4'-DDT	U0.05	13	82	270	46/568	U0.1	28/415S	U0.1/0.1SS	0.89/11S	—	—	U0.01	U0.01
gamma-Hexachlorocyclohexane	U0.01	10	—	10	5/464	U0.2	U10	U0.2	0.66	—	—	U0.01	U0.01
Volatile Organic Compounds													
Benzene	U0.30	—	—	—	0/91	U5	U5	—	—	—	—	—	—
Chloroform	U0.10	10	—	18	5/148	U5.0	U5.0	—	—	—	—	U1.0	420
Ethylbenzene	0.04	13	40	50	17/218	U5.0	48	—	—	—	—	U1.0	1.7
Toluene	U0.60	8.9	—	54	6/136	U10	17	—	—	—	—	—	—
Trichloroethene	U0.5	2.3	—	19,000,000	9/211	U5.0	U5.0	—	—	—	—	U1.0	93
Tetrachloroethene	0.01	68	190	14,000,000	17/185	U5.0	230	—	—	—	—	0.70	140
Total xylenes	0.10	14	110	180	39/140	U5.0	180	U5.0	U5.0	—	—	—	—
Ionizable Organic Compounds													
Phenols													
Phenol	0.90	130	690	2,900	286/556	U20	U40	U20	U40	—	—	2.1	80
4-Methylphenol	U0.30	180	1,500	100,000	212/419	U20	U40	U20	U20	—	—	—	—
Pentachlorophenol	U0.30	44	380	6,000	87/511	U40	U490	0.3	9.3	—	—	—	—
Resin Acids and Guaiacols													
2-Methoxyphenol (guaiacol)	U0.20	50	370	3,900	59/79	—	—	—	—	—	—	—	—
1,2-Chlorodehydroabietic acid	61	780	4,600	11,000	19/28	—	—	—	—	—	—	—	—
1,4-Chlorodehydroabietic acid	46	610	3,200	3,400	10/28	—	—	—	—	—	—	—	—
Dehydroabietic acid	34	1,400	20,000	83,000	29/30	—	—	—	—	—	—	—	—
Dichlorodehydroabietic acid	U57	600	—	710	5/30	—	—	—	—	—	—	—	—
Chlorodehydroabietic acid	U57	—	—	—	0/1	—	—	—	—	—	—	—	—

TABLE IV. (Continued)

[a] Information contained in each column is explained by column number in the text.

[b] Maximum values are highest detected concentrations. Minimum values are the lower of either the lowest detected concentration or the lowest detection limit for undetected values. Statistics (i.e., median and 90th percentile) are based on detected values only. Sediment samples were collected from 1979 to 1990.

B = Blank corrected down to detection limit shown.
S = Where two values are shown for DCT, the first value is DDT and the second value is the sum of DDT and its metabolites. The sum was used only when individual values were not reported.
U = Undetected at detection limit shown.
--- = Insufficient data are available.

[c] Reference sediment areas are Carr Inlet, Case Inlet, Sequim Bay, Samish Bay, Dabob Bay, Port Susan, and Point Williams.

[d] Nonreference sediments include urban bays and adjacent areas, such as Central Puget Sound, which exhibit elevated contamination above that found in more rural reference areas.

[e] Urban bays are Commencement Bay, Elliott Bay, Everett Harbor, and Sinclair Inlet.

[f] Fish tissue ranges include data for different species of bottom fish (e.g., English sole), bottom-feeding fish (e.g., Pacific cod), and pelagic fish (e.g., salmon). Crab and bivalve tissue ranges include data for dungeness and rock crabs, and various species of clams, mussels, and oysters.

< = The total value shown (for total chlorinated dioxin and furan congeners in tissue samples) includes a detection limit for at least one congener.

3. DESCRIPTION OF POLLUTANTS OF CONCERN IN PUGET SOUND

In this chapter, chemical characteristics, common uses, exposure routes, health effects, sources, and environmental fate are discussed for each pollutant of concern in Puget Sound when that information is available. Most of the information provided is generally available from reports published by the Agency for Toxic Substances and Disease Control Registry (a unit of the U.S. Public Health Service), Howard (1989, 1990), and Casarett and Doull (1986).

INORGANIC CHEMICALS

Descriptions are provided in this section for the 12 inorganic chemicals listed in Table 1 (see Chapter 1), including cyanides and organotin. Three inorganic chemicals that have been designated priority pollutants by EPA in 40 CFR 401.15 (i.e., beryllium, thallium, and selenium) were not recommended by Puget Sound experts during development of the pollutants of concern for this report. Although toxic, beryllium and thallium have not been found in Puget Sound in concentrations that exceed reference levels. Selenium was found in elevated concentrations in only one Puget Sound study. Spectral interferences occurring in connection with the particular instrumental analyses used were considered the probable cause of the elevated results (PSEP 1989a).

Antimony

Antimony is a natural element that is found in over 100 mineral species. Antimony is not an abundant element and is usually found as a sulfide rather than in the pure form. The brittle character of antimony improves the hardness and lowers the melting point of certain alloys. Antimony compounds are widely used for the production of flame retardants, fireworks, matches, ammunition, and storage batteries.

Exposure Routes and Risks--The greatest exposure to antimony for humans occurs in connection with selected occupations (e.g., mining, industrial processing of antimony-containing ores, and commercial uses of antimony

trioxide). Humans may ingest antimony in small amounts in water and food. Inhalation is also an exposure route, especially in the vicinity of smelting facilities and municipal incinerators. However, environmental exposures from all media appear to be minor sources of antimony for humans.

Although antimony is toxic to mammals, including humans, few epidemiological studies of antimony have been conducted due to the lack of recognizable public health problems associated with overall low levels of exposure to antimony. Limited information is available concerning toxicity to freshwater organisms, but there is almost no information concerning the acute or chronic toxicity of antimony to saltwater organisms.

Sources and Fate—Antimony may enter aquatic systems from the natural weathering of rocks, in soil runoff, in effluents from manufacturing facilities, and from municipal discharges. Smelting operations, fossil fuel combustion, and municipal incineration contribute to atmospheric antimony. Industries involved in the manufacture of alloys, paints, lacquers, glazes, glass, and pottery and in the production of organic chemicals are sources of antimony. Other sources include copper smelters, chloralkali plants, and smelter slag. The semiconductor industry also uses antimony in the production of infrared detectors and diodes. Antimony has been detected, often at very high levels, in 74 percent of sediment samples taken from nonreference areas in Puget Sound (Table IV). The highest levels of antimony in Puget Sound [several thousand parts per million (ppm)] are associated with copper smelter wastes (Tetra Tech 1985b).

Solubilities of antimony compounds range from insoluble to fully soluble. Inorganic antimony compounds may be only slightly water soluble or may decompose in aqueous media. Certain compounds undergo hydrolysis or oxidation and are not environmentally persistent. However, antimony associated with smelter wastes in Puget Sound is expected to be highly persistent (Tetra Tech 1985b; 1986c).

Arsenic

Arsenic is a naturally occurring element not commonly found in its pure state. Arsenic generally combines with one or more other elements. For example, arsenic commonly combines with oxygen, chlorine, or sulfur to form the inorganic species, while arsenic can also combine with carbon and hydrogen to form the organic species. Arsenic exists in a variety of chemical forms in marine and estuarine ecosystems, including inorganic species, methylated forms, arseno-lipids (fats), arseno-sugars, and other biochemical forms. Data for total

arsenic are difficult to interpret in relation to effects in a particular environment because of the multiplicity of organic and inorganic chemical species involved.

Exposure Routes and Risks—All humans are exposed to low levels of arsenic because of its wide distribution in the environment. The Pacific Northwest, in particular, contains naturally high levels of arsenic because of the erosion of rocks that are enriched in arsenic. Greater than average exposure may occur in certain occupations (e.g., metal smelting, wood preservation, and pesticide application) or because of proximity to natural mineral deposits, chemical waste disposal sites, or industrial sources.

Most humans are exposed to arsenic by ingesting food containing arsenic; drinking water and air provide lower amounts. Although some arsenic may be absorbed by the skin, skin absorption is a much less important exposure route than ingestion.

In general, the toxicity of arsenic is a function of its chemical and physical form. Organic arsenic is less toxic than inorganic arsenic. Therefore, because organic arsenic predominates over inorganic arsenic in most fish, macroalgae, mussels, and shrimp, health concerns from consumption of arsenic-contaminated tissues are generally low. Arsenic compounds that are more soluble tend to have more acute toxicity. For example, arsenic (III) compounds are generally more toxic than arsenic (V) compounds. Inorganic arsenic has been recognized as a human poison since ancient times; ingestion of large doses causes death. Ingestion of lower doses produces a variety of systemic effects. Animal studies suggest fetal effects, but this area has not been well studied in humans. Mottled pigmentation and small lesions on the skin are the most common indication of chronic oral exposure to inorganic arsenic. There is clear evidence that such lesions can develop into skin cancer. Although inhalation exposure causes much milder systemic effects, there is growing concern over the threat of lung cancer, especially with occupational exposure.

Inorganic arsenic is a recognized human carcinogen. However, there are few human studies of carcinogenicity and toxicity of arsenic that have included different arsenic forms. An additional complicating factor is the limited utility of animal studies as predictors of human effects because of the greater sensitivity to arsenic by humans. While there is evidence that exposure to low levels of arsenic has beneficial health effects in some species (e.g., rats, goats, chicks, and minipigs) (U.S. EPA 1984d), the beneficial daily dose is quite small (i.e., 10-50 μg/day). This amount is normally supplied in a human diet that does not include seafood, which may increase the daily intake to 200 μg/day (Casarett and Doull 1986).

Toxicity and other effects of arsenic on aquatic life are significantly modified by changes in water temperature, pH, organic content, phosphate concentrations, suspended solids, and presence of other substances and toxicants, as well as with arsenic speciation and duration of exposure. Large interspecies differences in toxicity are recorded even among species that are closely related taxonomically (U.S. FWS 1988).

Sources and Fate—Combustion of fossil fuels is a major source of arsenic in the atmosphere. Tobacco smoke is another source. No arsenic is currently produced in the United States except as a by-product of other operations, such as the smelting of copper, lead, zinc, gold, and silver ores. However, arsenic is imported for use in wood preservatives, pesticides, and herbicides. Use of arsenic-containing pesticides has declined in recent years because of the development of less broadly toxic substitutes. Arsenic is also used as an alloy additive to lead and copper and in the manufacture of low-melting glasses. Although relatively small, use of arsenic in transformers is increasing. Consumer products that once contained arsenic (e.g., paints, dyes, and rat poisons) are no longer in general use; therefore, exposure from these sources is minimal.

A former copper smelter plant located in Tacoma may have contributed as much as 25 percent of the total amount of anthropogenic arsenic in the world (Phillips 1990). In the United States, arsenic trioxide was produced only at this smelter, which closed in January 1986. Although 90 percent of United States surface waters contain less than 10 parts per billion (ppb) arsenic, higher concentrations have been reported in the Puget Sound area (up to 3,800 ppb) because of smelting operations and natural sources of arsenic (Crecelius et al. 1975).

Arsenic may be released to the atmosphere as a gas vapor or adsorbed to particulate matter. However, most arsenic in the air is adsorbed to particulate matter. It may be transported to other media by wet or dry deposition; photolysis is not considered an important fate process for arsenic compounds.

Arsenic in surface water can undergo a complex pattern of transformations. Arsenic is extremely mobile in aquatic systems and, as a result, rivers are a major source of arsenic to Puget Sound and oceans. Sorption to clays, iron oxides, manganese compounds, and organic materials is an important fate of arsenic in surface waters. However, as waters become more alkaline or saline, arsenic is less likely to be adsorbed. Arsenic in water and soil may be reduced and methylated by fungi, yeasts, algae, and bacteria, and these forms may volatilize and escape into the air. The rate of volatilization may vary considerably, depending on soil conditions (aerobic or anaerobic), pH, and the presence or absence of microbes.

Plants may accumulate arsenic via root uptake from soil solution, and certain species may accumulate substantial arsenic levels. Bioconcentration of arsenic also occurs in aquatic organisms, primarily in algae and lower invertebrates. However, biomagnification in aquatic food chains does not appear significant, although some fish and invertebrates contain high levels of arsenic compounds that are relatively inert toxicologically. In Puget Sound, higher concentrations of arsenic are accumulated by invertebrates and mussels located near pollutant sources, but there is no evidence for increased accumulation of arsenic in Puget Sound fish near industrial sources relative to reference conditions or for biomagnification of arsenic in the food chain (Ginn and Barrick 1988).

Cadmium

Cadmium is a naturally occurring element usually encountered in combination with other elements such as oxygen, chlorine, or sulfur. Cadmium compounds are all stable solids that do not evaporate.

Exposure Routes and Risks—For humans who are not subject to occupational exposure to cadmium, the primary exposure route is ingestion of food containing cadmium. Contamination of topsoil through the application of phosphate fertilizers or sewage sludge increases cadmium levels in foods. Another source of cadmium is tobacco smoke. Smokers have approximately twice as much cadmium in their bodies as nonsmokers. U.S. EPA (1990c) has designated cadmium a probable human carcinogen by the inhalation route.

Cadmium is not known to have any beneficial health effects, but can cause a number of adverse effects on humans and other organisms, including death. Although high-level exposures are rare, there is great concern regarding the effects of low-level, long-term exposure. Soluble cadmium compounds have greater toxicity than insoluble cadmium compounds because they are more readily absorbed by the body. Typical levels of cadmium exposure through ingestion of food and water or through inhalation of air (approximately 0.0004 mg/kg per day) are not a major health concern. However, cadmium is metabolized by humans very slowly and even low doses can result in the accumulation of toxic levels if the exposure continues for a long period of time. Because of the severe effects of cadmium exposure, there is pending federal legislation proposing a ban on the use of cadmium in pigments and on other nonessential uses.

Sources and Fate—Most cadmium in the United States is obtained as a by-product from the smelting of zinc, lead, or copper ores. Cadmium is used

primarily in metal plating and the manufacture of pigments, batteries, and plastics. Cadmium is not often encountered at levels of concern in water, although it can leach into water from pipes and solder or may enter water from chemical waste disposal sites. The largest source of cadmium to the general environment is the combustion of fossil fuels (such as coal or oil) or the incineration of municipal waste materials.

Cadmium in the atmosphere is persistent and easily respirable. It can be transported to soil and water through wet or dry deposition. Cadmium in surface water is relatively mobile. Because cadmium exists only in the 2+ oxidation state, aqueous cadmium is not strongly influenced by the oxidizing or reducing potential of water. Sorption by clays and iron oxides is important for reducing cadmium in water. Cadmium is not reduced or methylated by microorganisms.

Cadmium is readily accumulated by all organisms, through both food and water. Cadmium accumulates in freshwater and marine organisms at concentrations hundreds to thousands of times higher than the concentrations found in ambient water (Callahan et al. 1979). Aquatic bioconcentration is greatest for invertebrates such as molluscs and crustaceans (bioconcentration factors range up to 250,000), followed by fish and aquatic plants. Bioconcentration factors may range up to 1,000 for aquatic plants and up to 3,000 for fish (Callahan et al. 1979). Typical concentrations of cadmium in organisms from nonpolluted areas range from 1 to 10 μg/kg in fish and from 100 to 1,000 μg/kg in shellfish (U.S. EPA 1981; Casarett and Doull 1986). These values are similar to those documented in Puget Sound (Table IV). The highest concentration of cadmium in Puget Sound sediments is associated with smelter wastes in Commencement Bay (Tetra Tech 1985b).

Chromium

Chromium is a lustrous metal found in crystal or powder form. It is a natural element occurring primarily as the mineral chromite. Since 1961, all chromium ores have been imported by the U.S rather than mined. Chromium is widely distributed in the environment and occurs in four different oxidation states. Only two of these forms, trivalent chromium [Cr (III)] and hexavalent chromium [Cr (VI)], are environmentally important. Trivalent chromium is more common and stable than hexavalent chromium, which is the more toxic and commercially important form. In addition, hexavalent chromium is a strong oxidizing agent.

Exposure Routes and Risks—The primary routes of exposure to chromium for humans are inhalation, ingestion, and dermal contact. Although the general public is continuously exposed to trace amounts of chromium, occupational exposure is the major human health concern.

Chromium is an essential nutrient in trace amounts, but excess hexavalent chromium causes kidney damage, birth defects, and genetic mutations in animals and humans. The adverse health effects of excess trivalent chromium are similar, but much higher concentrations are required to produce these effects (U.S. EPA 1986a). Therefore, analysis for total chromium in environmental samples may not be sufficient to establish toxicity. It is not known whether trivalent chromium is a carcinogen. Although no data are available concerning the acute toxicity of trivalent chromium to saltwater organisms, hexavalent chromium is highly toxic to aquatic organisms.

Sources and Fate—Chromium is widely used in electroplating and metal finishing, in paints and fungicides, as a catalyst, and as a component of wood preservatives. Chromium can be found in emissions from copper smelters and coal combustion and in slag used as fill and sandblast grit. The two major sources of hexavalent chromium are cooling towers and chrome plating wastes. Although chromium has been detected in fish, water, and sediments throughout Puget Sound (Table IV), data are lacking on the relative amounts of trivalent and hexavalent chromium. However, most chromium in Puget Sound appears to be associated with natural sources (Barrick et al. 1988).

The dominant fate process for chromium in the aquatic environment varies with the species of chromium. While trivalent chromium rapidly adsorbs to particulate material, hexavalent chromium does not. Hexavalent chromium is accumulated by marine animals, but the trivalent form is not accumulated to a significant degree. Ultimately, the fate of both forms of chromium depends on a number of factors including pH, dissolved organic carbon levels, sediment organic matter content, and physical properties of sediments.

Copper

Copper is a metal that occurs naturally in rock, soil, water, sediment, and air, as well as in plants and animals. It is an essential element for all living organisms. Copper compounds occur both naturally and anthropogenically. Copper is mined extensively and is extremely malleable.

Exposure Routes and Risks—Because copper is common in the environment, humans may be exposed through inhalation, ingestion, and dermal contact. However, exposure to humans is likely to be limited because copper binds strongly to dust and dirt or becomes embedded in other materials. Such copper is not easily absorbed. Copper found in hazardous waste sites is usually in this particulate form. Soluble copper compounds used most commonly in agriculture may pose a greater health concern because they may be taken up by plants and animals or released into rivers and lakes. The risks from the transport of soluble copper compounds to surface water and groundwater may be lessened because they are rapidly adsorbed to particles.

Humans normally consume approximately 1 mg of copper per day. Although a large single dose of copper may cause kidney and liver damage or death, the body has effective mechanisms for blocking the entry of excess copper. Infants and small children are more sensitive to copper than adults, although there is no evidence regarding human reproductive or fetal effects from excessive exposure to copper. Copper is not a known carcinogen based on animal studies to date. There is little information regarding copper toxicity in humans, and the effects of inhalation and dermal contact have not been well studied. However, copper can be highly toxic to aquatic organisms at concentrations that are only somewhat higher than nutritional doses. Because copper is highly toxic to marine plants, copper-containing paints and preservatives have been widely used to control algal growth on boats and piers.

Sources and Fate—Copper is used primarily as an alloy in the manufacture of wire, sheet metal, pipe, and other metal products, especially brass and bronze. Copper is also used in agriculture to treat plant diseases such as mildew. Other uses include water treatment (as an algicide) and preservative treatments for wood, leather, and fabrics. Consequently, copper may be found in the effluents of industries associated with those uses.

Several processes determine the fate of copper in water including sorption, complex formation, and bioaccumulation. In addition, the fate of copper is highly dependent on pH, biological activity, and the presence of competing heavy metals. The bioconcentration factor of copper in fish is 10-100, which is relatively low compared with other species that easily bioaccumulate. For example, bioconcentration factors in oysters may reach 30,000 (Callahan et al. 1979; U.S. EPA 1984b). The absence of substantial bioaccumulation of copper by Puget Sound fish relative to levels of sediment contamination in some areas of the sound is consistent with the considerable ability of marine fish to regulate levels of most metals in muscle tissue (Ginn and Barrick 1988).

Cyanides

Cyanides are a diverse group of organic and inorganic compounds. Hydrogen cyanide is the best known and most toxic of the cyanides. It occurs rarely in nature and is usually prepared commercially from ammonia and methane. Cyanide ions can react with a variety of metals to form insoluble metal cyanides. Iron cyanides (ferricyanides and ferrocyanides) have a variety of industrial uses.

Exposure Routes and Risks—Cyanide is readily absorbed by humans through the skin and all mucous membranes. Inhalation is also an exposure route, although alkali salts of cyanide are toxic only if ingested. In general, an organism exposed to cyanide will either quickly metabolize the substance or be killed. In the case of hydrogen cyanide, death is caused through interference with the enzymes associated with cellular oxidation. Death occurs within minutes to several hours after exposure to even small amounts of sodium or potassium cyanide, depending on the exposure route. Death is more rapid after inhalation of cyanide fumes.

Sources and Fate—Cyanide is found in certain rat and pest poisons, silver and metal polishes, photographic solutions, and fumigants. It may be found in the effluents of copper smelters and metal manufacturing facilities.

The data on fate of cyanides in the aquatic environment are inconclusive and should be interpreted with caution. Volatilization and biodegradation appear to be the dominant processes affecting aquatic cyanide. Cyanides are sorbed by organic materials and, to a lesser extent, clay minerals. Hydrogen cyanide is not strongly partitioned into benthic or suspended sediments, primarily due to its high water solubility. However, the simple metal cyanides are insoluble and probably accumulate in sediments. Complex metal cyanides are transported in solution through the water column. Changes in the concentration ratio of metals to cyanide can alter the behavior of the metal-cyanide compounds. If the metals become more prevalent, formation of the simple metal cyanides is favored; if the cyanide becomes more prevalent, the complexed forms occur. Iron cyanides do not release cyanide without exposure to ultraviolet light; thus, sunlight can cause mobilization of cyanide in water containing iron cyanides.

There is little potential for bioaccumulation of cyanides; hydrogen cyanide is either metabolized or is fatal to organisms. Metal cyanides are less toxic and may have a greater tendency to bioaccumulate. Cyanides are biodegraded at low concentrations by almost all organisms.

Lead

Lead is a naturally occurring heavy metal that is a major constituent of more than 200 minerals, the most common of which is galena. The metal is bluish-white, very soft, highly malleable, ductile, a poor conductor of electricity, and very resistant to corrosion.

Exposure Routes and Risks—Humans accumulate lead by ingestion of food, water, and soil; through exposure to other sources such as ink and paint; and by inhalation of atmospheric lead. Controversy exists over the relative importance of these sources and their contribution to the toxic effects of lead in humans.

Exposure to lead has been linked to a wide range of effects in humans, including neurological disorders and impairment of blood synthesis. Considerable research has been conducted on lead levels in human tissues and on correlations between levels of lead in children and impaired intellectual development. Certain lead compounds (i.e., lead acetate and lead sulfate) have been designated probable human carcinogens by U.S. EPA (1990c) based on animal tests.

Little research has been conducted on the effects of lead on aquatic organisms, particularly marine organisms. Acute toxicity to lead has been documented for 13 saltwater species at concentrations in water that range from 315 to 27,000 ppb (U.S. EPA 1986a). Chronic effects have been documented for mysids and some species of macroalgae. In addition, lead can suppress reproduction in marine polychaetes.

Sources and Fate—The primary sources of lead to the environment include discarded batteries, leaded gasoline spills or leaks, motor vehicle emissions, paints, inks, dyes, and the emissions and slag of copper smelters. Lead is also used in some chemical processes and is present in solder and plumbing. Because lead is ubiquitous in urban areas, urban runoff is a common source of lead pollution. Concentrations of lead in sediments from urban bays of Puget Sound can be as much as 10 times higher than those found in reference areas of Puget Sound and, in isolated cases, may be as much as 1,000 times higher (Tetra Tech 1985b, Beller et al. 1988, Pastorok et al. 1988).

The fate of lead is influenced by the particular oxidation state [i.e., Pb (0), Pb (II), or Pb (IV)]. Lead exists principally as Pb (II) in most waters, and adsorption to sediments and suspended sediments is the predominant fate. Lead tends to form complexes with organic materials in water. Benthic microorgan-

isms can methylate inorganic lead to form a more volatile and toxic form. Bioaccumulation occurs with weakly sorbed lead, although biomagnification is not generally significant. Bioconcentration factors tend to decrease as the trophic level increases.

Mercury

Mercury is a naturally occurring element used in its pure form in many consumer products (e.g., thermometers and barometers). It is also found in compounds with other chemicals such as chlorine. Organic mercury (e.g., methyl mercury) can become highly concentrated in the flesh of carnivorous freshwater and saltwater fish (e.g., concentrations in pike and swordfish have exceeded 1 μg/g in tissue) (U.S. EPA 1984c). Therefore, otherwise low levels of mercury contamination in oceans and lakes can lead to contamination of these fish that may be toxic to humans. Mercury that is released to the environment remains there indefinitely and its form (inorganic or organic) may change over time.

Exposure Routes and Risks—Because mercury occurs naturally, exposure to low levels from all media is continuous. Certain segments of the general population are exposed to higher levels of mercury through the consumption of large amounts of fish that accumulate mercury. Inhalation of mercury associated with occupational exposure also is a source of higher levels of mercury. Occupations posing greater than normal risks of exposure include health-related, chemical, metallurgical, electrical, automotive, and building industries.

Mercury easily enters the body via inhalation and ingestion, and some mercury may be absorbed through the skin. In particular, methyl mercury is readily accumulated by fish, which has led to major concerns for human health from the consumption of mercury-contaminated fish. Mercury that has entered the body may take months to be eliminated. Long-term exposure to either organic or inorganic mercury can irreversibly damage the brain, kidneys, and developing fetuses. The form of mercury and the route of exposure influence the severity of health effects (ATSDR 1989a). For example, ingestion of organic mercury in contaminated fish or grain causes greater damage to the brain and to developing fetuses than to the kidneys. Inhaled inorganic mercury vapor tends to cause greater harm to the brain. Inorganic mercury ingested in contaminated food or water tends to cause greater harm to the kidneys. Short-term exposure causes similar effects, although full recovery is more likely. Mercury has not been proven carcinogenic. Effects of mercury reported in studies of aquatic

organisms included increased mortality, reduced growth and reproduction, and skeletal abnormalities. Mercury is considered the most toxic of the heavy metals to aquatic organisms.

Sources and Fate—Mercury in the environment has both natural and anthropogenic sources. The major source of atmospheric mercury is global degassing of mineral mercury from the soil and water, at a rate of approximately 30,000 metric tons per year (U.S. EPA 1984c). Subsequent deposition of atmospheric mercury in the ocean (approximately 11,000 metric tons per year) is the predominant source to the marine environment; land runoff accounts for less than half this amount. Releases of mercury to the air by human activities are estimated at 2,000-10,000 metric tons per year, mostly from the mining and smelting of mercury ores, industrial processes, and combustion of fossil fuels (especially coal). Other sources of emissions may include chloralkali manufacturing facilities, copper and zinc smelting operations, paint application, and waste oil combustion. Of these, fossil fuel combustion is the largest source.

Weathering of mercury-bearing minerals in rocks releases approximately 800 metric tons of mercury per year to surface waters. Mercury is also released to surface waters in effluents from numerous industrial sources including mining operations, ore processing, chloralkali production, metallurgy and electroplating, leather tanning, and the manufacture of chemicals, ink, paper, pharmaceuticals, and textiles. Mercury was used in marine paint to control mildew and barnacles before the 1970s, contributing to contamination of sediment near marinas and ship repair facilities. Mercury is released to cultivated soils through the direct application of inorganic and organic fertilizers (e.g., sewage sludge and compost), lime, and fungicides. Additional releases occur through the disposal of industrial and domestic products (e.g., thermometers, electrical switches, batteries) as solid wastes in landfills. In Puget Sound, a major historical source of mercury has been a chloralkali plant in Bellingham (Bothner 1973).

The global cycling of mercury is characterized by degassing from soils and surface waters, followed by atmospheric transport, deposition to land and surface waters, and sorption to soil and sediments. The atmosphere is the smallest environmental reservoir for mercury, containing only about 1,000 metric tons. Mercury is removed from the atmosphere through wet and dry deposition and by the sorption of mercury vapor to soil and surface waters. Transport and partitioning of mercury in surface waters and soils is a function of the particular form of mercury. Volatile forms evaporate to the atmosphere, while solid forms partition to sediments or suspended sediments or are transported in the water column, depending on solubility. Nonvolatile forms of mercury sorb to soil and sediment. Bacteria common to most waters are capable of converting virtually

any mercury compound to toxic methyl mercury, which has significance in bioaccumulation.

Nickel

Nickel is a natural element that forms compounds with sulfur and oxygen. Nickel is silvery-white, hard, malleable, ductile, somewhat ferromagnetic, and a fair conductor of heat and electricity. The commonly occurring oxidation states of nickel are Ni (0), Ni (I), and Ni (III). In addition to natural sources, nickel is produced either as a by-product from copper refining or is recycled or reclaimed from secondary sources.

Exposure Routes and Risks—Occupational inhalation of nickel, especially in the nickel-refining industry, presents the most serious exposure risk to humans. However, humans may also be exposed to nickel by ingestion or dermal contact because nickel is present naturally in the environment. Dietary nickel levels have been estimated to range from 100 to 300 μg/day and average 165 μg/day (Casarett and Doull 1986).

There is growing evidence that nickel may be an essential trace element for mammals. However, nickel is not well absorbed by the body and is excreted almost completely within 5 days. Dermal exposure may also cause allergic reactions. There is limited evidence of the carcinogenicity of nickel and certain nickel compounds in humans. However, there is sufficient evidence indicating carcinogenic effects to workers exposed to nickel sulphide fumes and dust at nickel-refining facilities. Reduced larval survival and growth were reported in studies of aquatic organisms exposed to high levels of nickel. Nickel is apparently quite toxic to freshwater algae at concentrations of approximately 50 μg/L; acute toxicity to saltwater species may occur as low as 150 μg/L (e.g., juvenile mysid) (U.S. EPA 1986a).

Sources and Fate—Nickel occurs naturally and enters the environment through natural weathering processes. Nickel is also released as industrial effluent or as atmospheric emissions from its use in electroplating, in metallic alloys, as a catalyst for various organic processes, in batteries, and in enamels, ceramics, and glass. Nickel is widely used to produce stainless steel, coins, nickel steel for armor plate, and as a catalyst for hydrogenating vegetable oils. Nickel has also been found in the effluents of copper smelters, chloralkali plants, pulp mills, and dry-cleaning facilities.

Nickel has been found at somewhat elevated concentrations in some sediments from nonreference areas of Puget Sound (Table IV). However, the mean concentration of nickel is virtually identical in reference and nonreference areas of the sound, suggesting a predominantly natural source.

Nickel can be extremely mobile in aquatic systems because many nickel compounds are highly soluble in water. However, in unpolluted environments, nickel is primarily associated with suspended particles and organic material or is coprecipitated with hydrous iron and manganese oxides. Photolysis and volatilization are not important fate processes for nickel. Although nickel is bioaccumulated, the bioconcentration factors suggest that partitioning into biota is not an important fate process.

Silver

Silver is an element that occurs naturally as a soft metal. A common anthropogenic source of silver is the disposal of photographic wastes in municipal wastewater treatment systems. Silver from photographic processes is usually released as soluble silver thiosulfate, which is converted to insoluble forms during wastewater treatment. Environmentally important forms of silver include silver nitrate and silver sulfide, which are the forms usually found at hazardous waste sites.

Exposure Routes and Risks—In the aquatic environment, silver is one of the most toxic metals to organisms, especially invertebrates and fish (U.S. EPA 1980). The toxicity of silver to aquatic organisms ranks second only to mercury among the heavy metals (Table II). Most people are exposed to very low levels of silver, primarily through food and drinking water, which come at least in part from naturally occurring silver in soil and water. Occupational exposure is another source, especially in medicine, jewelry-making, soldering, and photography. Silver may also enter the body by inhalation or dermal contact; however, these are much less important routes of exposure to silver than ingestion. Most of the silver that is consumed or inhaled leaves the body within 1 week. Little information is available about the fate of silver in the body resulting from dermal contact.

Greying of the skin and other body tissues is a well-known and permanent result of long-term exposure to silver compounds. Generally, many exposures to silver are required to produce this effect. Inhalation of dust containing relatively high levels of silver compounds (e.g., silver nitrate or silver oxide) may cause breathing problems, lung and throat irritation, and stomach pain. Dermal

exposure has caused mild allergic reactions in some people. Higher occupational exposures may cause kidney problems, but existing studies are inconclusive. There are no other reported health effects of silver exposure.

Sources and Fate—Discharges of photographic materials into wastewater is the major source of silver released into the environment. It is also released as a by-product from the mining of copper, lead, zinc, and gold ores. Direct mining of silver is another large source. Hazardous waste sites are sources of silver compounds, and silver is released naturally through the weathering of silver-bearing rocks and soil.

Other important sources of silver include the manufacture of electrical contacts, silver paints, and batteries, as well as steel refining, cement manufacturing, fossil fuel consumption, municipal waste incineration, and cloud seeding. Ore smelting and fossil fuel consumption processes emit fine particles of silver to the atmosphere. Sources of elevated dietary silver include seafood from areas near sewage outfalls or industrial sources and crops grown in areas with high ambient levels of silver in the air or soil.

Silver is capable of being transported long distances in air and water. It is stable and remains in the environment until it is mined, but may change forms depending on environmental conditions. Sorption and precipitation are the dominant processes controlling partitioning in water and movement in soil. Silver may leach from soil into groundwater. Silver is bioconcentrated to a moderate extent in fish and invertebrates. There appears to be little potential for silver biomagnification in tested aquatic food chains (Callahan et al. 1979).

Zinc

Zinc is found naturally in air, soil, and water and is present in all foods. It is an essential nutrient for all organisms in trace amounts. As a refined metal, zinc is used in its pure form, as an alloy with other metals, and in compounds with chemicals.

Exposure Routes and Risks—Zinc enters the body easily through ingestion and inhalation. Dermal absorption is a relatively small exposure route for zinc. Most zinc not needed by the body is excreted. Higher than average exposure to zinc can occur from drinking water that has been stored in galvanized metal containers, from sources contaminated with industrial zinc wastes, and from the air at galvanizing, smelting, welding, or brass foundry operations. Higher levels

of exposure can also result from use of dietary supplements and by injection of certain drugs (e.g., insulin) that contain zinc salts.

Gastrointestinal problems can occur after ingestion of excess zinc; severity of the problems can increase with long-term ingestion. Excess zinc may also interfere with the body's ability to absorb and use other essential minerals such as copper and iron. Inhalation of zinc may cause metal fume fever, a temporary syndrome. Zinc is not known to cause cancer or birth defects. Insufficient levels of zinc have also been associated with adverse health effects. Significant decreases in reproduction and growth have been reported in fathead minnows, guppies, and polychaetes after exposure to zinc.

Sources and Fate—Zinc is used most commonly as a protective coating for other metals. In addition, it is used in alloys such as bronze and brass, for electrical uses, and in organic chemical extractions and reductions. Zinc chloride is a primary ingredient in smoke bombs. Salts of zinc are used as solubilizing agents in many drugs (e.g., insulin). Zinc and copper alloys are used in coinage.

Zinc is released to the air as dust and fumes from zinc production facilities, lead smelters, brass works, automobile emissions, fuel combustion, incineration, and soil erosion. Refuse incineration, coal combustion, smelter operations, and metallurgical industries are major sources of zinc in air. Zinc releases to air account for only a small portion of the total environmental release. Erosion of soil particles containing zinc is the largest overall source of zinc to the aquatic environment but the effect on water quality at any one location is likely minor (ATSDR 1989b). More concentrated sources of zinc to aquatic environments include urban runoff, mine drainage, and municipal and industrial effluents. Metal corrosion and tire abrasion also contribute to urban runoff. Industries that directly discharge zinc to water include iron and steel, zinc smelting, plastics, and electroplating. Municipal wastewaters are major contributors of zinc in estuarine environments.

Adsorption to sediments is the dominant fate of zinc in the aquatic environment. Zinc partitions to sediments or suspended solids in surface waters through adsorption onto hydrous iron and manganese oxides, clay minerals, and organic material. The tendency of zinc to be adsorbed is affected by the nature and concentration of the sorbent, and by the pH and salinity of the water. Zinc tends to sorb more readily at higher pH levels and desorption of zinc from sediments occurs as salinity increases. Zinc does not volatilize significantly from water. Although bioaccumulated by all organisms, zinc probably does not biomagnify in the food chain (Callahan et al. 1979; Ginn and Barrick 1988).

ORGANOTINS

Use of organotins (including methyltins, butyltins, and ethyltins) as the active biocide in antifouling paints for ships and marine structures has increased dramatically over the past two decades. Organotins, especially tributyltin (TBT), have been targeted for environmental concern. In 1988, Congress passed the Organotin Antifouling Paint Control Act to restrict the use of organotins. Analysis of organotins is made more difficult because they behave as both metals and organic compounds, and their aquatic chemistry is not completely understood.

Exposure Routes and Risks—The primary risk of exposure to organotins is from leaching of paint into ambient water, where the organotins are adsorbed to suspended or benthic sediments. Organotins are bioaccumulated by aquatic organisms exposed to contaminated sediments, but no action levels or other maximum acceptable concentration in tissue has been established.

Organotins are very toxic to marine organisms and TBT is the most toxic of the organotins. TBT is acutely toxic, by design, to aquatic life at concentrations exceeding 0.5 μg/L, with acute lethal concentrations reported for embryo, larval, and post-larval clams, mussels, and oysters at concentrations of approximately 0.6—4.0 μg/L (Center for Lake Superior Environmental Studies 1988). TBT bioconcentration factors for mussels and oysters have been reported in the range of 6,800 to 11,400 (Center for Lake Superior Environmental Studies 1988).

Sources and Fate—Because of their use in antifouling paints, primary sources of organotins are marinas, small boat harbors, vessel repair facilities, and berthing areas. Organotins have been used as agricultural fungicides to control plant diseases, as insecticides, and as biocides in pulp mills, breweries, textile mills, and leather-processing facilities. Organotins are also used to stabilize polyvinyl chloride.

The fate of organotins, like other toxic organic compounds, is determined by physical, chemical, and biological processes. Photolysis and biological degradation act to modify TBT in water. The adsorption of TBT to suspended particulate material and sediments results in TBT concentrations that are approximately 3 orders of magnitude higher than those found in water.

Among pathways available for degrading TBT in water, aerobic metabolism appears the most important. Many groups of plants and animals appear capable of readily degrading TBT. Rapid half-lives of 4-14 days have been reported in algae. TBT appears to be degraded by progressive debutylation to dibutyltin,

monobutyltin, and inorganic tin (Center for Lake Superior Environmental Studies 1988). The intermediate products appear to be less toxic than TBT.

The metabolism of TBT in aquatic life and humans appears only slightly slower than microbial metabolism. TBT is bioconcentrated by fish and invertebrates to approximately 3,000 times the ambient water concentration. The contribution of dietary accumulation to the total body burden is much less than that from bioconcentration. Biomagnification is unlikely because TBT is eliminated (i.e., depurated) at all trophic levels, including fish and humans. However, definitive tests are necessary to resolve many remaining questions of the environmental effects and fate of organotins.

NONIONIC ORGANIC COMPOUNDS — AROMATIC HYDROCARBONS

In this section, characteristics of polycyclic aromatic hydrocarbon (PAH) compounds and related aromatic hydrocarbon compounds are summarized. Characteristics of additional nonionic organic compounds (compounds that do not dissociate in water) are summarized in subsequent sections.

Polycyclic Aromatic Hydrocarbon Compounds

PAH compounds are a group of chemicals formed most commonly during incomplete combustion of organic materials (e.g., coal, oil and gas, garbage, and wood). HPAH compounds (compounds having ≥ 4 aromatic rings) are present in high concentration in these combustion products. LPAH compounds (3 or fewer aromatic rings) tend to be found in high concentration in uncombusted fossil fuels. PAH compounds are ubiquitous in the environment and natural sources of these compounds include forest fires and volcanoes, as well as oil seeps and erosion of coal beds. In Puget Sound, major sources of PAH compounds to Puget Sound include industrial and municipal discharges, mining and erosion of coal beds, and at least historically, forest fires (Barrick 1982; Barrick et al. 1984; Bates et al. 1984; Yake et al. 1984; Barrick and Prahl 1987).

There are no uses for most individual PAH compounds except as research chemicals. However, PAH compounds occur in several important mixtures, including creosote, which is comprised of greater than 85 percent PAH compounds. Because they have many of the same sources, individual PAH compounds are not distinguished in this discussion. Information for individual PAH compounds is included in Appendix A, primarily because of differing degrees of toxicity and persistence. LPAH compounds include acenaphthene, acenaphthylene, anthracene, fluorene, and phenanthrene. HPAH compounds include

fluoranthene, pyrene, benz(a)anthracene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(g,h,i)pyrene, benzo(a)pyrene, chrysene, dibenz(a,h)anthracene, and indeno(1,2,3-cd)pyrene.

Exposure Routes and Risks—Because PAH compounds are found throughout the environment, humans may be exposed to PAH compounds by inhalation, ingestion, and dermal contact. Inhalation is by far the most common exposure route.

Although PAH compounds may have similar toxicological effects and environmental fates, the health effects of individual PAH compounds are not identical. Reliable health-based studies exist only for a few compounds; potential health effects of the remaining compounds must be inferred from available information. PAH compounds enter the body easily, and the rate of absorption is increased when the PAH compounds are present in oily mixtures. Animal studies indicate that most PAH compounds are metabolized within a few days. Several PAH compounds [i.e., benz(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, chrysene, dibenz(a,h)anthracene, and indeno-(1,2,3-cd)pyrene] have been identified as animal carcinogens.

Although there is no available information regarding cancer in humans following inhalation exposure to individual PAH compounds, epidemiologic studies show increased mortality from lung cancer in humans exposed to coke-oven emissions, roofing-tar emissions, and cigarette smoke. Because each of these sources contains various mixtures of individual PAH compounds as well as other potentially carcinogenic chemicals, the contribution of any individual PAH to the total carcinogenicity of these mixtures in humans has not been evaluated. Despite these limitations, the studies provide qualitative evidence of the potential for mixtures containing PAH compounds to cause cancer in humans. Adverse immunological, reproductive, and genetic effects have been shown in studies on mice. No information is available regarding similar effects on humans.

Sources and Fate—The primary source of PAH compounds in the air is the burning of wood and fuel for residential heating. Other common sources are vehicle exhaust, asphalt roads, coal tar and coal tar production, agricultural burning, hazardous waste sites, tobacco smoke, creosote-treated wood, coking plants, coal gasification, smokehouses, aluminum production, and municipal waste incinerators. PAH compounds are also found in most foods (e.g., cereals, grains, vegetables, fruits, and meats). Most of the PAH compounds in surface waters and soils are believed to result from atmospheric deposition. For any given body of water, the major source of PAH compounds could vary depending on the prox-

imity of other sources such as municipal wastewater treatment plants and wood treatment facilities. Most nonreference area sediments in Puget Sound have significantly elevated PAH levels compared with reference areas (Table IV). Sources of PAH compounds in the marine environment include wood treatment facilities, docks and pilings, coal incineration and handling facilities, pulp mills, organic chemical plants (e.g., manufacture of dyes, plastics, and pesticides), and urban runoff from automobile emissions. The composition of PAH emissions to the atmosphere varies with the combustion source [e.g., emissions from residential wood combustion contain more acenaphthylene than other PAH compounds, while auto emissions contain more benzo(g,h,i)perylene and pyrene].

In surface waters, PAH compounds may volatilize, photooxidize, biodegrade, adsorb to sediments, or bioaccumulate in aquatic organisms (with bioconcentration factors ranging from 100 to 2,000). In sediments, PAH compounds can biodegrade or bioaccumulate in aquatic organisms. In soils, PAH compounds can biodegrade or bioaccumulate in plants, and they may also enter groundwater and be transported within an aquifer.

PAH compounds attach to dust and other airborne particles and are capable of being transported long distances, hence their detection in relatively uncontaminated areas. PAH compounds photooxidize and react in the atmosphere with other pollutants. Atmospheric half-lives are generally less than 30 days.

The PAH compounds are classified in the pollutant tables according to low or high molecular weight. PAH compounds within each weight classification generally share similar environmental fates. Transport and partitioning characteristics are also roughly correlated to molecular weights of PAH compounds, in large part because the solubility of PAH compounds tends to decrease with increasing molecular weight. HPAH compounds have stronger adsorption tendencies and less significant volatilization, and some HPAH compounds are carcinogens. LPAH compounds show moderate adsorption tendencies, more significant volatilization, and greater microbial degradation. No LPAH are established carcinogens.

Methylnaphthalenes and Methylphenanthrenes

Methylnaphthalenes are alkylated forms of naphthalene and are generally found with other PAH compounds. Methylphenanthrenes are analogous forms of phenanthrenes. Both of these methylated groups of compounds are often detected in Puget Sound sediments (Tetra Tech 1985b; Barrick and Prahl 1987; CH2M Hill 1989) and originate primarily as fossil fuel products including oil and coal (Barrick 1982; Barrick et al. 1984). Because of their similarities in chemical

structure and spatial distribution, they have similar or identical sources, effects, and fates as those described in the section on PAH compounds.

NONIONIC ORGANIC COMPOUNDS — CHLORINATED AROMATIC HYDROCARBONS

In the next sections, characteristics are described for the most common chlorinated benzenes, polychlorinated dibenzofurans and dibenzodioxins, and PCBs, which are all different types of chlorinated aromatic hydrocarbons.

Dichlorobenzenes

Dichlorobenzenes are anthropogenically produced chemicals that are not known to occur naturally. They exist primarily in the vapor state and are ubiquitous in the environment. In general, they have low water solubilities and low flammabilities and are chemically unreactive.

Exposure Routes and Risks—The general population is exposed to dichlorobenzenes through the consumption of contaminated drinking water and food (particularly fish; although dichlorobenzenes bioaccumulate to only low ppb levels in fish) and through inhalation of contaminated air. Data suggest that inhalation may be a greater source of exposure than other routes with respect to occupational exposure, and is a much more significant source of exposure than the dermal route.

After absorption by the body, dichlorobenzenes are rapidly distributed to fat, liver, lung, heart, brain, and muscle tissue. These chemicals are eliminated from the body within 5-6 days. Epidemiological studies are insufficient to characterize human effects of exposure to dichlorobenzenes. Studies of acute and chronic toxicity of dichlorobenzene isomers indicate that the compounds have similar target organs and effects. There is no evidence that dichlorobenzenes are carcinogens.

Sources and Fate—Dichlorobenzenes are used in a number of organic chemical syntheses and in solvents, electrical equipment insulators, pesticides, herbicides, and fungicides. Chemical waste dump leachates and direct manufacturing effluents are the major sources of dichlorobenzenes.

Sorption, bioaccumulation, and volatilization are expected to be competing fate processes of dichlorobenzenes due to their lipophilic nature, relatively low vapor pressure, and low water solubility relative to polar organic compounds. However, dichlorobenzenes are relatively soluble compared with other nonpolar organic compounds, such as HPAH (Howard 1989). The rate at which each of these competing processes occur will determine the predominant fate, especially in water. Adsorption to sediments is likely due to the low water solubility. Dichlorobenzenes may be released directly to the atmosphere and soil in connection with their use as fumigants. Their detection in groundwater near hazardous waste disposal areas indicates that leaching occurs. Experimental bioconcentration factor values suggest that significant bioconcentration does not occur, although high bioconcentration factors were reported in a study of guppies. The data suggest that bioaccumulation occurs in both animals and humans and that dichlorobenzenes are biologically persistent. Volatilization from soil surfaces may be an important fate process. Dichlorobenzenes may be slowly biodegraded in soil under aerobic conditions. Chemical transformation by hydrolysis, oxidation, or direct photolysis is not an importar . fate process.

Hexachlorobenzene

Hexachlorobenzene (HCB) is a solid white crystalline compound, formed as a by-product during the manufacture of chemicals used as solvents, other chlorine-containing compounds, and pesticides. HCB is the most persistent of the chlorinated benzenes in the environment. It has a half-life ranging from 3 to 6 years in soil and approximately 30 days in water in one test (ATSDR 1990a; Tabak et al. 1981). HCB is not very water soluble and sorbs strongly to sediments. There are no current commercial uses of HCB in the United States. HCB was used as a pesticide until 1985, when the last registration of HCB as a pesticide was voluntarily canceled.

Exposure Routes and Risks—Proximity to industrial sources or hazardous waste sites provides primary exposure routes for HCB. HCB may be transported to the air by dust particles. Dermal contact is a major source of exposure. HCB exposure may also occur by ingestion of certain foods (e.g., dairy products, meat, and poultry). More HCB is absorbed if it is consumed in combination with fat or oil.

HCB is rapidly transported to tissues in the body after exposure and tends to accumulate in fat tissue. Most HCB leaves the body, but small amounts may remain for years, especially in the fat tissue. Specific effects of HCB exposure via inhalation and dermal contact are not yet known. However, skin disorders

have been reported in humans following digestion of HCB. There is also evidence that HCB is toxic to nursing mammals exposed through maternal milk. There is sufficient evidence that HCB is a liver and thyroid carcinogen in test animals and that HCB is considered a probable human carcinogen.

Sources and Fate—There are currently no documented producers of HCB in the United States, and importation of this compound has also ceased. However, HCB may still enter the environment as a by-product of the manufacture of chlorinated solvents, other chlorinated compounds, and several pesticides. In addition, an estimated 3,500-11,500 kg of HCB was inadvertently produced during the manufacture of chlorinated solvents in 1984 (Carpenter et al. 1986). HCB can also be produced during combustion processes such as the incineration of municipal wastes. Total releases from this source are estimated to range from 57 to 454 kg per year (Carpenter et al. 1986). HCB was also used in the production of pyrotechnic materials (e.g., smoke bombs), aluminum, graphite electrodes, and dyes and as a component in wood preservatives. Direct discharges from manufacturing facilities are the primary sources of HCB in water and sediments; effluents from sewage treatment plants present a significant, but lesser, contribution.

HCB is one of the most persistent pollutants. It bioaccumulates in the environment, in animals, and in humans. Low levels of HCB have been found in almost all humans tested, most likely as a result of ingestion of HCB in foods. Environmental HCB is generally found tightly bound to soil and sediments. However, because of the low water solubility of HCB, it is not usually found in water (HCB was found in less than 1 percent of the groundwater samples taken from more than 862 hazardous waste sites) (Carpenter et al. 1986). Because HCB is not generally found in the atmosphere, photodegradation may be the process that limits atmospheric levels of HCB. Biodegradation appears to be slow in oxygenated waters. Leaching of HCB from soils is not significant, although biodegradation removes HCB from soils over a period of years. Isensee et al. (1976) did not report any degradation in soils after 1 yr of incubation under either aerobic or anaerobic conditions; however, Fathepure et al. (1988) have more recently reported anaerobic microbial dechlorination of such highly chlorinated aromatic compounds.

Polychlorinated Dibenzofurans

Polychlorinated dibenzofurans (PCDFs) are members of a group of widespread and environmentally stable halogenated tricyclic aromatic hydrocarbons. PCDFs are not intentionally produced, but rather enter the environment as trace

impurities in PCBs and chlorinated phenols and as a result of combustion processes. PCDFs are structurally similar to PCBs and polychlorinated dibenzo-*p*-dioxins (PCDDs), with similar signs and symptoms of toxicity. Public concern over PCDFs is related largely to their prevalence in the environment and to their similarities to PCDDs, rather than to reported health effects.

Exposure Routes and Risks—PCDFs have been detected in fly ash and in flue gas from municipal and industrial incinerators. Residues of PCDFs have been found in human breast milk. The most toxic PCDF congeners are 2,3,7,8-tetrachlorodibenzofuran, 1,2,3,7,8-pentachlorodibenzofuran, and 2,3,4,7,8-pentachlorodibenzofuran. PCDFs are severely toxic to animals and humans in acute concentrations, resulting in retarded growth, birth defects, liver damage, and skin lesions. Data on long-term exposure to PCDFs are not available. PCDFs are not known carcinogens.

Sources and Fate—PCDFs enter the environment as contaminants in a number of chemical products, including herbicides, polychlorinated phenols, HCB, PCBs, and thermal insulation materials. PCDFs are also formed from the combustion of chlorinated material and can be found in incinerator fly ash. PCDFs may be found at wood treatment facilities, at sites of transformer fires, near incinerators, and at other facilities where these products are used or burned.

PCDFs are very persistent in sediments and have been found to bioaccumulate in seal, fish, turtle, and human fat tissue. PCDFs are generally quite soluble in organic solvents, but water solubility decreases with chlorination.

Polychlorinated Dibenzo-*p*-dioxins

PCDDs [including 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD)] belong to a class of compounds commonly referred to as chlorinated dioxins. According to the position and number of chlorine atoms, it is possible to form 75 different congeners of chlorinated dioxins. Dioxins are fairly stable in the presence of heat, acids, and alkalies. Of the dioxins, 2,3,7,8-TCDD is regarded as the most toxic, and it is widely distributed in the environment. PCDDs are not produced intentionally and have no known uses. They are produced as unwanted by-products of the manufacture of chlorophenols and their derivatives, including chlorophenoxy herbicides, germicides, and wood preservatives. The production and use of 2,4,5-trichlorophenoxyacetic acid and 2,4,5-trichlorophenol as defoliants has been a major source of PCDDs. Although this production was

discontinued in the United States in 1979, present sources of exposure exist from remaining stores, improper disposal methods, and hazardous waste sites.

Exposure Routes and Risks—Consumers may be exposed through ingestion of contaminated food (e.g., fish, cow's milk), inhalation of contaminated dust or air, or dermal contact with contaminated plants, wood, and paper products. Apart from certain industrial effluents and leachates from chemical dump sites, no 2,3,7,8-TCDD has ever been reported in drinking water. Therefore, it is expected that exposure through consumption of drinking water is negligible. In mammals, 2,3,7,8-TCDD is readily absorbed through the gastrointestinal tract. Although exposure from dermal contact and inhalation has also been reported, it has been predicted that ingestion of contaminated food accounts for 98 percent of the daily human uptake of PCDDs (Syracuse Research Corporation 1989). Occupational exposure occurs during the bleaching process in pulp and paper mills, at certain hazardous waste sites, at municipal and industrial incinerators, and at chlorophenol production facilities. Accidental releases may occur through transformer fires.

Some PCDDs (including 2,3,7,8-TCDD) are among the most toxic compounds known. PCDDs have been proven toxic to unborn animals, and they reduce fertility in some species. Although 2,3,7,8-TCDD is highly toxic to all species that have been tested, there are large differences in sensitivity among species. Health effects similar to those caused by exposure to 2,3,7,8-TCDD are expected for other PCDDs, although higher doses are required. The symptoms of toxicity in humans after exposure to 2,3,7,8-TCDD include altered liver function and fat metabolism, pathologic changes in the blood, and skin lesions. Some of these symptoms may be attributed to other chemicals of which PCDDs are minor contaminants. Based on animal studies, EPA (1990c) has determined that 2,3,7,8-TCDD is a probable human carcinogen.

Sources and Fate—The primary sources of PCDD contamination are associated with the industrial manufacture of chlorophenols and their derivatives and with the subsequent disposal of wastes from these industries. Municipal incineration facilities may also emit PCDDs. The data do not indicate the relative importance of these sources in contributing to environmental emissions. Current data indicate that the maximum levels of PCDDs are likely to be found in soil and drainage sediment samples near chlorophenol manufacturing facilities and chemical waste disposal sites. There are few studies of atmospheric PCDD levels. In the United States, the highest PCDD levels have been reported at hazardous waste sites and in fish and wildlife tissue from areas contaminated with 2,3,7,8-TCDD (Syracuse Research Corporation 1989).

PCDDs are persistent contaminants that are not likely to undergo chemical or biological transformation in air, water, or soil. The role of photochemical transformation in determining the fate of these chemicals in various ambient media is unknown, but PCDDs are susceptible to photochemical reactions in the presence of organic acids. In water, a substantial proportion of PCDDs may be present sorbed to sediments or in biota. Although very persistent, 2,3,7,8-TCDD may be removed from water through volatilization and photolysis. In air, PCDDs are expected to be present as a vapor and sorbed to particles. PCDDs in soils are most likely transported to the atmosphere by contaminated dust particles and direct volatilization from the surface and transported to surface water by erosion. PCDDs are resistant to photochemical degradation and biodegradation in soil. Several bioconcentration factors have been reported for 2,3,7,8-TCDD, but none can be considered definitive values. Experimental bioconcentration factors for aquatic organisms range from 2,000 to 39,000 (U.S. EPA 1985c; Syracuse Research Corporation 1989), with EPA's best estimate established at 5,000 (U.S. EPA 1985c).

Polychlorinated Biphenyls

PCBs are environmentally persistent compounds that have a strong tendency to accumulate in aquatic sediments and in tissues of aquatic organisms. There are 209 PCB congeners (Mullin et al. 1984). Various mixtures of these compounds were marketed in the past and can sometimes be identified in the environment. However, the compounds do not all degrade at the same rate, confounding attempts to identify original sources of contamination.

Exposure Routes and Risks—PCBs enter the body through ingestion of contaminated food, inhalation of contaminated air, and dermal contact. The most common route of exposure is through the consumption of fish and shellfish from PCB-contaminated water. Exposure from consumption of contaminated drinking water is minimal.

Although the effects of PCBs have been diminishing since manufacturing of the chemicals ceased in 1977, it is expected that there are continued health effects in connection with certain occupations. Studies performed on humans show that irritations such as acnelike lesions and rashes can occur in PCB-exposed workers. PCBs are also classified by U.S. EPA (1990c) as probable human carcinogens. The sublethal toxic effects of PCBs are well documented for phytoplankton, mammals, and birds (Casarett and Doull 1986).

Sources and Fates—PCBs have been used primarily as coolants and lubricants in transformers, capacitors, and other electrical equipment. Sources of PCBs include transformer leaks or spills, old hydraulic fluids, lubricants, pesticides, and surface coatings. However, manufacturing of PCBs was banned in the United States in 1977 because of human health hazards resulting from high PCB levels in fish and shellfish, environmental persistence, and bioaccumulation levels. Importation of PCBs was banned by the federal Toxic Substances Control Act in 1979. Although there are no new industrial (point) sources of PCBs, many of the transformers and capacitors that contain PCBs are still in service. These products present ongoing risks of exposure through illegal or inadequate disposal methods and accidental release (e.g., transformer explosions and fires). PCBs have been found in high concentrations in small areas of urban bays in Puget Sound (Malins et al. 1982; Tetra Tech 1985b; Chan et al. 1985a,b; Stinson et al. 1987; Beller et al. 1988). However, PCBs are widely distributed and occur in most sediment and tissue samples taken from the sound.

PCBs are relatively insoluble in water compared with many other organic compounds and are highly soluble in solvents. PCBs also resist hydrolysis and oxidation. These factors make them highly persistent in the environment and susceptible to bioconcentration. However, contaminants similar to PCDFs may account for some of the harmful effects attributed to PCBs. There is ample evidence that PCBs bioaccumulate in the food chain; the degree of bioaccumulation may depend on fat content, the octanol-water partitioning coefficients, and the structure of PCBs, rather than on the quantity of PCBs ingested. Additional factors are the size and age of the aquatic organism and water temperature. The degree of chlorination affects metabolism, bioconcentration, degradation, adsorption, and volatilization of PCBs. Generally, metabolization, degradation, and volatilization decrease with chlorination, while bioconcentration and adsorption increase. Sorption to sediments is an important fate process for PCBs that depends on salinity, sediment organic content, and sediment size. If anaerobic degradation of PCBs in sediments occurs, it is a very slow process.

NONIONIC ORGANIC COMPOUNDS — MISCELLANEOUS EXTRACTABLE COMPOUNDS

In the next sections, the characteristics are described for several nonionic organic compounds that do not fit within the classes of compounds discussed in previous sections. These compounds include N-nitrosodiphenylamine, MEK, bis(2-ethylhexyl)phthalate, hexachlorobutadiene, and dibenzofuran.

N-Nitrosodiphenylamine

N-Nitrosodiphenylamine is an EPA-designated priority pollutant from the class of chemicals known as nitrosamines. It may occur naturally during the metabolism of nitrates and amines. There is little published information on this chemical, and relatively little is known of its aquatic fate and environmental and human health effects.

Exposure Routes and Risks—Humans are primarily exposed to N-nitrosodiphenylamine through dermal contact with rubber and vinyl products. Ingestion is a secondary source. Although there is sufficient evidence of carcinogenicity in animals, N-nitrosodiphenylamine has only a suspected human carcinogen.

Sources and Fate—N-Nitrosodiphenylamine is used in the vulcanization of rubber, as a chemical intermediate in the production of dyes and pharmaceuticals, and as an antioxidant. N-Nitrosodiphenylamine is more stable than most nitrosamines, but it is sensitive to light and undergoes photolytic degradation. When heated to decomposition, it emits toxic fumes of nitrogen oxides. Photolytic degradation and sorption to sediment appear to be the most important fate processes. It is also possible that intestinal microflora of vertebrates can both synthesize and degrade N-nitrosodiphenylamine in the environment (Callahan et al. 1979).

Methylethyl Ketone

MEK, also known as 2-butanone, is used as a solvent in lacquers, adhesives, rubber cements, inks, paint removers, and cleaning solutions. MEK is also present in vehicle exhaust.

Exposure Routes and Risks—Primary human exposure routes include inhalation of contaminated workroom air, vehicle exhaust, and other forms of air pollution. There are limited data available indicating that MEK is a natural component of some foods, so ingestion is also a source of exposure.

Sources and Fate—MEK is discharged into water as wastewater effluent and enters the atmosphere from industrial emissions. It is strongly associated with photochemical smog events, although it is usually absent from ambient air. It is formed by the natural photooxidation of hydrocarbons emitted from automo-

biles and other industrial sources. MEK may be a natural component of certain foods. MEK is removed from water generally by evaporation or slow biodegradation. It will not hydrolyze under normal conditions. Although it will not indirectly photooxidize in surface waters, it may be subject to direct photolysis. It does not significantly adsorb to sediments or bioconcentrate in aquatic organisms. MEK will partially evaporate from near-surface soils and leach into groundwater; it is subject to slow biodegradation in both soil and groundwater. MEK exists primarily in the gas phase in the atmosphere and will photodegrade at a moderate rate or will be removed by rain.

Bis(2-ethyl)hexylphthalate

Bis(2-ethyl)hexylphthalate is a colorless liquid that is insoluble in water, miscible with mineral oil, and soluble in most organic solvents. It is used in large quantities as a plasticizer for polyvinyl chloride and other polymers. Because of its use as a plasticizer, it is a common laboratory contaminant. Bis(2-ethyl)hexylphthalate is also used as a replacement for PCBs in dielectric fluids for electrical capacitors. It may also be naturally produced by plants and animals.

Exposure Routes and Risks—Exposure to bis(2-ethyl)hexylphthalate occurs from inhalation of contaminated air, consumption of contaminated food (especially fish, milk, and cheese) and water, and dermal contact with products containing bis(2-ethyl)hexylphthalate. Although occupational exposure during the production of plastics is the source of the greatest concentrations of bis(2-ethyl)hexylphthalate, the most widespread exposure results from its use in plastic products.

Although there are no data available to evaluate the carcinogenicity of bis(2-ethyl)hexylphthalate in humans, it has reportedly caused cancer in test animals (Casarett and Doull 1986) and has been classified as a probable human carcinogen based on these results (U.S. EPA 1990c). However, other authors (Technical Resources, Inc. 1989) have concluded that there is inconclusive evidence of carcinogenicity in animals. There have also been limited reports in the early 1970s of chronic reproductive effects in aquatic organisms exposed to low concentrations of bis(2-ethyl)hexylphthalate (Casarett and Doull 1986).

Sources and Fate—Bis(2-ethyl)hexylphthalate is widely distributed in the environment as a result of its use as a plasticizer. Disposal of plastic products in landfills, as well as incineration, releases bis(2-ethyl)hexylphthalate into the

environment. Other sources are the treated wastewater from coal mining operations, aluminum forming, foundries, and water-based ink and dye manufacturing facilities. In 1980, three firms were reported to be producing bis(2-ethyl)hexylphthalate in the United States (USITC 1981).

Bis(2-ethyl)hexylphthalate biodegrades in water and has been shown to bioconcentrate in aquatic organisms. Bis(2-ethyl)hexylphthalate strongly sorbs to sediment due to its low water solubility and is relatively persistent in the environment. Evaporation and hydrolysis are not important fate processes for aquatic bis(2-ethyl)hexylphthalate. Atmospheric bis(2-ethyl)hexylphthalate will be carried long distances and may be returned to soil by wet deposition. Bis(2-ethyl)hexylphthalate in soil neither evaporates nor leaches into groundwater. It may biodegrade under aerobic conditions. It is unknown whether photolysis or photooxidation are important atmospheric processes.

Hexachlorobutadiene

Hexachlorobutadiene (HCBD) is used in the chemical industry as a solvent for natural and synthetic rubber and other polymers, as well as in heat transfer liquid, transformer liquid, and hydraulic fluid. HCBD is a by-product of the manufacture of tetrachloroethene, trichloroethene, and tetrachloromethane.

Exposure Routes and Risks—The primary route and highest level of exposure to HCBD is associated with the inhalation of workplace air, although the general public can be exposed through the ingestion of contaminated drinking water. Although studies exist for freshwater organisms, no data are available concerning the chronic toxicity of HCBD or any other chlorinated butadienes to saltwater aquatic life. Acute toxicity may occur as low as 32 μg/L (EPA 1984c). There are also very few data available reporting the effects of HCBD on humans.

Sources and Fate—HCBD has been found in drinking water supplies, in aquatic organisms, and in aquatic sediments. It has been found in the effluent of chemical producers and in wastewater discharges. HCBD has been detected at elevated concentrations in sediments from the Hylebos Waterway in Commencement Bay (Tetra Tech 1985b), and was also found in elevated concentrations in some fish tissue samples from the same area. Historical discharges from chemical manufacturing plants along the waterway are probable sources of this contaminant.

HCBD is expected to evaporate rapidly from soils and may biodegrade under aerobic conditions. No leaching from soil is expected because of strong sorption tendencies, although leaching is more likely in sandy soils. HCBD will volatilize rapidly from water and has a half-life from months to years in the atmosphere. In water, sorption to sediments and suspended sediments is also likely due to the high log K_{ow} value of HCBD (3.74 to 4.28) (Callahan et al. 1979; Veith et al 1979).

Dibenzofuran

Little information has been published about the sources, effects and fate of dibenzofuran. Dibenzofuran has been documented at wood treatment facilities in Puget Sound (Barrick et al. 1986; CH2M Hill 1989) and has been detected at elevated concentrations in many contaminated sediments and fish tissues (Tetra Tech 1985b; Beller et al. 1988; Pastorok et al. 1988). It is present in the fly ash from municipal incinerators and is a component of insecticides. No information was found regarding the toxic effects of exposure to dibenzofuran on aquatic organisms. The environmental distribution of dibenzofuran in Puget Sound is correlated with that of LPAH compounds, indicating that dibenzofuran and aromatic hydrocarbons have similar sources in the sound (Tetra Tech 1985b; Barrick et al., 1988).

NONIONIC ORGANIC COMPOUNDS — PESTICIDES

In this section, the characteristics of three major groups of chlorinated pesticides are summarized: aldrin and dieldrin; DDT and its major breakdown products, DDD and DDE; and hexachlorocyclohexane (HCH), which is also known as lindane. These and numerous other pesticides of potential concern in Puget Sound are described in detail in PSEP (1988). Three of these pesticides of potential concern (diazinon, diuron, and endosulfan I) have recently been recommended for routine monitoring in Puget Sound (PSEP 1991) but were identified after tables for this report had been completed (see additional discussion in the *Selection of the Pollutants of Concern* section of Chapter 1).

Aldrin and Dieldrin

Aldrin and dieldrin are two closely-related chemicals that are used primarily as insecticides. Because of their chemical and toxicological similarities, they are considered together by regulatory bodies. Aldrin has been used as a soil insecticide to control root worms, beetles, and termites. Dieldrin has been used

for the treatment of wood, soil, and seeds; to control mosquitoes and tsetse flies; as a sheep dip; and for mothproofing. All uses of aldrin and dieldrin on food crops were banned in 1975.

Exposure Routes and Risks—In the past, the most common exposure route for humans was ingestion of plants grown in treated soil and animal products exposed to the chemicals. The risk of exposure from these routes has been reduced since agricultural uses of aldrin and dieldrin were banned. The most likely current source of exposure for humans is through inhalation of indoor air in wood structures treated with excessive amounts of aldrin or dieldrin. Some risk of exposure may remain through new applications of individually owned stockpiles of the chemicals or as a result of improper or illegal disposal methods.

Although aldrin and dieldrin are clearly toxic to humans, the severity of health effects depends on the concentration and length of exposure. Brief exposure to high levels of the chemicals may cause headaches, dizziness, nausea, convulsions, and death (at extremely high doses). Although human studies of the carcinogenicity of aldrin and dieldrin are largely inconclusive, EPA has classified these two chemicals as probable human carcinogens based on the results of animal tests (EPA 1990c).

Sources and Fate—Although the chemicals were banned in 1975 for general agricultural use, they were available after 1975 to control termites and for mothproofing. However, even these uses have been prohibited or voluntarily discontinued. There should be no new sources of the chemicals, although emissions may result from treated wood and from new applications of old stocks. Most of the aldrin and dieldrin levels recorded for Puget Sound air, water, and soil were reported prior to 1976 and may be overestimated (Tetra Tech 1985b).

Aldrin is readily converted to dieldrin in all media. Dieldrin is fairly persistent and is resistant to biodegradation. Dieldrin readily bioaccumulates and biomagnifies through the food chain. Rapid volatilization is the principal removal mechanism of aldrin from soil and water. Dieldrin sorbs tightly to soil and sediment and volatilizes more slowly. Although surface runoff of dieldrin is a major pathway of loss from treated soil, leaching from soil to groundwater appears minimal.

DDT, DDE, and DDD

DDT is primarily composed of three compounds that are white, crystalline, tasteless, and almost odorless solids. DDT does not occur naturally. It has been one of the most widely used insecticides. DDE and DDD are breakdown products of DDT and also occur as contaminants of technical grade DDT. DDT was banned from general agricultural use in the United States in 1972, but it is still produced and exported for use in other areas of the world.

Exposure Routes and Risks—DDT, DDE, and DDD enter the body primarily by ingestion, although exposure is possible through inhalation. Dermal exposure is not significant because the compounds are absorbed very poorly through the skin. Under certain conditions, DDT, DDE, and DDD may remain in soil for long periods of time and be transferred to crops grown in the soil; imported foods are a continuing source of exposure. In 1981, ingestion of DDT and DDE in food was estimated at 2.24 μg per day (Gartrell et al. 1986). Other primary sources of exposure may be through occupational involvement at hazardous waste sites.

DDT, DDD, and DDE are stored most readily in fat tissues and are eliminated very slowly. Breast-fed children are a group at special risk because DDT in maternal milk is found in higher concentrations than in cow's milk or other food. DDT has reportedly caused rashes and irritation of the eyes, nose, and throat. Acute exposure at high doses primarily affects the nervous system. In addition, synergistic and antagonistic effects of DDT have been shown in animal tests. DDT administered to animals along with known carcinogens resulted in both greater and lesser tumor production than when the carcinogens were tested without DDT. Although no studies exist that indicate a definite link between DDT and tumors in animals by the inhalation route, or with tumors in humans by either the inhalation or ingestion route, U.S. EPA (1990c) has classified the compounds as probable human carcinogens.

Sources and Fate—Although there are significantly fewer sources of DDT, DDE, and DDD in the environment, the products were used so extensively in the past that they are still found in virtually all air, water, and soil samples. Levels in most air and water samples are low, and exposure by these pathways is not of great concern. New releases of the compounds in the United States are expected to be negligible. In 1985, there were two producers of DDT for export in the United States (HSDB 1988). These facilities may have released small amounts of DDT via fugitive or noncontrolled emissions. Studies of peat lands located in the middle latitudes of the United States indicate that there are continuing sources

of the compounds; the cause has been attributed to atmospheric transport from areas where DDT is still in use. Other studies have shown that levels of DDT and its metabolites have been decreasing consistently in all media and in food samples.

Atmospheric DDT is subject to photodegradation and wet and dry deposition. DDT preferentially binds to soil and sediment, where it may be subject to photodegradation near the surface and biodegradation with depth. Under certain conditions, DDT may persist for long periods of time or may be converted to DDE, which persists even longer. DDT, DDE, and DDD are only slightly soluble in water; therefore, loss of these compounds in runoff is primarily due to transport of the particles to which they are bound. Volatilization of DDT and DDE accounts for substantial losses from soil and water. It is estimated that DDT evaporates from water within 50 hours. Laboratory studies of the air/water partition coefficient of DDE indicate that it volatilizes from marine water 10 to 20 times faster than from fresh water (Atlas et al. 1982). DDT, DDE, and DDD are highly fat-soluble compounds with long half-lives, resulting in bioaccumulation and biomagnification that increase with advances up the food chain.

A survey of nine localities conducted during times of high usage of DDT showed DDT detected in all localities at levels ranging from 1 ng/m^3 of air to 2,520 ng/m^3 (Stanley et al. 1971). Present levels are expected to be significantly lower. DDT and DDE have been reported in surface waters at levels of 0.001 μg/L, while DDD generally has not been found. National soil monitoring programs conducted in the early 1970s have reported average levels in soil ranging from 0.18 to 0.37 ppm (Crockett et al. 1974).

γ-Hexachlorocyclohexane

HCH isomers were developed in 1942 as simple and effective pesticides. Of its isomers, γ-HCH is the most effective and is marketed as lindane. γ-HCH is used primarily as a pesticidal treatment for wood, as a seed treatment, and as a fumigant. It has not been produced commercially in the United States since 1983; however, it is still imported and distributed domestically. In Puget Sound, γ-HCH is used primarily in urban areas (PSEP 1988). Its use as an insecticide is declining because of its environmental persistence and tendency to bioaccumulate.

Exposure Routes and Risks—The primary routes of human exposure are ingestion, inhalation, and dermal contact. Occupational exposure was a significant source prior to 1977, when EPA began regulating γ-HCH. The primary risk

of exposure to the general population is through the consumption of foods contaminated with pesticide residues.

There is limited evidence of the carcinogenicity of γ-HCH in test animals, although other isomers of HCH have been linked with cancer in test animals. Evidence regarding carcinogenicity of γ-HCH in humans is inconclusive. Acute toxicity has been associated with γ-HCH concentrations in the range of 5-28 μg/L for marine shrimp and killifish (PSEP 1988).

Sources and Fate—γ-HCH is present in agricultural runoff from areas where pesticides were used that contained γ-HCH. Other sources are the effluents of industries using γ-HCH, such as wood and seed treatment plants. γ-HCH has been found infrequently in surface sediments in Everett Harbor (Pastorok et al. 1988). Of the pesticides and herbicides found in Puget Sound, γ-HCH is of secondary concern based on its restricted use and relatively low mobility (PSEP 1988). The use of γ-HCH in the Puget Sound basin has been estimated to range from 0 to 1,300 pounds/year. Total use in the Puget Sound basin is estimated to be 2,640 pounds/year.

γ-HCH is environmentally persistent. The fate of γ-HCH in aquatic systems is determined by its bioavailability to transformation processes. Although sorption to suspended sediment and biota is not extensive, sorption is probably an important process for transporting γ-HCH to anaerobic sediments where transformations occur. Hydrolysis, oxidation, and photolysis are not important processes in aquatic environments. However, bioaccumulation occurs in aquatic organisms.

VOLATILE NONIONIC ORGANIC COMPOUNDS

In this section, the characteristics of the following volatile organic compounds are described: benzene, chloroform, ethylbenzene, toluene, tri- and tetrachloroethene, and total xylenes.

Benzene

Benzene is a natural product of volcanoes, forest fires, and crude oil seeps and is present in many plants and animals. Benzene is also a major industrial chemical manufactured from coal and tar. Benzene is used to make other chemicals (primarily ethylbenzene, cumene, and cyclohexane), as well as some types of plastics, detergents, and pesticides. It is a component of gasoline, glues

and adhesives, household cleaning products, paint strippers, some art products, and tobacco smoke. As a pure chemical, benzene is a clear and colorless liquid. Benzene has been found at 337 of the 1,177 NPL hazardous waste sites (ATSDR 1989c).

Exposure Routes and Risks—Occupational exposure accounts for the highest levels of benzene found in humans, particularly in rubber manufacturing facilities, oil refineries, and chemical plants and at gasoline retail stations. Benzene evaporates quickly, making inhalation by far the most likely exposure route for humans. The most widespread exposure to the general public is from inhalation of tobacco smoke and vehicle exhaust. Small amounts of benzene may also be found in some foods and in contaminated drinking water. Dermal exposure is possible through contact with benzene-containing products (e.g., gasoline).

Benzene is harmful to human health, although the degree of harm depends to a great extent on the length and amount of exposure. Short-term exposure of humans and animals to high levels of benzene generally causes drowsiness, dizziness, and headaches; death from such exposure has also been reported. Long-term exposure to various levels of benzene has caused leukemia and subsequent death of workers exposed for periods from 5 to 30 years. Long-term exposure to benzene may affect normal blood production, possibly resulting in severe anemia and internal bleeding. Overwhelming evidence exists that benzene is a human carcinogen. Human and animal studies also indicate that benzene is harmful to the immune system and has been linked with genetic changes. Reproductive effects have been reported in animal studies, although human studies have been too limited to form a clear link with benzene. There is general agreement among investigators that benzene metabolites, rather than benzene itself, are the primary toxic agents.

Sources and Fate—Environmental sources of benzene may be both anthropogenic and natural. The most significant source results from the combustion of gasoline. Other minor sources include septic tank effluent, structural fires, off-gassing from particle board, cigarette smoke, and possible natural food sources. Annual benzene emissions from anthropogenic sources are approximately 236,000 metric tons (ATSDR 1989c). However, environmental levels are low due to efficient environmental removal processes.

Chemical degradation reactions, primarily the reaction with the hydroxyl radical, limit the atmospheric persistence of benzene to only a few days (and possibly only a few hours if the concentration of hydroxyl radicals is sufficiently

high). Biodegradation, principally aerobic, is the most important environmental fate mechanism for benzene associated with water, soil, and sediment.

Chloroform

Chloroform is a colorless, clear, dense, volatile liquid that is produced by both direct and indirect processes. It is ubiquitous in the environment. Chloroform is widely used as a process ingredient in the manufacture of fluorocarbon refrigerants and propellants, fire extinguishers, electronic circuitry, and plastics. Chloroform is also used in anesthetics and pharmaceuticals, fumigants and insecticides, solvents, and sweeteners and as a chemical intermediate.

Exposure Routes and Risks—The major route for human exposure to chloroform is from ingestion of contaminated drinking water and inhalation of contaminated air, especially in industrial areas. A less important route of exposure is ingestion of contaminated food. Chloroform is absorbed almost completely through the gastrointestinal tract and tends to accumulate in fat and liver tissues. Regardless of the mode of entry into the body, chloroform is metabolized and excreted unchanged through the lungs.

Neurological, hepatic, renal, and cardiac effects have been associated with exposure to chloroform and have been documented in both humans and animals. Animal studies suggest that chloroform is carcinogenic and may be teratogenic. Human data regarding the effects of acute and chronic oral exposure to chloroform are insufficient, and animal tests did not clearly establish a no-effects level of exposure for toxicity.

Sources and Fate—Chloroform is likely to enter the environment in industrial effluents (e.g., pulp mill discharges), as well as from its indirect production in the chlorination of drinking water, municipal sewage, and cooling water.

The primary fate of chloroform is volatilization to the atmosphere. Chloroform may be transported long distances in the atmosphere and will react in the gas phase with photochemically produced hydroxyl radicals. Chloroform releases to land that do not evaporate will leach into groundwater, where they may reside for long periods of time; near-surface releases are expected to evaporate rapidly. Sorption to soil or sediment is not considered a significant fate process. Chloroform may be subject to significant biodegradation based on laboratory

tests, although the data are conflicting. Chloroform shows little or no tendency to bioconcentrate in tests of freshwater fish.

Ethylbenzene

Ethylbenzene is a colorless liquid that smells like gasoline, evaporates at room temperature, and burns easily. Ethylbenzene occurs naturally in coal tar and petroleum and is also found in many manufactured products including paints, inks, and insecticides.

Exposure Routes and Risks—Ethylbenzene enters the body rapidly through the lungs and digestive tract. Entry through the skin after contact with liquids containing ethylbenzene is also an exposure route. Because of its volatile nature, ethylbenzene is most likely to be inhaled. Ethylbenzene is broken down into other chemicals once it enters the body. Levels of ethylbenzene are likely to be higher in all media (especially groundwater) near hazardous waste sites and petroleum refineries. Consumers may be exposed to ethylbenzene through a wide range of products including pesticides, solvents, carpet glues, varnishes, paints, and tobacco products. The highest source of exposure to consumers is probably self-service gasoline stations. Indoor air has a higher average concentration of ethylbenzene (approximately 1 ppb) than outdoor air because of the use of household cleaners and paints (Howard 1989; ATSDR 1990b). Occupational exposure occurs in the petroleum industry and in chemical manufacturing, and workers using varnish, spray paints, and adhesives also may be subject to greater exposure.

Exposure to low levels of atmospheric ethylbenzene has caused eye and throat irritation in humans. Exposure to higher levels has caused decreased mobility and dizziness. No studies have reported death in humans, although animal studies suggest that ethylbenzene may be fatal. Short-term exposure to high concentrations of ethylbenzene may cause liver and kidney damage, nervous system changes, and blood changes in animals, although there are conflicting laboratory results. Long-term exposure data are not available. No conclusions have been established regarding carcinogenic properties of ethylbenzene.

Sources and Fate—Ethylbenzene is most commonly found as a vapor in the air because it volatilizes easily from water and soil. Atmospheric ethylbenzene can be removed by wet deposition or through photooxidation within approximately 2.5 days (Howard 1989). Photolytic transformations and biodegradation may also remove ethylbenzene from surface water. In soil, ethylbenzene

is degraded most easily by microorganisms. It does not readily sorb to soils or sediments and can move rapidly into groundwater. Ethylbenzene does not appear to bioaccumulate significantly in the food chain.

Toluene

Toluene is produced both naturally and anthropogenically. It is found primarily in petroleum products and in solvents and thinners for paints and lacquers. Toluene is a by-product of the production of styrene.

Exposure Routes and Risks—The primary route of exposure to toluene for humans is from inhalation of contaminated air. Exposure levels are increased with proximity to gasoline stations and vehicle exhaust, or in occupational atmospheres where toluene-based solvents are used. Exposure to toluene also occurs as the result of intentional substance abuse. Workers in the chemical, petroleum, and paint and dye industries are at greatest risk of exposure. Neurological damage is the primary adverse effect of toluene on both humans and animals. The cancer-causing potential of toluene in rats and mice is still being studied.

Sources and Fate—Toluene is released into the atmosphere principally from the volatilization of petroleum fuels and toluene-based solvents and thinners and from motor vehicle exhaust. Large amounts of toluene are discharged into waterways or spilled onto land during the storage, transport, and disposal of fuels and oils. Natural sources of toluene include volcanoes, forest fires, and crude oils.

If toluene is released to soil, it volatilizes to the air from near-surface soil and leaches to groundwater. Biodegradation occurs both in soil and groundwater, but is likely to be slow (especially at high concentrations that may be toxic to microorganisms). The presence of acclimated microbial populations may speed biodegradation. Toluene does not significantly hydrolyze in soil or water. In water, toluene concentrations will decrease due to evaporation and biodegradation. Aquatic removal can be rapid or may take several weeks depending on temperature, mixing conditions, and presence of acclimated microorganisms. Toluene does not significantly adsorb to sediments or bioconcentrate in aquatic organisms.

Total Xylenes

Total xylenes refers to the three isomers of xylene (meta-, ortho-, and para-xylene). This term is also sometimes applied to a mixture of xylenes and smaller amounts of other chemicals (primarily ethylbenzene). Xylenes occur in petroleum and coal and are formed during forest fires. Xylenes are colorless liquids with a sweet odor that leach into soil, surface water, and groundwater, generally as a result of petroleum use, transport, and storage. Xylenes are often a component of solvents and paint thinners and are used as a cleaning agent. Xylenes are also used in the manufacture of certain polymers and are ingredients in jet fuel, gasoline, and fabric- and paper-coating materials.

Exposure Routes and Risks—Occupational inhalation of xylenes is the most common exposure route for humans, although ingestion of contaminated food and water and dermal contact may be additional exposure routes.

Short-term exposure by humans to xylenes may cause skin, eye, nose, and throat irritation; impaired memory and reaction time; and stomach, liver, and kidney damage. Exposure to high doses of xylenes within short periods of time may cause nervous system damage and death. Data are insufficient to prove human carcinogenicity.

Sources and Fate—Primary sources of xylenes are fugitive industrial emissions and automobile exhaust. Additional sources of exposure are inhalation of tobacco smoke, gasoline, paint, varnish, shellac, and rust preventives. Other potential sources include hazardous waste sites and accidental releases of materials containing xylene (e.g., solvents).

Xylenes may persist in groundwater for several years. Sorption to soils and sediments occurs, although there is considerable variation and uncertainty in estimates of persistence in these media. Xylenes may persist in other aquatic systems for greater than 6 months before degrading. Limited biodegradation is the only significant source of xylene degradation in subsurface soils and most aquatic systems. Because xylenes evaporate easily, the highest levels are found in the atmosphere. After several days, xylenes in the atmosphere are degraded by photooxidation. Modest bioaccumulation levels have been shown in fish (e.g., bioconcentration factors less than 2.2), but food chain biomagnification has not been observed (Howard 1989).

IONIZABLE ORGANIC COMPOUNDS

In the following sections, characteristics are summarized for organic compounds that can dissociate in water. These compounds include various phenols, resin acids, and guaiacols.

Phenol

Pure phenol is a colorless or white solid. The commercial product is a liquid. Phenol has a distinct odor that is sweet and acrid. It evaporates more slowly than water, is moderately soluble, and is flammable. Phenol is primarily a manufactured chemical, though it also occurs naturally from decomposition of organic matter. Phenol has been obtained by distillation from petroleum, through synthesis by oxidation of cumene or toluene, and by vapor-phase hydrolysis of chlorobenzene. The largest single use of phenol is as an intermediate in the production of resins, and it is also used as an intermediate in the production of certain synthetic fibers, as an algicide, as a disinfectant, and in medicinal preparations.

Exposure Routes and Risks—Exposure to phenol for humans is possible through inhalation, ingestion, and dermal contact. Because phenol is used in many manufacturing processes and in consumer products, exposure can occur both at work and in the home. Phenol is present in many medicinal products (e.g., ointments, ear and nose drops, cold sore medication, mouthwashes, gargles, analgesic rubs, and throat lozenges) and is also found in drinking water, air, certain foods, and in wood and tobacco smoke.

The magnitude, frequency, likelihood, or relative contribution of each exposure route and source to total phenol exposure cannot be estimated using current data. In general, more phenol will enter the body if large areas of skin come in contact with dilute solutions of phenol than if small areas of the skin come in contact with concentrated solutions. After exposure to airborne phenol, as much as 50 percent of the phenol that enters the body will enter through the skin. Most of the phenol entering the body is excreted within 24 hours.

The severity of effects from exposure to phenol increases as both the level and duration of exposure increases. Ingestion of very high concentrations of phenol has resulted in death. The effects on humans of inhalation of phenol are unknown. Tests on animals of inhalation of phenol show lung irritation, muscle tremors, and loss of coordination. Exposure to higher concentrations of phenol for a period of weeks has caused severe damage to the heart, kidneys, liver, and

lungs, followed by death in some cases. Reproductive effects of phenol are unknown in humans, although effects have been shown in animal tests. Although phenol applied to the skin of mice has caused cancer, it is not known whether phenol is a human carcinogen. Phenol acted synergistically in tests with known carcinogens, causing more cancer than when the carcinogens were applied without phenol. Phenol also has beneficial effects; it is an antiseptic and an anesthetic and is used to remove skin blemishes. Studies of effects of phenol exposure on algae and fathead minnows reported reduced reproduction and growth, while effects on other aquatic organisms included increased enzyme activity in liver and muscle and decreased activity in the brain.

Sources and Fate—Phenol is released primarily to air and water during its manufacture and use. Phenol-formaldehyde resins have major uses in the construction, automotive, and appliance industries and can be found in associated effluents. Phenol is found in coal tar and is released in wastewater from the manufacture of plastics, adhesives, iron and steel, aluminum, leather, and rubber, and in wood treatment plants and paper pulp mills. Phenol is released during the burning of wood and in vehicle exhaust. Natural sources of phenol in water are animal wastes and decomposition of organic wastes.

Single, small releases of phenol do not remain in the air; the half-life is generally less than 1 day. Removal from soil by biodegradation is accomplished within 2–5 days (Howard 1989). Phenol can remain in water for more than 9 days. However, single large discharges or continuous small discharges of phenol may remain in each of these media for much longer periods of time. Phenol has not been found in soil except at hazardous waste sites, probably because it is efficiently removed from soil at low concentrations. Phenol in soil biodegrades and leaches to groundwater. Volatilization from soil and water is a slow process and is not expected to be a source of atmospheric phenol. Sorption to sediments is not an important transport mechanism of phenol in water. However, phenol has been detected occasionally in sediments from Puget Sound, including near pulp mills and in some sediments collected in nonurban areas (Tetra Tech 1985b), possibly as the result of aquaculture applications.

4-Methylphenol (4-Cresol)

4-Methylphenol is a constituent of pulp mill waste, is found at wood treatment facilities, and is used in the manufacture of organic chemicals.

Exposure Routes and Risks—Human exposure is primarily through inhalation and dermal contact in occupational environments and source areas. 4-Methylphenol is moderately toxic to benthic organisms.

Sources and Fate—4-Methylphenol is released to the atmosphere in vehicle exhaust, during coal tar and petroleum refining and wood pulping, and during its use in manufacturing and metal refining. 4-Methylphenol is contained in effluents from industries engaged in wood products manufacturing, leather tanning, iron and steel manufacturing, plastics production, textile production, rubber processing, and electronics manufacturing, and in effluents from municipal wastewater treatment facilities. Elevated concentrations of 4-methylphenol have been documented in surface sediments near pulp mills in Puget Sound (Tetra Tech 1985b, Norton et al. 1987; Pastorok et al. 1988).

In the air, 4-methylphenol reacts with photochemically produced hydroxyl radicals during the day and with nitrate radicals at night. It can return to soil through wet deposition, and it can be oxidized by metal cations in rainwater. Biodegradation is expected to be the dominant fate process. Complete removal from soils is possible within 6 days, although the process is slower under anaerobic conditions (Howard 1989). Disproportionate losses of 4-methylphenol (and phenol) relative to dimethylphenols have been observed in groundwater at a wood treating facility, presumably from biodegradation (Goerlitz et al. 1985).

Volatilization, bioconcentration in fish, and adsorption to sediment are not important fate processes for 4-methylphenol. Photolysis is expected to be significant only in surface waters of oligotrophic lakes. 4-Methylphenol is not expected to persist in sediments (Howard 1989). However, persistent high concentrations of 4-methylphenol have been observed in some Puget Sound sediments near industrial sources. These results can be explained either by large mass loadings of 4-methylphenol from the source or *in situ* production of 4-methylphenol in the sediments. One untested possibility is that 4-methylphenol could be produced by bacterial conversion of proteins (via *p*-hydroxyphenylacetate) accumulating in the sediments (Balba and Evans 1980).

Pentachlorophenol

Pentachlorophenol (PCP) is one of the most heavily used pesticides in the United States. It is used as an industrial wood preservative, as well as in consumer wood preserving formulations and in herbicides and pesticides. PCP does not occur naturally and exists in two forms of differing water solubilities. In its pure form, PCP exists as colorless crystals. The impure form of PCP,

which exists as a dark grey to brown dust in beads or flakes, is the type found at hazardous waste sites.

Exposure Routes and Risks—Humans can be exposed to low levels of PCP through inhalation of indoor and outdoor air and through ingestion of contaminated food and drinking water. Dermal contact with treated wood can also result in exposure to PCP. Occupation and proximity to hazardous waste sites present the greatest risk of exposure to PCP. Occupational levels of PCP inhalation in indoor air at wood treatment plants and lumber mills are much higher than for the general public; exposure levels are estimated at 0.9-14 mg per day compared with approximately 0.006 mg per day for the general public. An additional estimated 0.5 mg per day of PCP may be absorbed through the skin of workers who handle treated wood (ATSDR 1989e).

Exposure to high levels of PCP can cause adverse effects on the liver, kidneys, skin, blood, lungs, nervous system, and gastrointestinal tract. High levels of exposure can also be fatal. Longer-term exposure to lower levels of PCP can cause damage to the liver, blood, and nervous system. Animal studies indicate that short-term, high-level exposure to PCP can cause similar damage, with the severity of effects increasing as the duration of exposure increases. Acute toxicity and increased risk of reproductive failure have been shown in animals exposed to PCP, but there is insufficient evidence of human carcinogenicity. Studies of aquatic organisms indicate more severe effects on fish than on other aquatic biota; acute and chronic toxicity to saltwater organisms occur at concentrations as low as 53 and 34 μg/L, respectively (U.S. EPA 1986a). In addition, pentachloroanisole, a degradation product of PCP, has an even greater potential to bioaccumulate than PCP itself (Callahan et al. 1979).

Sources and Fate—PCP is released directly to air via volatilization from treated wood and evaporation of PCP-treated industrial process waters from cooling towers. Eighty percent of domestic consumption of PCP is attributed to the treatment of utility poles (ATSDR 1989d). Although generally insignificant, past emissions from production facilities may be more important in Puget Sound because one of the two major historical producers of PCP in the United States was located in the Commencement Bay area. In addition, a number of other chemicals (e.g., HCB, pentachlorobenzene, pentachloronitrobenzene, and benzenehexachloride isomers) are known to metabolize to PCP.

PCP releases to water occur by direct discharge from source and nonpoint sources, by wet deposition from the atmosphere, and by runoff and leaching from soil. Approximately 90 percent of wood treatment plants evaporate their

wastewaters and consequently they have no direct discharges to water. The remaining 10 percent of these plants discharge to municipal wastewater treatment facilities. PCP has been detected in at 10.4 percent of 2,738 hazardous wastes sites sampled for the substance (ATSDR 1989d).

PCP is also registered for use as an insecticide, a fungicide, an herbicide, a molluscicide, an algicide, a disinfectant, and as an ingredient in antifouling paint. However, nonwood uses account for no more than 2 percent of current PCP use (ATSDR 1989d).

PCP is not affected by hydrolysis and oxidation, but is rapidly photolyzed and can be biotransformed by microorganisms and metabolized by animals and plants. Sorption to soils and sediments occurs and is more important under acidic conditions than under neutral or basic conditions. The compound bioaccumulates to modest levels in algae, aquatic invertebrates and fish (ATSDR 1989d), but food chain biomagnification has not been observed.

Mono- and Dichlorodehydroabietic Acids

Chlorine bleaching processes used in both the sulfite and kraft pulp industries have been demonstrated to result in the formation of chlorinated resin acids such as chlorinated dehydroabietic acids. Chlorinated derivatives of dehydroabietic acid are by far the predominant chlorinated resin acids reported in bleached pulp effluents, presumably because its stability relative to other resin acids enables it to survive the strong oxidizing conditions of chlorine bleaching. Dehydroabietic acid, typically the predominant resin acid found in the environment, is derived from abietic acid and is relatively stable as a result of its aromatic ring structure. Chlorinated resin acids, because of their unique origin, are powerful geochemical tracers of pulp mills that use chlorine bleaching processes. Chlorinated resin acids have been reported in elevated concentrations in sediments near pulp mill discharges in Puget Sound as well as in other parts of the United States and Europe (Pastorok et al. 1988). Dehydroabietic acids are persistent in the environment and toxic to marine organisms. The toxicity of dehydroabietic acid decreases markedly at pH greater than 7 because of formation of the sodium salt.

Exposure Routes and Risks—There are insufficient data available to describe routes of exposure and associated risks of individual resin acids to biota.

Sources and Environmental Fate—Dehydroabietic acid is persistent in sediments, with a half-life of greater than 20 years estimated in sediments of Lake Superior (PSEP 1988).

2-Methoxyphenol

2-Methoxyphenol (guaiacol) is a by-product of wood and coal tar decomposition and is a major constituent of creosote. Guaiacol is released as a pollutant primarily in pulp mill wastes; it is one of the major breakdown products of lignin, which is the primary component of wood. Guaiacol has been detected infrequently in contaminated sediments in Everett Harbor, but has been found in high concentrations in sediments in other areas of Puget Sound, the United States, and Europe (Pastorok et al. 1988; Tetra Tech 1985b).

Chlorinated Guaiacols

Chlorinated guaiacols are well-documented by-products of pulp bleaching processes. The compounds are formed when lignin is treated with the chlorine bleaching process. Chlorinated guaiacols are excellent geochemical tracers of pulp mills because of their unique origin. Based on studies showing that unchlorinated guaiacols dehydroabietic acid, a related compound, is toxic and environmentally persistent, it is expected that chlorinated guaiacols will behave similarly.

Chlorinated guaiacols are moderately toxic to benthic organisms and fish. The most toxic isomer is 3,4,5,6-tetrachloroguaiacol, followed by 3,4,5-trichloroguaiacol and 4,5,6-trichloroguaiacol. Tri- and tetrachloroguaiacol exhibited considerable toxicity in fish taken from a river near a pulp mill in Switzerland (LC_{50} values were 0.75 and 0.32 mg/L for tri- and tetrachloroguaiacol, respectively (Leuenberger et al. 1985).

Trichloroethene

Trichloroethene (TCE) is used widely as a degreasor for metals in the metals and electronics industries. TCE is an intermediate in the manufacture of polyvinylchloride and is also found in the effluent of pulp mills, chloralkali plants, and dry cleaners.

Exposure Routes and Risks—The primary exposure route for humans is inhalation of contaminated air, followed by ingestion of contaminated drinking water. Although possibly carcinogenic, TCE is not considered highly toxic to humans or animals.

Sources and Fate—Although it has been detected in both marine and fresh waters, 80 to 95 percent of TCE evaporates to the atmosphere. TCE is most likely to enter the atmosphere from its widespread use as a metal degreaser. There are no known natural sources of TCE. It has been found in very high concentrations in a few nearshore sediment samples from Hylebos Waterway in Puget Sound (Tetra Tech 1985b). TCE is a volatile compound that moves quickly through groundwater, and it is a common contaminant of groundwater in industrial areas. It does not appear to bioconcentrate in fish tissue. In anaerobic environments, TCE degrades to dichloroethene and vinyl chloride. These breakdown products can be useful in determining the sources, fate, and transport of TCE to the environment.

Tetrachloroethene

Tetrachloroethene is a volatile compound used as a metal degreaser in the metals and electronics industries, in dry cleaning and textile processing, and in the production of fluorocarbons, pesticides, adhesives, paints, and coatings. There are no known natural sources of tetrachloroethene.

Exposure Routes and Risks—The primary source of human exposure is inhalation of occupational air contaminated by tetrachloroethene. Consumption of drinking water contaminated by rain or by pipes lined with vinyl is another source of tetrachloroethene. Tetrachloroethene is a carcinogenic compound that otherwise is not highly toxic to humans or aquatic organisms. It is moderately toxic to benthic organisms.

Sources and Fate—Tetrachloroethene is likely to enter the environment by fugitive air emissions from dry cleaning and metal degreasing industries and by accidental releases of products contaminated with tetrachloroethene. Wastewater effluents from metal finishing, laundering, aluminum forming, organic chemical and plastics manufacturing, and municipal treatment plants also contribute to environmental tetrachloroethene. Tetrachloroethene can also be found in the effluent of pulp mills and chloralkali plants and in the groundwater near many industrial areas. Tetrachloroethene has been found in highly elevated concentra-

tions in Puget Sound sediments near a chemical manufacturing facility and in somewhat elevated concentrations in fish tissue from the same vicinity (Tetra Tech 1985b). Tetrachloroethene breaks down in anaerobic environments to trichloroethene, dichloroethene, and vinyl chloride, and the presence of these breakdown products can be useful in determining the sources, fate, and transport of this chemical.

REFERENCES

Andreasson, J. 1990a. Edmonds wastewater treatment plant class II inspection; April 17-19, 1989. Washington Department of Ecology, Environmental Investigations and Laboratory Services Program, Olympia, WA.

Andreasson, J. 1990b. Tacoma central wastewater treatment plant class II inspection; June 26-28, 1989. Washington Department of Ecology, Environmental Investigations and Laboratory Services Program, Olympia, WA.

Atlas E., R. Foster, C. Glam. 1982. Air-sea exchange of high molecular weight organic pollutants: laboratory studies. Environ. Sci. Technol. 16:283-286.

ATSDR 1989a. Draft toxicity profile for mercury. U.S. Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, GA.

ATSDR 1989b. Draft toxicity profile for zinc. U.S. Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, GA.

ATSDR 1989c. Toxicity profile for benzene. ATSDR/TP-88/03. U.S. Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, GA.

ATSDR 1989d. Draft toxicity profile for pentachlorophenol. U.S. Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, GA.

ATSDR 1990a. Draft toxicity profile for hexachlorobenzene. U.S. Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, GA.

ATSDR 1990b. Draft toxicity profile for ethylbenzene. U.S. Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, GA.

Baird, C.E. 1 August 1985. Personal Communication (letter to Dr. Robert A. Pastorok, PTI Environmental Services, Bellevue, WA). City of Everett Public Works Department, Everett, WA.

Balba, M.T., and W.C. Evans. 1980. Methanogenic fermentation of the naturally occurring aromatic amino acids by a microbial consortium. Biochem. Soc. Trans. 8:625-627.

Barrick, R.C. 1982. Flux of aliphatic and polynuclear aromatic hydrocarbons to central Puget Sound from Seattle (Westpoint) primary sewage effluent. Environ. Sci. Technol. 16:682-692.

Barrick, R.C., E.T. Furlong, and R. Carpenter. 1984. Hydrocarbon and azaarene markers of coal transport to aquatic sediments. Environ. Sci. Technol. 18:846-854.

Barrick, R.C., H. Beller, and M. Meredith. 1986. Preliminary investigation, Eagle Harbor, Bainbridge Island, Washington. Prepared for Black & Veatch, Engineers-Architects and Washington Department of Ecology. Tetra Tech, Inc., Bellevue, WA.

Barrick, R.C., and F.G. Prahl. 1987. Hydrocarbon geochemistry of the Puget Sound region. III. Sources and fates of polynuclear aromatic hydrocarbons in sediments. Estuar. Coast. Shelf Sci. 25:175-191.

Barrick, R., S. Becker, R. Pastorok, et al. 1988. Sediment quality values refinement: 1988 update and evaluation of Puget Sound AET. Prepared by PTI Environmental Services, Bellevue, WA.

Bates, T., S. Hamilton, and J. Cline. 1984. Vertical transport and sedimentation of hydrocarbons in the central main basin of Puget Sound, Washington. Environ. Sci. Technol. 18:5.

Battelle. 1985a. Summary report of sediment quality criteria development workshop (Nov. 28-30, 1984). Prepared for the U.S. Environmental Protection Agency, Criteria and Standards Division. Battelle Marine Research Laboratory, Washington, DC.

Battelle. 1986. Detailed chemical and biological analyses of selected sediments from Puget Sound. Draft Final Report. Prepared for U.S. Environmental Protection Agency Region 10, Seattle, WA. Battelle Marine Research Laboratory, Washington, DC.

Battelle. 1986. Sediment quality criteria methodology validation: calculation of screening level concentrations from field data. Final Report. Prepared for U.S. Environmental Protection Agency, Criteria and Standards Division. Battelle Marine Research Laboratory, Washington, DC.

Beller, H., R. Pastorok, S. Becker, et al. 1988. Elliott Bay action program: analysis of toxic problem areas. Final Report. Prepared for U.S. Environmental Protection Agency Region 10, Office of Puget Sound. PTI Environmental Services, Bellevue, WA.

Bernhardt, J. 1982. Assessment of surface water quality in the vicinity of Reichold Chemicals, Inc., Tacoma, Washington (April 21, 1981). Washington Department of Ecology, Olympia, WA.

Bothner, M.J. 1973. Mercury: some aspects of its marine geochemistry in Puget Sound, Washington. Ph.D. Thesis University of Washington, Seattle, WA.

Callahan, M.A., M.W. Slimak, N.W. Gabel, et al. Water-related environmental fate of 129 priority pollutants. EPA 440/4-79-029a. U.S. Environmental Protection Agency, Office of Water Planning and Standards, Monitoring and Data Support Division, Washington, DC.

Carpenter, C., G. Schweer, G. Stinnett, et al. 1986. Exposure assessment for hexachlorobenzene. EPA 560/5-86-019. U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances (as cited in ATSDR 1990. Toxicity profile for hexachlorobenzene. U.S. Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, GA).

Casarett and Doull. 1986. Toxicology. C.D. Klaassen, M.O. Amdur, and J. Doull (eds). Macmillan Publishing Company, New York, NY.

Center for Lake Superior Environmental Studies. 1988. Ambient water quality criteria for tributyltin — 1988. Prepared for Battelle Memorial Research Institute and the U.S. Environmental Protection Agency, Office of Research and Development, Environmental Research Laboratories, Narragansett, RI. Center for Lake Superior Environmental Studies, University of Wisconsin-Superior, Superior, WI.

CH2M Hill. 1989. Final remedial investigation report for Eagle Harbor site, Kitsap County, Washington. Prepared for Environmental Protection Agency, Hazardous Site Control Division. CH2M Hill, Bellevue, WA.

Chan, S., M. Schiewe, and D. Brown. 1985a. Analysis of sediment samples for U.S. Army Corp of Engineers Seattle harbor navigation project, operations and maintenance sampling and testing of Duwamish River sediments. Draft Report. Prepared for U.S. Army Corp of Engineers, Seattle District. U.S. Marine Fisheries Service, National Oceanic and Atmospheric Administration, Seattle, WA.

Chan, S., M. Schiewe, and D. Brown. 1985b. Analysis of sediment samples for U.S. Army Corp of Engineers East, West, and Duwamish Waterway navigation improvement project, operations and maintenance sampling and testing of Duwamish River sediments. Unpublished. Prepared for U.S. Army Corp of Engineers, Seattle District. U.S. Marine Fisheries Service, National Oceanic and Atmospheric Administration, Seattle, WA.

Clark, R.C. 1983. The biogeochemistry of aromatic and saturated hydrocarbons in a rocky intertidal marine community in the Strait of Juan de Fuca. Ph.D. Thesis. University of Washington, Seattle, WA.

Cooley, R., R. Matasci, and M.S. Merrill. 1984. TPPS Technical Report A2: Collection system evaluation. Data Appendices. Metro Toxicant Program Report No. 48. Municipality of Metropolitan Seattle, Seattle, WA.

Crecelius, E.A., D.L. Woodruff, and M.S. Myers. 1989. 1988 Reconnaissance survey of environmental conditions in 13 Puget Sound locations. Submitted to U.S. Environmental Protection Agency Region 10, Seattle, WA. Battelle, Marine Sciences Laboratory, Sequim, WA, and National Oceanic and Atmospheric Administration, Seattle, WA.

Crecelius E.A., M.H. Bothner, and R. Carpenter. 1975. Geochemistries of arsenic, antimony, mercury, and related elements in sediments of Puget Sound. Environ. Sci. Technol. 9:325-333.

Crockett, A. G. Wiersma, H. Tai, et al. 1974. Pesticides in soils. Pesticide residue levels in soils and crops, FY-70 — national soils monitoring program (II). Pest Monit J. 8:69-97 (as cited in ATSDR 1989e. Draft toxicity profile for p,p'-DDT, p,p'-DDE, p,p'-DDD. U.S. Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, GA).

Ecology. 1984 (revised 1990). Dangerous waste regulations. Chapter 173-303 WAC. Washington Department of Ecology, Olympia, WA.

Ecology. 1990. Draft environmental impact statement for the Washington Sediment Management Standards Chapter 173-204 WAC. Washington Department of Ecology, Olympia, WA and PTI Environmental Services, Bellevue, WA.

Faigenblum, J. 1988. Chemicals and bacteriological organisms in recreational shellfish. U.S. Environmental Protection Agency, Region 10, Office of Puget Sound, Seattle, WA, and Washington Department of Social and Health Services, Office of Environmental Health Programs, Olympia, WA. 109 pp. + appendices.

Fathepure B.Z., J.M. Tiedje and S.A. Boyd. 1988. Reductive dechlorination of hexachlorobenzene to tri- and dichlorobenzenes in anaerobic sewage sludge. Appl. Environ. Microbiol. 54:327-330 (as cited in ATSDR 1990. Toxicity profile for hexachlorobenzene. U.S. Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, GA).

Gahler, A.R., R.L. Arp, J.M. Cummins, et al. 1982. Chemical contaminants in edible non-salmonid fish and crabs from Commencement Bay, Washington. U.S. Environmental Protection Agency, Environmental Services Division, Seattle, WA.

Galvin, D.V., and R.K. Moore. 1982. Toxicants in urban runoff. Metro Toxicant Program Report No. 2. Municipality of Metropolitan Seattle, Water Quality Division, Toxicant Control Planning Section, Seattle, WA.

Gartrell R., J. Craun, D. Podrebarac, et. al. 1986. Pesticides, selected elements, and other chemicals in adult total diet samples. October 1980 — March, 1982. J. Assoc,. Off. Anal. Chem. 69:146-161 (as cited in ATSDR

1989e. Draft toxicity profile for p,p'-DDT, p,p'-DDE, p,p'-DDD. U.S. Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, GA).

Ginn, T.G., and R.C. Barrick. 1988. Bioaccumulation of toxic substances in Puget Sound organisms. pp. 157-168. In: Oceanic Processes in Marine Pollution, Volume 5. D.A. Wolfe and T.P. O'Connor (eds.), Robert E. Krieger Pub. Co., Malabar, FL.

Goerlitz, D.F., D.E. Troutman, E.M. Godsy, et al. 1985. Migration of wood-preserving chemicals in contaminated groundwater in a sand aquifer at Pensacola, Florida. Environ. Sci. Technol,. 19:955-961.

Goldberg, E.D., M. Koide, V. Hodge, et al. 1983. U.S. mussel watch: 1977-1978 results on trace metals and radionuclides. Est. Coast. S. 16:69-93.

Hallinan, P. 1988. Kingston Wastewater Treatment Plant class II inspection. Washington Department of Ecology, Water Quality Investigations Section, Olympia, WA.

Hallinan, P. 1989. Daishowa Pulp and Paper Mill class II inspection; Port Angeles, Washington; November 14-16, 1988. Washington Department of Ecology, Environmental Investigations and Laboratory Services Program, Olympia, WA.

Hallinan, P., and C.E. Ruiz. 1990. Georgia Pacific, Bellingham class II inspection. Washington Department of Ecology, Environmental Investigations and Laboratory Services Program, Olympia, WA.

Hallinan, P. 1990a. British Petroleum Oil Refinery class II inspection. Washington Department of Ecology, Environmental Investigations and Laboratory Services Program, Olympia, WA.

Hallinan, P. 1990b. Seattle Steel Mill class II inspection; September 11-13, 1989. Washington Department of Ecology, Environmental Investigations and Laboratory Services Program, Olympia, WA.

Heffner, M. 1988. Clallam Bay Sewage Treatment Plant and Sekiu Sewage treatment plant July 28 and 29, 1987 class II inspection reports. Washington Department of Ecology, Water Quality Investigations Section, Olympia, WA.

Heffner, M. 1989a. Pennwalt class II inspection report. Washington Department of Ecology, Environmental Investigations and Laboratory Services Program, Olympia, WA.

Heffner, M. 1989b. Intalco class II inspection; July 25-27, 1988. Washington Department of Ecology, Environmental Investigations and Laboratory Services, Olympia, WA.

Heffner, M. 1990a. Lilyblad class II inspection report; December 29-30, 1988. Washington Department of Ecology, Environmental Investigations and Laboratory Services, Olympia, WA.

Heffner, M. 1990b. U.S. Oil and Refining Company; June 13-14, 1989; class II inspection report. Washington Department of Ecology, Environmental Investigations and Laboratory Services, Olympia, WA.

Heffner, M. 1990c. Central Kitsap Wastewater Treatment Plant class II inspection; November 29-30, 1988. Washington Department of Ecology, Environmental Investigations and Laboratory Services Program, Olympia, WA.

Howard, P.H. 1989. Handbook of environmental fate and exposure data for organic chemicals. Vol. 1. Lewis Publishers, Chelsea, MI.

Howard, P.H. 1990. Handbook of environmental fate and exposure data for organic chemicals. Vol. 2. Lewis Publishers, Chelsea, MI.

HSDB. 1988. Hazardous substance database databank # 200 (DDT), 1625 (DDE), 285 (DDD) (as cited in ATSDR 1989e. Draft toxicity profile for p,p'-DDT, p,p'-DDE, p,p'-DDD. U.S. Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, GA).

Isensee A.A., E.R. Holden, E.A. Woolson, et al. 1976. Soil persistence and aquatic bioaccumulation potential of hexachlorobenzene (HCB). J. Agric. Food Chem. 24:1210-1214.

Johnson, A., and S. Prescott. 1982a. Receiving environment survey in the Puyallup River at the Tacoma Central (No. 1) Sewage Treatment Plant, 7/28/81, 8/25/81, 2/1/82. Washington Department of Ecology, Olympia, WA.

Johnson, A., and S. Prescott. 1982b. Receiving environment survey at the St. Regis Paper Company, Tacoma, Washington. August 11-12, 1981. Washington Department of Ecology, Olympia, WA.

Johnson, A., and S. Prescott. 1982c. Receiving environment survey in Hylebos Waterway at the Pennwalt Corporation Facility, Tacoma, Washington. June 2, 1981. Washington Department of Ecology, Olympia, WA.

Johnson, A., and S. Prescott. 1982d. Receiving environment survey in Hylebos Waterway at the Sound Refining Facility, Tacoma, Washington. June 30, 1981. Washington Department of Ecology, Olympia, WA.

Johnson, A., and D. Norton. 1989. Screening survey for chemical contaminants and toxicity in drainage basins at Paine Field; August 10-12, 1987. Washington Department of Ecology, Environmental Investigations and Laboratory Services Program, Olympia, WA.

Jones & Stokes. 1983. Water quality management program for Puget Sound. Prepared for U.S. Environmental Protection Agency Region 10, Seattle, WA. Jones & Stokes Associates, Inc., Sacramento, CA.

Joy, J. 1987. An investigation of stormwater and effluent discharge from the Boeing Company facility in Auburn. January 1987. Washington Department of Ecology, Water Quality Investigations Section, Olympia, WA.

Konasewich, D.E., P.M. Chapman, E. Gerencher, et al. 1982. Effects, pathways, processes, and transformation of Puget Sound contaminants of concern. NOAA Technical Memorandum OMPA-20. National Oceanic and Atmospheric Administration, Boulder, CO.

Landolt, M.L., D.L. Kalman, A. Nevissi, et al. 1987. Potential toxicant exposure among consumers of recreationally caught fish from urban embayments of Puget Sound. NOAA Technical Memorandum NOS OMA 33. National Oceanic and Atmospheric Administration, Rockville, MD. 111 pp.

Leuenberger, C., W. Giger, R. Coney, et al. 1985. Persistent chemicals in pulp mill effluents. Water Res. 19:885-894.

Malins, D.C., B.B. McCain, D.W. Brown, et al. 1980. Chemical contaminants and biological abnormalities in central and southern Puget Sound. NOAA Technical Memorandum OMPA-2. National Oceanic and Atmospheric Administration, Boulder, CO.

Malins, D.C., B.B. McCain, D.W. Brown, et al. 1982. Chemical contaminants and abnormalities in fish and invertebrates from Puget Sound. NOAA Technical Memorandum OMPA-19. National Oceanic and Atmospheric Administration, Boulder, CO.

Martin, D., and S. Pavlou. 1985. Sources of contamination in Puget Sound. Puget Sound Notes: November 1985. U.S. Environmental Protection Agency, Washington, DC. and Washington Department of Ecology, Olympia, WA.

Metro. 1981 (revised 1983). Analytical support and data validity: organics. Prepared for Toxicant Pretreatment Planning Study. Municipality of Metropolitan Seattle, Seattle, WA.

Mullin, M.D., C.M. Pochini, S. McCrindle, et al. 1984. High-resolution PCB analysis: synthesis and chromatographic properties of all 209 PCB congeners. Environ. Sci. Technol. 18:468-476.

NCASI. 1986. Procedure for the analysis of resin and fatty acids in pulp mill effluents. Technical Bulletin No. 501. National Council of the Paper Industry for Air and Stream Improvement., New York, NY.

Nauen, C.E. 1983. Compilation of legal limits for hazardous substances in fish and fishery products. FAO Fisheries Circular No. 764. Food and Agriculture Organization of the United Nations, Rome, Italy.

Neff, J.M., J.Q. Word, and T.C. Gulbransen. 1987. Recalculation of screening level concentrations for nonpolar organic contaminants in marine sediments. Final Report. Prepared for U.S. Environmental Protection Agency, Criteria and Standards Division, Office of Water Regulations and Standards, Washington, DC. Battelle, Washington Environmental Program Office, Washington, DC.

Norton, D. 1985a. Washington Department of Ecology project position data PP1.8: log sort yards as metals sources to Commencement Bay waterways. Washington Department of Ecology, Olympia, WA.

Norton, D. 1985b. Washington Department of Ecology project position data PP4.5: metals in Sitcum Waterway sediments. Washington Department of Ecology, Olympia, WA.

Norton, D. 5 February 1986. Personal Communication. Memorandum to Tom Eaton regarding results of priority pollutant analyses on water sediment, and clam samples collected in lower Budd Inlet near McFarland Cascade, Olympia, WA. Washington Department of Ecology, Olympia, WA.

Norton, D., M. Stinson, and B. Yake. 1987. Investigation of 4-methylphenol in bottom sediments from log rafting areas of Commencement Bay, Washington. Washington Department of Ecology, Water Quality Investigations Section, Olympia, WA.

Norton, D. 1988. McNeil Island: intertidal screening survey for toxic chemicals in water, sediment, and clam tissue. Washington Department of Ecology, Environmental Investigations and Laboratory Services, Olympia, WA.

Norton, D. 1990a. Use of sediment traps to monitor contaminant flux to city waterway sediments: interim report. Washington Department of Ecology, Environmental Investigations and Laboratory Services Program, Olympia, WA.

Norton, D. 1990b. Chemical contaminants in surface runoff from the abandoned Cascade Pole (Olympia) wood treating facility. Washington Department of Ecology, Environmental Investigations and Laboratory Services Program, Olympia, WA.

Osborn, J. 1980a. Commencement Bay waterways survey; September 23-24, 1980. U.S. Environmental Protection Agency Region 10, Seattle, WA.

Osborn, J. 1980b. Commencement Bay/Port of Tacoma field study; June 2, 1980. U.S. Environmental Protection Agency Region 10, Seattle, WA.

Osborn, J.G., D.E. Weitkamp, and T.H. Schadt. 1985. Alki wastewater treatment plant outfall improvements predesign study. Technical Report No. 6.0, Marine Biology. Municipality of Metropolitan Seattle, Seattle, WA.

Palmork, K.H., S. Wilhelmson, and T. Neppelberg. 1973. The contribution from various industries of polynuclear aromatic hydrocarbons to the marine environment. Fisheries Improvement Comm. Report No. 1973/E33. Intern. Council for Explor. of the Sea.

Pastorok, R., H. Beller, S. Becker, et al. 1988. Everett Harbor action program: analysis of toxic problem areas. Final Report. Prepared for U.S. Environmental Protection Agency Region 10, Office of Puget Sound, Seattle, WA. PTI Environmental Services, Bellevue, WA, and Tetra Tech, Inc., Bellevue, WA.

Phillips, D.J.H. 1990. Arsenic in aquatic organisms: a review, emphasizing chemical speciation. Aqua. Tox. 16:151-186.

PSAMP. 1990a. Puget Sound ambient monitoring program 1989: marine sediment monitoring. Prepared for Washington Department of Ecology, Olympia, WA. Tetra Tech, Inc., Bellevue, WA.

PSAMP. 1990b. Puget Sound ambient monitoring program quality assurance reviews of chemical and bioassay analyses. 1990 Field Survey. Final Report. Prepared for Washington Department of Ecology, Olympia, WA. PTI Environmental Services, Bellevue, WA.

PSDDA. 1988. Puget Sound dredged disposal analysis baseline survey of Phase I disposal sites. Prepared for U.S. Environmental Protection Agency Region 10, Office of Puget Sound, Seattle, WA. PTI Environmental Services, Bellevue, WA.

PSDDA. 1989. Puget Sound dredged disposal analysis baseline survey of Phase II disposal sites. Prepared for U.S. Environmental Protection Agency Region 10, Office of Puget Sound, Seattle, WA. PTI Environmental Services, Bellevue, WA.

PSEP. 1986. Users manual for the pollutant of concern matrix. Prepared for U.S. Environmental Protection Agency Region 10, Office of Puget Sound, Seattle, WA. Tetra Tech, Inc., Bellevue, WA.

PSEP. 1988. Pesticides of concern in the Puget Sound basin: a review of contemporary pesticide usage. Prepared for U.S. Environmental Protection Agency Region 10, Office of Puget Sound, Seattle, WA. Tetra Tech, Inc., Bellevue, WA.

PSEP. 1988b. Health risk assessment of chemical contaminants in Puget Sound seafood. Prepared for U.S. Environmental Protection Agency, Region 10, Office of Puget Sound. Tetra Tech, Inc., Bellevue, WA. 102 pp. + appendices.

PSEP. 1989a. Recommended protocols for measuring metals in Puget Sound water, sediment, and tissue samples. Prepared for U.S. Environmental Protection

Agency Region 10, Office of Puget Sound, Seattle, WA. PTI Environmental Services, Bellevue, WA.

PSEP. 1989b. Recommended guidelines for measuring organic compounds in Puget Sound sediment and tissue samples. Prepared for U.S. Environmental Protection Agency Region 10, Office of Puget Sound, Seattle, WA. PTI Environmental Services, Bellevue, WA.

PSEP. 1991. Puget Sound pesticide reconnaissance survey. Prepared for U.S. Environmental Protection Agency Region 10, Office of Coastal Waters, Seattle, WA. PTI Environmental Services, Bellevue, WA.

PTI. 1990. South Puget Sound reconnaissance survey quality assurance reports for chemical analyses. Prepared for U.S. Environmental Protection Agency Region 10, Seattle, WA. PTI Environmental Services, Bellevue, WA.

Quinlan, E.A., P.M. Chapman, R.N. Dexter, et al. 1985. Toxic chemicals and biological effects in Puget Sound: states and scenarios for the future. Draft Report. NOAA Technical Memorandum. National Oceanic and Atmospheric Administration, Boulder, CO.

Reif, D. 1988a. Bellingham Post Point Pollution Control Plant class II inspection. Washington Department of Ecology, Water Quality Investigations Section, Olympia, WA.

Reif, D. 1988b. Port Townsend Paper Company class II inspection. Washington Department of Ecology, Environmental Investigations and Laboratory Services Program, Olympia, WA.

Reif, D. 1990. Shell Oil's Anacortes Refinery class II inspection; February 22-23, 1989. Washington Department of Ecology, Environmental Investigations and Laboratory Services Program, Olympia, WA.

Romberg, G.P., S.P. Pavlou, and B.A. Crecelius. 1984. Presence, distribution, and fate of toxicants in Puget Sound and Lake Washington. Metro Toxicant Program Report No. 6A. Toxicant Pretreatment Planning Study Technical Report C1. Municipality of Metropolitan Seattle, Seattle, WA.

Sherwood, M.J., A.J. Mearns, D.R. Young, et al. 1980. A comparison of trace contaminants in diseased fishes from three areas. Southern California Coastal Water Research Project, Long Beach, CA.

Sittig, M. (ed.). 1980. Priority toxic pollutants. NOYES Data Corporation, Park Ridge, NJ.

Stanley C., J. Barney, M. Helton, et al. 1971. Measurement of atmospheric levels of pesticides. Environ. Sci. Technol. 5:430-435 (as cited in ATSDR

1989e. Draft toxicity profile for p,p'-DDT, p,p'-DDE, p,p'-DDD. U.S. Public Health Service. Agency for Toxic Substances and Disease Registry. Atlanta, GA).

Stinson, M., D. Norton, and A. Johnson. 1987. An investigation into potential sources of PCB contamination in Hylebos Waterway. Washington Department of Ecology, Water Quality Investigations Section, Olympia, WA.

Stinson, M., and D. Norton. 1987a. Investigation of stormwater discharges to Wheeler Osgood Waterway. Washington Department of Ecology, Water Quality Investigations Section, Olympia, WA.

Stinson, M., and D. Norton. 1987b. Metals concentrations in Asarco discharges and receiving wastes following plant closure. Washington Department of Ecology, Olympia, WA.

Stranks, D.W. 1976. Wood preservatives: their depletion as fungicides and fate in the environment. Forestry Technical Report No. 10. Ottawa Department of the Environment, Canadian Forest Service.

Syracuse Research Corporation. 1989. Toxicological profile for 2,3,7,8-tetrachlorodibenzo-*p*-dioxin. ATSDR/TP-88/23. Prepared for U.S. Public Health Service, Agency for Toxic Substances and Disease Registry in collaboration with the U.S. Environmental Protection Agency, Atlanta, GA.

Tabak, H.H., S.A. Quave, C.I. Mashni, et al. 1981. Biodegradation studies with organic priority pollutant compounds. J. Water Pollut. Control Fed. 53:1503-1518.

Technical Resources, Inc. 1989. Fifth annual report on carcinogens -- Summary 1989. Prepared for the U.S. Department of Health and Human Services, National Institute of Environmental Health Sciences, Research Triangle Park, NC. Technical Resources, Inc., Rockville MD.

Tetra Tech. 1985a. Quality assurance management plan for remedial investigations. Prepared for Black & Veatch, Engineers-Architects and Washington Department of Ecology Remedial Action Division. Tetra Tech, Inc., Bellevue, WA.

Tetra Tech. 1985b. Commencement Bay nearshore/tideflats remedial investigation. Vols. 1, 2, 3, and 4. Prepared for Washington Department of Ecology, Olympia, WA, and U.S. Environmental Protection Agency Region 10, Seattle, WA. Tetra Tech, Inc., Bellevue, WA.

Tetra Tech. 1986a. Guidance manual for health risk assessment of chemically contaminated seafood. Prepared for U.S. Environmental Protection Agency Region 10, Seattle, WA. Tetra Tech, Inc., Bellevue, WA.

Tetra Tech. 1986b. Bioaccumulation monitoring guidance: 6. assessment of potential human health hazards from priority pollutants in edible marine organisms. U.S. Environmental Protection Agency, Office of Marine and Estuarine Protection, Marine Operations Division. Tetra Tech, Inc., Bellevue, WA.

Tetra Tech. 1986c. Commencement Bay/nearshore tideflats feasibility study. Source evaluation refinement. Prepared for Washington Department of Ecology, Olympia, WA, and U.S. Environmental Protection Agency Region 10, Seattle, WA. Tetra Tech, Inc., Bellevue, WA.

Tetra Tech. 1986d. Eagle Harbor Phase II preliminary investigation. Prepared for Black & Veatch, Engineers-Architects and Washington Department of Ecology, Olympia, WA. Tetra Tech, Inc., Bellevue, WA.

Tetra Tech. 1990. Puget Sound ambient monitoring program 1989: marine sediment monitoring. Final Report. Prepared for Washington Department of Ecology, Olympia, WA. Tetra Tech, Inc., Bellevue, WA.

Trial, W., and J. Michaud. 1985. Alki wastewater treatment plant outfall improvements predesign study. Technical Report No. 8.3. Municipality of Metropolitan Seattle, Seattle, WA.

U.S. EPA. 1980. Ambient water quality criteria for silver. EPA 440/5-80-071. U.S. Environmental Protection Agency, Office of Water Regulations and Standards, Washington, DC.

U.S. EPA. 1981. Health assessment document for cadmium. U.S. Environmental Protection Agency, Office of Research and Development, Washington, DC.

U.S. EPA. 1983a. Methods for chemical analysis of water and wastes. EPA 600/4-79-020. Environmental Monitoring and Support Laboratory, Cincinnati, OH.

U.S. EPA. 1983b. Results of the nationwide urban runoff program. Vol. I. Final Report. NTIS No. PB84-185552. U.S. Environmental Protection Agency, Washington, DC.

U.S. EPA. 1984a. Guidelines establishing test procedures for the analysis of pollutants under the Clean Water Act. U.S. Environmental Protection Agency, Washington, DC. Federal Register 49(209):43234-43436.

U.S. EPA. 1984b. Ambient water quality criteria for copper — 1984. U.S. Environmental Protection Agency, Office of Research and Development, Washington, DC.

U.S. EPA. 1984c. Mercury health effects update; health issue assessment; final report. EPA 600/8-84-019F. U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Washington, DC.

U.S. EPA. 1984d. Health assessment document for inorganic arsenic. EPA 600/8-83-021F. U.S. Environmental Protection Agency, Office of Research and Development, Office of Health and Environmental Assessment, Research Triangle Park, NC.

U.S. EPA. 1985a. National primary drinking water regulations; volatile synthetic organic chemicals; final rule and proposed rule. U.S. Environmental Protection Agency, Washington, DC. Federal Register 50(219):46880-47022.

U.S. EPA. 1985b. Water quality criteria; availability of documents. U.S. Environmental Protection Agency, Washington, DC. Federal Register 50(145):30784-30796.

U.S. EPA. 1985c. Health assessment document for polychlorinated dibenzo-*p*-dioxins. EPA 600/8-84-014F. U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Washington, DC.

U.S. EPA. 1986a. Quality criteria for water 1986. EPA 440/5-86-001. U.S. Environmental Protection Agency, Office of Water Regulations and Standards, Washington, DC.

U.S. EPA. 1986b. Verified reference doses (RfDs) of the U.S. Environmental Protection Agency. U.S. Environmental Protection Agency, Office of Research and Development, Washington, DC.

U.S. EPA. 1988. Briefing report on the Apparent Effects Threshold approach. Submitted to the EPA Science Advisory Board by U.S. Environmental Protection Agency Region 10, Office of Puget Sound, Seattle, WA. PTI Environmental Services, Bellevue, WA.

U.S. EPA. 1990a. U.S. EPA Contract Laboratory Program statement of work for inorganics analysis, multi-media, multi-concentration. Document No. ILM01.0. U.S. Environmental Protection Agency, Washington, DC.

U.S. EPA. 1990b. U.S. EPA Contract Laboratory Program statement of work for organics analysis, multi-media, multi-concentration. Document No. OLM01.0. U.S. Environmental Protection Agency, Washington, DC.

U.S. EPA. 1990c. Health effects assessment summary tables: fourth quarter FY-1990. OSWER (OS-230), ORD (RD-689), OERR 9200 6-303 (90-4). U.S. Environmental Protection Agency, Washington, DC.

USITC. 1981. Synthetic organic chemicals, Unites States production and sales, 1980. USITC Publication No. 1183. U.S. International Trade Commission, U.S. Government Printing Office, Washington, DC. (as cited in Technical Resources, Inc. 1989).

U.S. FDA. 1982. Action levels for poisonous or deleterious substances in human food and animal feed. U.S. Food and Drug Administration, Washington, DC.

U.S. FDA. 1984. Polychlorinated biphenyls (PCBs) in fish and shellfish; reduction of tolerance; final decision. U.S. Food and Drug Administration, Rockville, MD. Federal Register 49(100)21514-21520.

U.S. FWS. 1988. Arsenic hazards to fish, wildlife, and invertebrates: a synoptic review. U.S. Department of the Interior, Fish and Wildlife Service, Patuxent Wildlife Research Center, Laurel, MD.

U.S. Navy. 1985. Final environmental impact statement. Carrier battle group Puget Sound region ship homeporting project. Technical Appendix, Vol. 2. Prepared for U.S. Department of the Navy, Western Division, Naval Facilities Engineering Command, San Bruno, CA.

Vieth, G.D., D.L. Defoe, and B.V. Bergstedt. 1979. Measuring and estimating the bioconcentration factor of chemicals in fish. J. Fish. Res. Board Can. 36:1046-1048.

Yake, W., J. Joy, and A. Johnson. 1984. Chemical contaminants in clams and crabs from Eagle Harbor, Washington state with emphasis on polynuclear aromatic hydrocarbons. Washington Department of Ecology, Water Quality Division, Olympia WA.

Young, D.R., G.V. Alexander, and D. McDermott-Ehrlich. 1979. Vessel-related contamination of southern California harbors by copper and other metals. Mar. Poll. B., 10:50-56.

Zarba, C. March 1989. Personal Communication. Memorandum to U.S. Environmental Protection Agency Region 10, Office of Puget Sound, Seattle, WA.

Zinner, L. 1991. Sequim wastewater treatment plant class II inspection; August 1990. Washington Department of Ecology, Environmental Investigations and Laboratory Services Program, Olympia, WA.

APPENDIX A:

SUPPLEMENTAL INFORMATION

TABLE A-1. SUMMARY OF BASIC ANALYTICAL TECHNIQUES FOR ORGANIC COMPOUND ANALYSES

Analytical Step	Analytical Procedure[a]
Sample drying	Centrifugation or sodium sulfate
Extraction	Shaker/roller; Soxhlet®; sonication
Extract drying	Separatory funnel partitioning as needed to remove water (pH must be controlled); sodium sulfate for all other extract drying. Kuderna-Danish apparatus (to *ca.* 1 mL), rotary evaporation (to 2 mL) or comparable technique; purified nitrogen gas for concentration to smaller volumes
Extract cleanup	Removal of elemental sulfur (sediments only) with mercury or activated copper
	Removal of organic interferents with GPC, size exclusion chromatography (e.g., phenogel, Sephadex®), bonded octadecyl columns, HPLC, silica gel, or alumina
Extract analysis	GC/MS for volatiles and semivolatiles; GC/ECD for pesticides and PCB mixtures

[a] GPC - gel permeation chromatography, HPLC - high performance liquid chromatography, GC/MS - gas chromatography/mass spectrometry, GC/ECD - gas chromatography/electron capture detection, PCB - polychlorinated biphenyls.

[b] The steps described generally apply to low parts per billion, broad scan analyses. Some of the options for extract cleanup and analysis are best suited for certain compound groups rather than for broad scan analyses.

TABLE A-2. REFERENCE DOSE (RID) VALUES FOR PRIORITY POLLUTANTS

PP#	Pollutant	CAS #	RID (mg/kg/day)
114	antimony	7440-36-0 (a)	0.00004
95	alpha-endosulfan	115-29-7	0.00005
96	beta-endosulfan	115-29-7	0.00005
127	thallium (in soluble salts)	563-68-8 (a)	0.00007
123	mercury	7439-97-6 (a)	0.0003
33	1,3-dichloropropene	10061-02-6	0.0003
98	endrin	72-20-8	0.0003
56	nitrobenzene	98-95-3	0.0005
53	hexachlorocyclopentadiene	77-47-4	0.0007
46	bromomethane	74-83-9	0.0014
59	2,4-dinitrophenol	51-28-5	0.002
126	silver	7440-22-4 (a)	0.003
31	2,4-dichlorophenol	120-83-2	0.003
119	chromium (VI)	7440-47-3 (a)	0.005
66	bis(2-ethylhexyl)phthalate	117-81-7	0.02
7	chlorobenzene	108-90-7	0.02
121	cyanide	57-12-5 (a)	0.02
124	nickel	7440-02-0 (a)	0.02
64	pentachlorophenol	87-86-5	0.03
39	fluoranthene	206-44-0	0.04
42	bis(2-chloroisopropyl)ether	39638-32-9	0.04
44	dichloromethane (methylene chloride)	75-09-02	0.06
25	1,2-dichlorobenzene	95-50-1	0.09
11	1,1,1,-trichloroethane	71-55-6	0.09
38	ethylbenzene	100-41-4	0.1
54	isophorone	78-59-1	0.1
86	toluene	108-88-3	0.2
65	phenol	108-95-2	0.6
70	diethyl phthalate	84-66-2	0.8
71	dimethyl phthalate	131-11-3	1
119	chromium (III)	7440-47-3 (a)	1
68	di-n-butyl phthalate	87-74-2	10
45	chloromethane (methyl chloride)	74-87-3	(b)
60	4,6-dinitro-o-cresol	534-52-1	(b)
26	1,3-dichlorobenzene	541-73-1	(b)
97	endosulfan sulfate	1031-07-8	(c)
27	1,4-dichlorobenzene	106-46-7	(c)
2	acrolein	107-02-8	(c)
125	selenium	7782-49-2	(c)

(a) CAS numbers for these substances vary depending on their specific form (e.g., inorganic salts or organic complexes).
(b) Data inadequate for quantitative risk assessment.
(c) Not determined.
Reference: U.S. EPA (1990c), Table A.

TABLE A-3. CARCINOGENIC PRIORITY POLLUTANTS RANKED BY POTENCY FACTORS

PP#	Pollutant	CAS Number	Potency (a)	Level of Evidence (b)
129	2,3,7,8-TCDD (dioxin)	1746-01-6	150000	B2
5	benzidene	92-87-5	230 (O)	A
61	N-nitrosodimethylamine	62-57-1	51 (W)	B2
89	aldrin	309-00-2	17	B2
90	dieldrin	60-57-1	16	B2
(c)	polychlorinated biphenyls	(c)	8.9	B2
102	alpha-HCH	319-84-6	6.3	B2
100	heptachlor	76-44-8	4.5	B2
117	beryllium	7440-41-7 (d)	4.3 (W)	B2
88	vinyl chloride	75-01-4	1.9	A
103	beta-HCH	319-85-7	1.8	C
115	arsenic	7440-38-2 (d)	1.8 (W)	A
9	*hexachlorobenzene*	118-74-1	1.6	B2
105	gamma-HCH	58-89-9	1.3	B2-C
91	chlordane	57-74-9	1.3	B2
18	bis(2-chloroethyl)ether	111-44-4	1.1	B2
113	toxaphene	8001-35-2	1.1	B2
37	1,2-diphenylhydrazine	122-66-7	0.80	B2
35	2,4-dinitrotoluene	121-14-2	0.68	B2
3	acrylonitrile	107-13-1	0.54 (W)	B1
28	3,3'-dichlorobenzidine	91-94-1	0.46	B2
92	4,4'-DDT	50-29-3	0.34	B2
93	4,4'-DDE	72-55-9	0.34	B2
94	4,4'-DDD	72-54-8	0.24	B2
15	1,1,2,2-tetrachloroethane	79-34-5	0.20	C
6	carbon tetrachloride	56-23-5	0.13	B2
10	1,2-dichloroethane	107-06-2	0.091	B2
52	*hexachlorobutadiene*	87-68-3	0.078	C
14	1,1,2-trichloroethane	79-00-5	0.057	C
86	tetrachloroethane	127-18-4	0.051	B2
4	benzene	71-43-2	0.029 (O)	A
12	hexachloroethane	67-72-1	0.014	C
21	2,4,6-trichlorophenol	88-06-2	0.011	B2
44	dichloromethane (methylene chloride)	75-09-02	0.0075 (I+W)	B2
23	chloroform	67-66-3	0.0061 (W)	B2
62	N-nitrosodiphenylamine	86-30-6	0.0049	B2
118	cadmium (e)	7440-43-9 (d)	(e)	(e)
73	benzo(a)pyrene	50-32-8	(e)	B2
119	chromium (VI) (e)	7440-47-3 (d)	(e)	(e)
29	1,1-dichloroethane	75-35-4	(e)	C
124	nickel (subsulfide, refinery dust) (e)	7440-02-0 (d)	(e)	(e)

TABLE A–3 (Continued).

(a) From U.S. Environmental Protection Agency (1990c), Table B. All potency values represent dietary slope factors except: (O) = slope calculated from occupational exposure, W = slope calculated from human drinking water exposure, and (I) = slope calculated from animal inhalation studies.
(b) A = Human carcinogen (sufficient evidence of carcinogenicity in humans); B = Probable human carcinogen (B1 – limited evidence of carcinogenicity in humans; B2 – sufficient evidence of carcinogenicity in animals with inadequate or lack of evidence in humans); C = Possible human carcinogen (limited evidence of carcinogenicity in animals or lack of human data).
(c) Specific Aroclor mixtures of polychlorinated biphenyls are listed as individual priority pollutants with individual CAS numbers.
(d) CAS numbers for these substances vary depending on their specific form (e.g., organic salts or or organic complexes).
(e) Chromium (VI), cadmium, and nickel are not considered to be carcinogenic via dietary exposure.

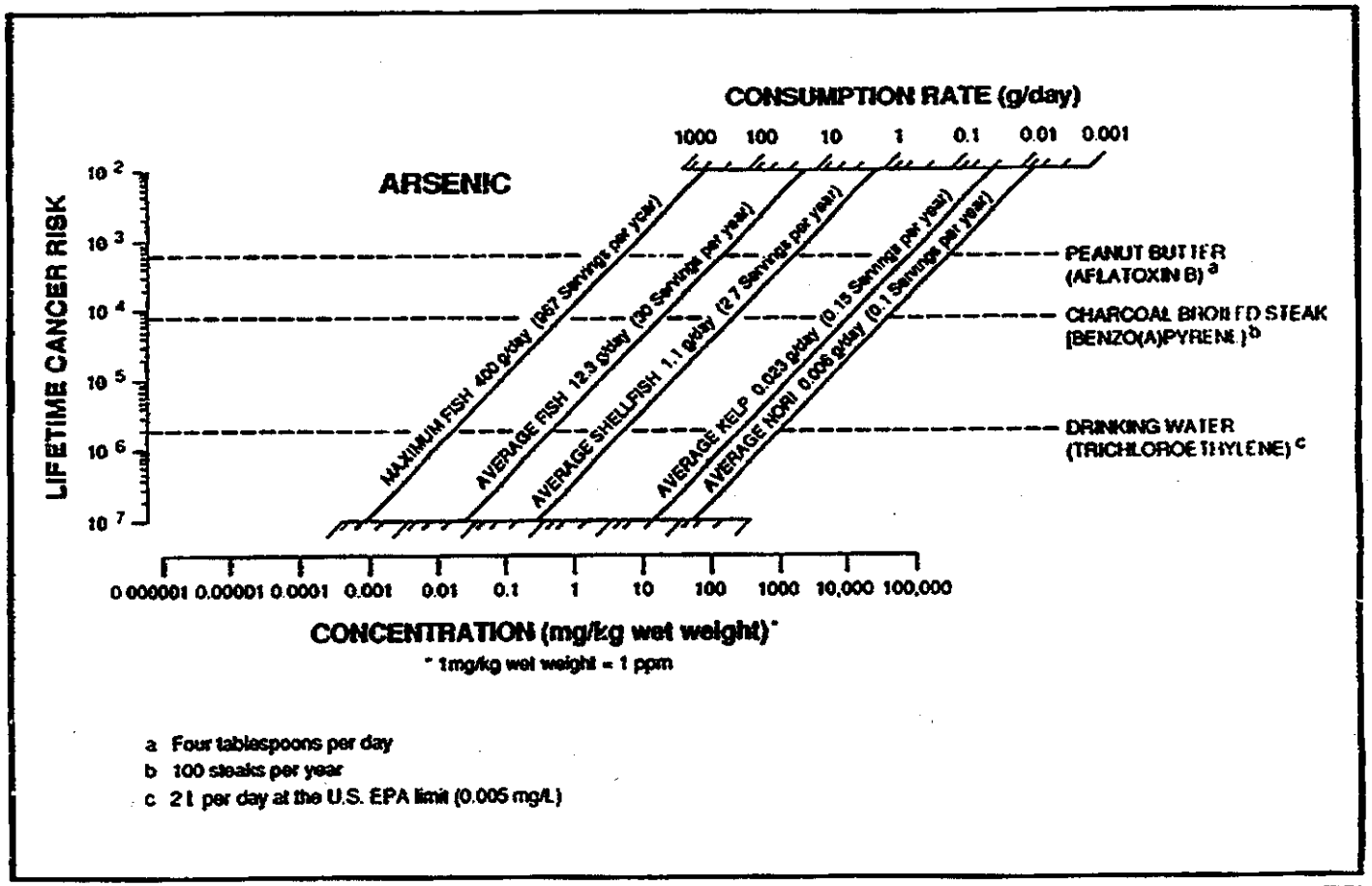

Figure A-1. Graphical risk characterization for arsenic in seafood (PSEP 1988b).

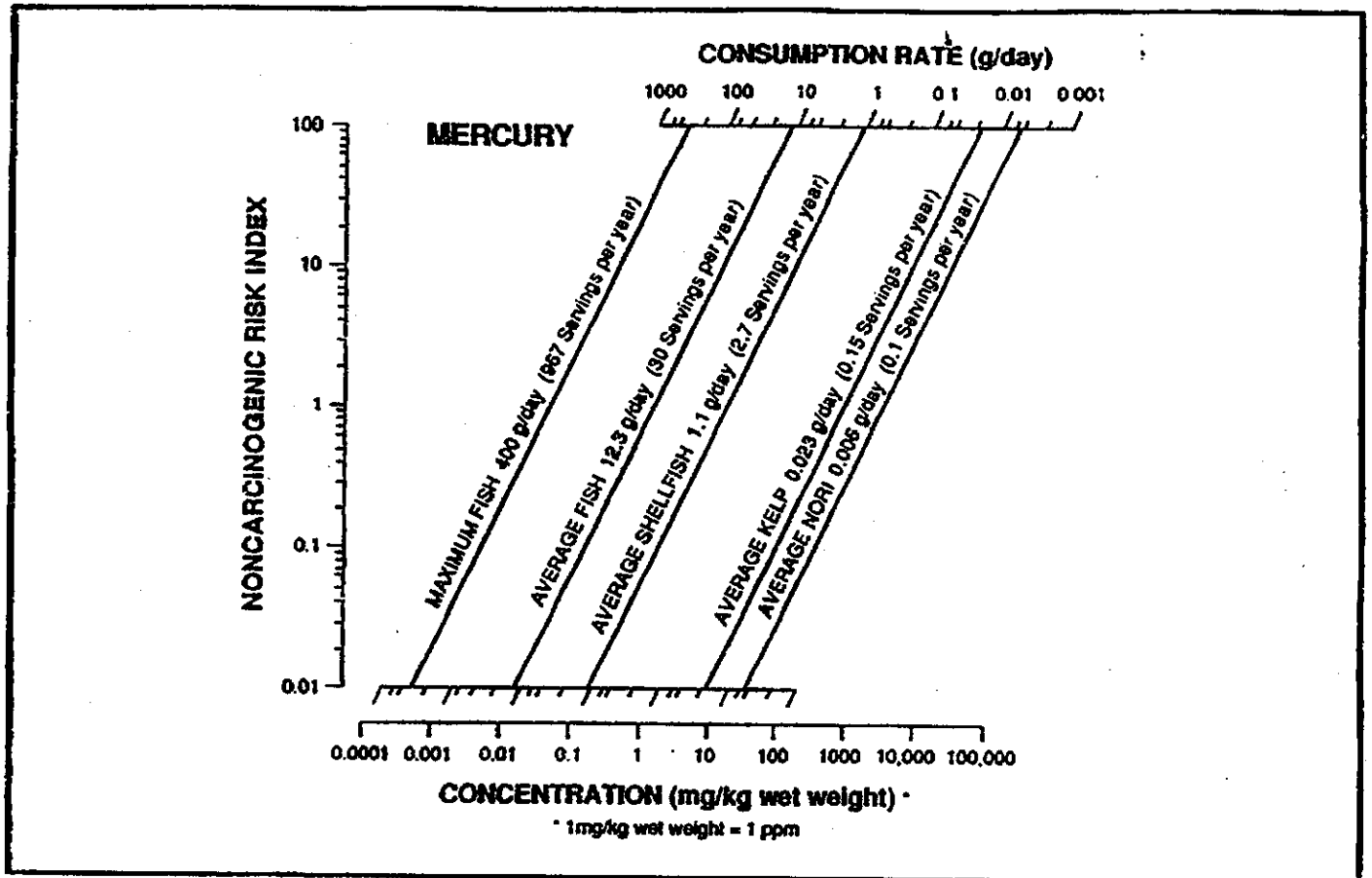

Figure A-2. Graphical risk characterization for mercury in seafood (PSEP 1988b).

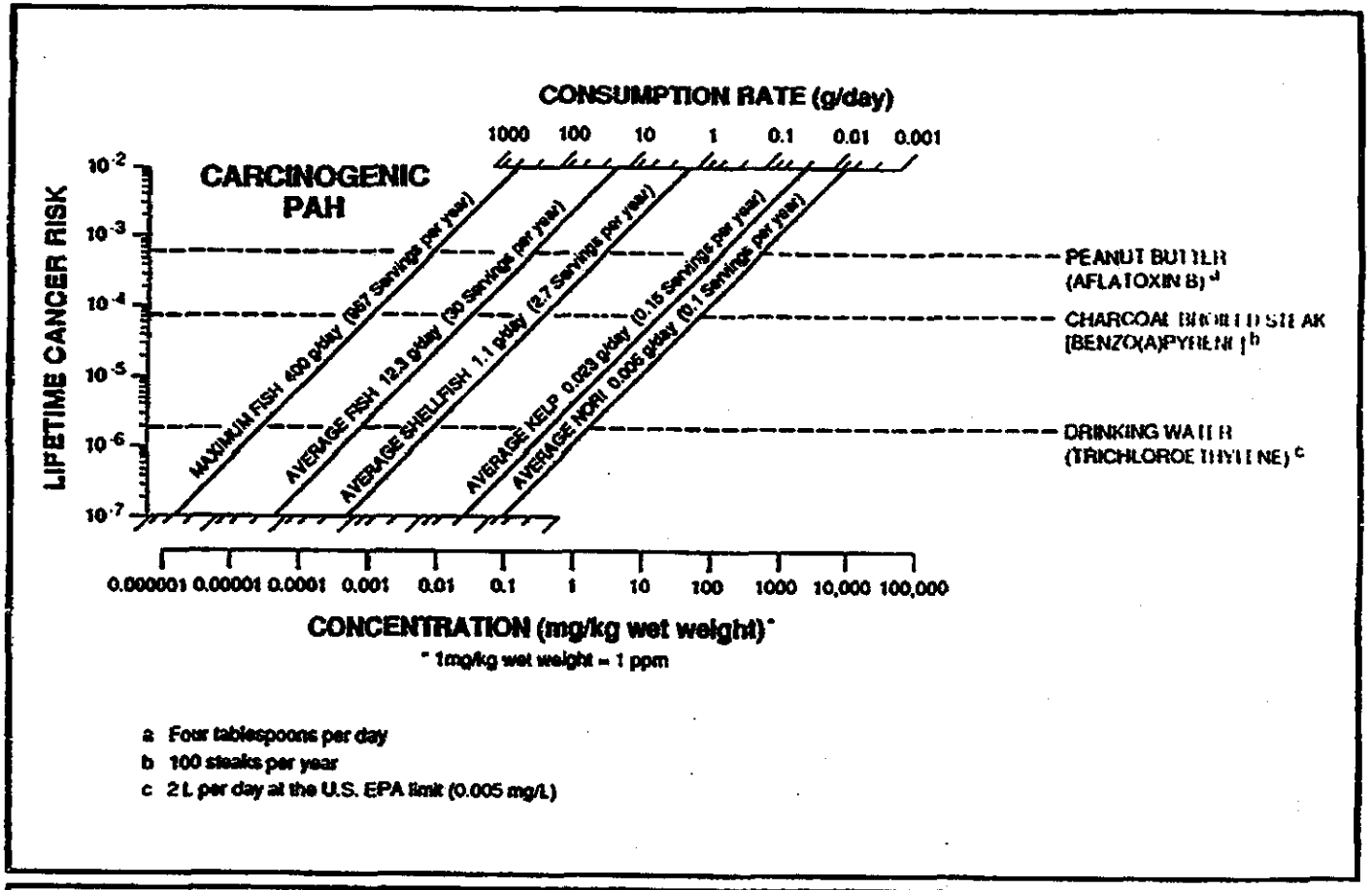

Figure A-3. Graphical risk characterization for carcinogenic PAH in seafood (PSEP 1988b).

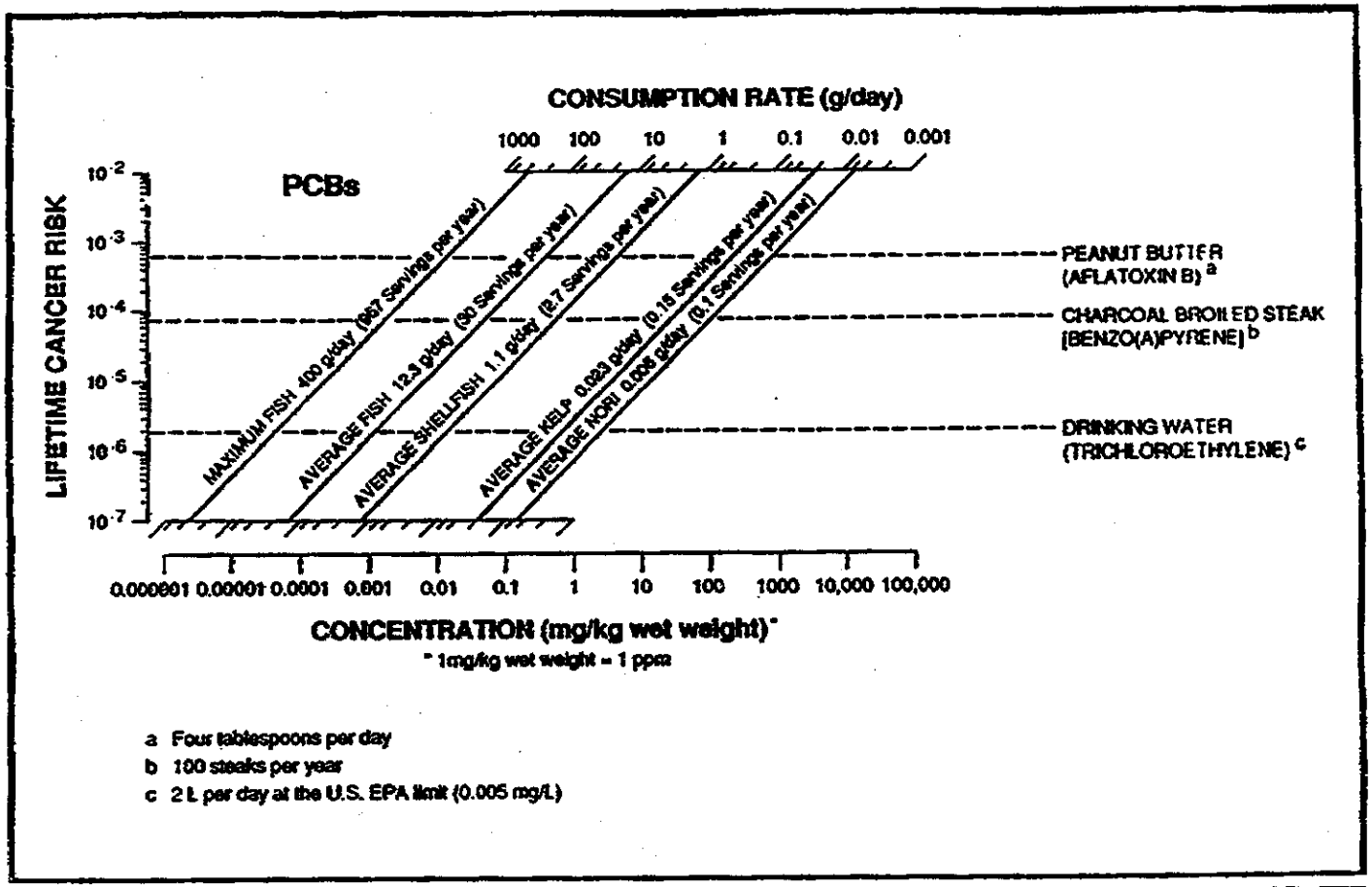

Figure A-4. Graphical risk characterization for PCBs in seafood (PSEP 1988b).