WATER QUALITY CRITERIA FOR POLYCHLORINATED BIPHENYLS (PCBs)

MINISTRY OF ENVIRONMENT, LANDS AND PARKS PROVINCE OF BRITISH COLUMBIA

TECHNICAL APPENDIX

N. K. Nagpal, Ph. D.
Water Quality Branch
Water Management Division

January, 1992

ACKNOWLEDGEMENTS

The author gratefully acknowledges comments provided by many individuals from both the British Columbia and the Federal governments, during the preparation of this document. The following is a list of the reviewers, in alphabetical order, and their affiliation.

Richard Arsenault, Environment Canada
Julia Beatty Spence, Ministry of Environment, Smithers, British Columbia
Ray Copes, Ministry of Health, Victoria, British, Columbia
Bob Cox, Ministry of Environment, Victoria, British Columbia
Laurena Hansel, Ministry of Environment, Surrey, British Columbia
Vic Jensen, Ministry of Environment, Penticton, British Columbia
Les McDonald, Ministry of Environment, Cranbrook, British Columbia
Mike McFarlane, Ministry of Environment, Victoria, British Columbia
Larry Pommen, Ministry of Environment, Victoria, British Columbia
Chris Pharo, Environment Canada, Vancouver
Roland Rocchini, Ministry of Environment, Victoria, British Columbia
Sherry Walker, Environment Canada, Ottawa
Doug Walton, Ministry of Environment, Surrey, British Columbia
Barry Willoughby, Ministry of Health, Victoria, British, Columbia

1. INTRODUCTION

This document discusses the characteristics of polychlorinated biphenyls (PCBs), their effects on various water uses, and the derivation of criteria to protect these uses. The water use categories considered here include drinking water, aquatic life, wildlife, livestock watering, irrigation, recreation and aesthetics, and industrial water supplies. Polychlorinated biphenyls are commercial products which found widespread applications because of their stability, inertness, and their excellent dielectric properties. Although their use in industry is now limited, PCBs still persist in the environment partly due to their resistance to breakdown and partly due to improper disposal.

Both humans and aquatic life are sensitive to the toxic effects of PCBs. Where applicable, and where sufficient information exists, criteria are recommended to protect water users from PCBs. Standards, objectives, criteria, and accompanying rationale from other jurisdictions are reviewed and their suitability for British Columbia waters is considered.

The information presented in this document is mostly based on recent reviews. In addition, a considerable number of research articles pertinent to the subject, appearing in recent issues of various journals, were also considered during the development of the criteria presented here.

2. CHARACTERISTICS AND USES OF PCBS

2.1 General characteristics

PCBs are a group of synthetic aromatic compounds which contain a varying number of chlorine atoms substituted on a biphenyl molecule (Figure 1). In theory, there are 209 possible PCB isomers and congeners (Table 1). It is unlikely that all of them will be formed in the chlorination process; nevertheless, commercially produced PCBs (e.g., Aroclors) are a complex mixture of chlorobiphenyls (Table 2).

Several industrialised countries produced PCBs, which were marketed under various trade names (Table 3). By far, most of the available information in the literature is on Aroclors. These PCB preparations were produced in North America by Monsanto Corporation; no PCBs have been produced in North America since 1977, although

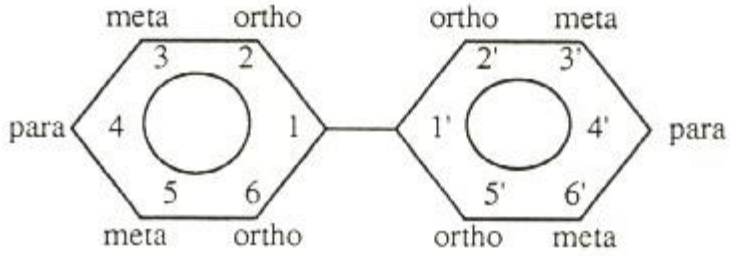
some are still being produced in Europe (Hutzinger *et al.*, 1974; McDonald and Tourangeau, 1986). All Aroclor formulations are characterised by a four digit number. The first two digits indicate the type of molecule (e.g., 12 indicates a biphenyl molecule), whereas the last two digits give the percent of chlorine by weight substituted on the molecule. For instance, Aroclor 1242 (abbreviated as A1242 or A-1242 in the text to follow) is a mixture of chlorinated biphenyls containing 42% of chlorine by weight. The product Aroclor 1016, which is also a mixture of chlorinated biphenyls, is an exception to this rule (see Tables 2 and 5 below). There exist other Aroclor products such as Aroclor 5442 which are mixtures of chlorinated terphenyls, but may contain chlorinated biphenyls as well (Hutzinger et al., 1974).

Most individual chlorobiphenyls are solids at room temperature, whereas the commercial mixtures are mobile oils (e.g., A-1221, A-1232, A-1242, and A-1248), viscous fluids (*e.g.*, A-1254), or sticky resins (e.g., A-1260 and A-1262) (Hutzinger *et al.*, 1974). The outstanding characteristics of PCBs are their (i) thermal stability, (ii) resistance to oxidation, acids, bases, and other chemical agents, and (iii) excellent dielectric properties. Other important properties of PCBs from an environmental point of view relate to their solubility and volatility.

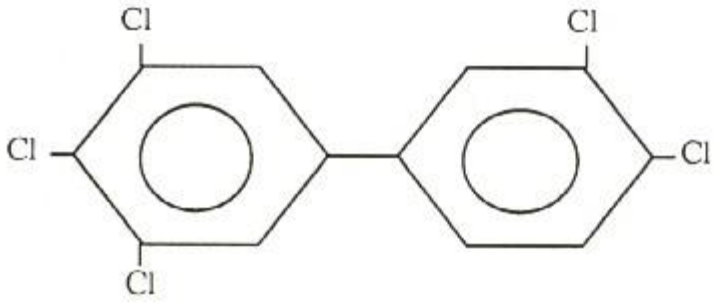
FIGURE 1
STRUCTURE AND NOMENCLATURE OF POLYCHLORINATED BIPHENYLS

FIGURE 1

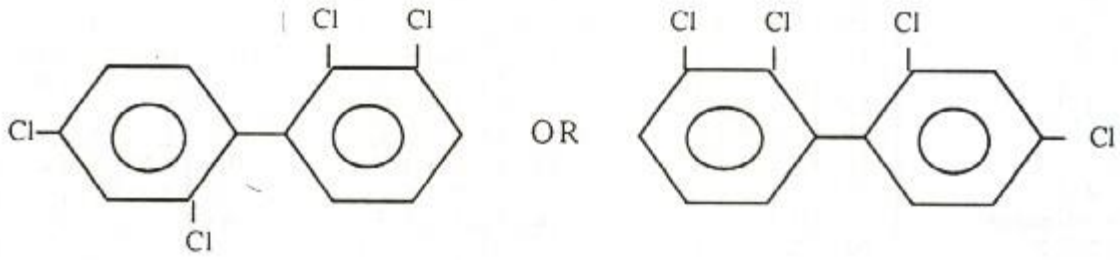
Structure of a biphenyl molecule and nomenclature of polychlorinated biphenyls



(a) A biphenyl molecule showing numbering and substitution (i.e., ortho, meta, and para) system



(b) 3,3',4,4',5-Pentachlorobiphenyl (IUPAC #126)
(A true coplanar PCB)



(c) 2,2',3,4'-Tetrachlorobiphenyl (correct nomenclature of an arbitrarily chosen PCB; sometimes also labeled as 2,3,2',4'-Tetrachlorobiphenyl)

TABLE 1

Systematic (IUPAC) Numbering of Polychlorinated Biphenyl Compounds

(From Ballschmiter and Zell, 1980)

No.	Structure	No.	Structure	No.	Structure	No.	Structure
Monochlorobiphenyls		51	2,2',4,6'	105	2,3,3',4,4'	159	2,3,3',4,5,5'
1	2	52	2,2',5,5'	106	2,3,3',4,5	160	2,3,3',4,5,6
2	3	53	2,2',5,6'	107	2,3,3',4',5	161	2,3,3',4,5',6
3	4	54	2,2',6,6'	108	2,3,3',4,5'	162	2,3,3',4',5,5'
Dichlorobiphenyls		55	2,3,3',4	109	2,3,3',4,6	163	2,3,3',4',5,6
4	2,2'	56	2,3,3',4'	110	2,3,3',4',6	164	2,3,3',4',5',6
5	2,3	57	2,3,3',5	111	2,3,3',5,5'	165	2,3,3',5,5',6
6	2,3'	58	2,3,3',5'	112	2,3,3',5,6	166	2,3,4,4',5,6
7	2,4	59	2,3,3',6	113	2,3,3',5',6	167	2,3',4,4',5,5'
8	2,4'	60	2,3,4,4'	114	2,3,4,4',5	168	2,3',4,4',5',6
9	2,5	61	2,3,4,5	115	2,3,4,4',6	169	3,3',4,4',5,5'
10	2,6	62	2,3,4,6	116	2,3,4,5,6	Heptachlorobiphenyls	
11	3,3'	63	2,3,4',5	117	2,3,4',5,6	170	2,2',3,3',4,4',5
12	3,4	64	2,3,4',6	118	2,3',4,4',5	171	2,2',3,3',4,4',6
13	3,4'	65	2,3,5,6	119	2,3',4,4',6	172	2,2',3,3',4,5,5'
14	3,5	66	2,3',4,4'	120	2,3',4,5,5'	173	2,2',3,3',4,5,6
15	4,4'	67	2,3',4,5	121	2,3',4,5',6	174	2,2',3,3',4,5,6'
Trichlorobiphenyls		68	2,3',4,5'	122	2',3,3',4,5	175	2,2'3,3',4,5',6
16	2,2',3	69	2,3',4,6	123	2',3,4,4',5	176	2,2',3,3',4,6,6'
17	2,2',4	70	2,3',4',5	124	2',3,4,5,5'	177	2,2',3,3',4',5,6
18	2,2',5	71	2,3',4',6	125	2',3,4,5,6'	178	2,2',3,3',5,5',6
19	2,2',6	72	2,3',5,5'	126	3,3',4,4',5	179	2,2',3,3',5,6,6'
20	2,3,3'	73	2,3',5',6	127	3,3',4,5,5'	180	2,2',3,4,4',5,5'
21	2,3,4	74	2,4,4',5	Hexachlorobiphenyls		181	2,2',3,4,4'5,6
22	2,3,4'	75	2,4,4',6	128	2,2',3,3',4,4'	182	2,2',3,4,4',5,6'
23	2,3,5	76	2',3,4,5	129	2,2',3,3',4,5	183	2,2',3,4,4',5',6
24	2,3,6	77	3,3',4,4'	130	2,2',3,3',4,5'	184	2,2',3,4,4',6,6'
25	2,3',4	78	3,3',4,5	131	2,2',3,3',4,6	185	2,2',3,4,5,5',6
26	2,3',5	79	3,3',4,5'	132	2,2',3,3',4,6'	186	2,2',3,4,5,6,6'
27	2,3',6	80	3,3',5,5'	133	2,2',3,3',5,5'	187	2,2',3,4',5,5',6
28	2,4,4'	81	3,4,4',5	134	2,2',3,3',5,6	188	2,2',3,4',5,6,6'

29	2,4,5	Pentachlorobiphenyls		135	2,2',3,3',5,6'	189	2,3,3',4,4',5,5'
30	2,4,6	82	2,2',3,3',4	136	2,2',3,3',6,6'	190	2,3,3',4,4',5,6
31	2,4',5	83	2,2',3,3',5	137	2,2',3,4,4',5	191	2,3,3',4,4',5',6
32	2,4',6	84	2,2',3,3',6	138	2,2',3,4,4',5'	192	2,3,3',4,5,5',6
33	2',3,4	85	2,2',3,4,4'	139	2,2',3,4,4',6	193	2,3,3',4',5,5',6
34	2',3,5	86	2,2',3,4,5	140	2,2',3,4,4',6'	Octachlorobiphenyls	
35	3,3',4	87	2,2',3,4,5'	141	2,2',3,4,5,5'	194	2,2',3,3',4,4',5,5'
36	3,3',5	88	2,2',3,4,6	142	2,2',3,4,5,6	195	2,2',3,3',4,4',5,6
37	3,4,4'	89	2,2',3,4,6	143	2,2',3,4,5,6'	196	2,2',3,3',4,4',5',6
38	3,4,5	90	2,2',3,4',5	144	2,2',3,4,5',6	197	2,2',3,3',4,4',6,6'
39	3,4',5	91	2,2',3,4',6	145	2,2',3,4,6,6'	198	2,2',3,3',4,5,5',6
Tetrachlorobiphenyls		92	2,2',3,5,5'	146	2,2',3,4',5,5'	199	2,2'3,3',4,5,6,6'
40	2,2',3,3'	93	2,2',3,5,6	147	2,2',3,4',5,6	200	2,2',3,3',4,5',6,6'
41	2,2',3,4	94	2,2',3,5,6'	148	2,2',3,4',5,6'	201	2,2',3,3',4',5,5',6
42	2,2',3,4'	95	2,2',3,5',6	149	2,2',3,4',5',6	202	2,2',3,3',5,5',6,6'
43	2,2',3,5	96	2,2',3,6,6'	150	2,2',3,4',6,6'	203	2,2',3,4,4',5,5',6
44	2,2',3,5'	97	2,2',3',4,5	151	2,2',3,5,5',6	204	2,2',3,4,4',5,6,6'
45	2,2',3,6	98	2,2',3',4,6	152	2,2',3,5,6,6'	205	2,3,3',4,4',5,5',6'
46	2,2',3,6'	99	2,2',4,4',5	153	2,2',4,4',5,5'	Nonachlorobiphenyls	
47	2,2',4,4'	100	2,2',4,4',6	154	2,2',4,4',5,6	206	2,2',3,3',4,4',5,5',6
48	2,2',4,5	101	2,2',4,5,5'	155	2,2',4,4',6,6'	207	2,2',3,3',4,4',5,6,6'
49	2,2',4,5'	102	2,2',4,5,6'	156	2,3,3',4,4',5	208	2,2',3,3',4,5,5',6,6'
50	2,2',4,6	103	2,2',4,5',6	157	2,3,3',4,4',5'	Decachlorobiphenyls	
		104	2,2',4,6,6'	158	2,3,3',4,4',6	209	2,2',3,3',4,4',5,5',6,6'

TABLE 2

Approximate molecular composition of Aroclor mixtures (U.S.EPA, 1980)

PCB Formulation Chlorobiphenyl	A-1016	A-1221	A-1232	A-1242	A-1248	A-1254	A-1260
	--- % ---						
Biphenyl	trace	11.0	6.0	-	-	-	-
Monochlorobiphenyl	1.0	51.0	26.0	1.0	-	-	-
Dichlorobiphenyl	20.0	32.0	29.0	17.0	1.0	-	-
Trichlorobiphenyl	57.0	4.0	24.0	40.0	23.0	-	-

Tetrachlorobiphenyl	21.0	2.0	15.0	32.0	50.0	16.0	-
Pentachlorobiphenyl	1.0	0.5	0.5	10.0	20.0	60.0	12.0
Hexachlorobiphenyl	trace	-	-	0.5	1.0	23.0	46.0
Heptachlorobiphenyl	-	-	-	-	-	1.0	36.0
Octachlorobiphenyl	-	-	-	-	-	-	6.0
Nonachlorobiphenyl	-	-	-	-	-	-	-
Decachlorobiphenyl	-	-	-	-	-	-	-

TABLE 3
Trade names of PCB formulations (McDonald and Tourangeau, 1986)

Apirolio	Dykanol	Phenoclor (France)
Aroclor (US, Great Britain)	Elemex	Pydraul (US)
Aroclor B	Eucarel	Pyralene (France)
Asbestol	Fenclor (Italy)	Pyranol (US, Canada)
Askarel	Hyvol	Pyroclor (Great Britain)
Chlorestol	Inclor	Saf-t-kuhl
Chlorinol	Inerteen (US, Canada)	Santotherm FR (Japan)
Chlophen (Germany)	Kanechlor (Japan)	Santovec 1 and 2
DK Decachlorobiphenyl (Italy)	Nepolin	Sovol (USSR)
Diaclor	No-flamol	Therminol FR Series

2.2 Uses

The major Canadian use of PCBs was in dielectric fluid for industrial electrical equipment (McDonald and Tourangeau, 1986). Other products containing PCBs included:

· -waxes
· -adhesives
· -paints
· -heat exchange fluids
· -de-dusting agents

· -vacuum pump oils
· -caulking compounds
· -hydraulic fluids
· -printing inks
· -cutting oils
· -sealants
· -plasticizers
· -carbonless copying paper
· -specialised lubricants
· -bridge bearing
· -fire retardants
· -cable insulating paper
· -lubricants
· -flame-proofing

Some uses of PCBs classified according to the type of Aroclors are shown in Table 4. PCBs mixed with DDT (Lichtenstein *et al.*, 1969), organophosphorus compounds (Fuhremann and Lichtenstein, 1972), and carbaryl (Plapp, 1972) have been reported to enhance the insecticidal properties of these compounds. Although recommended for incorporation into pesticide formulations, PCBs were apparently never used as pesticides (Hutzinger *et al.*,1974).

TABLE 4
Uses of PCBs Classified to Type of Aroclor
(Moore and Ramamoorthy, 1984)

Base material	Aroclor type	Purpose and Effect
Polyvinyl chloride	A-1248, A-1254, A-1260	Secondary plasticizers to increase flame retardance and chemical resistance
Polyvinyl acetate	A-1221, A-1232, A-1242	Improved quick-track & fibre-tear properties
Polyester resins	A-1260	Stronger fibreglass; reinforced resins & economical fire retardants
Polystyrene	A-1221	Plasticizer
Epoxy resins	A-1221, A-1248	Increased resistance to oxidation & chemical attack; better adhesive properties

Styrene-butadiene co-polymers	A-1254	Better chemical resistance
Neoprene	A-1268	Fire retardant; injection moldings
Crepe rubber	A-1262	Plasticizer in paints
Nitrocellulose lacquers	A-1262	Co-plasticizers
Ethylene vinyl acetate	A-1254	Pressure sensitive adhesives
Chlorinated rubber	A-1254	Enhances resistance, flame retardance, electrical insulation properties
Varnish	A-1260	Improved water & alkali resistance
Wax	A-1262	Improved moisture & flame resistance

3. FORMS AND TRANSFORMATIONS IN THE ENVIRONMENT

Several physical, chemical, and biological reactions influence the behaviour of polychlorinated biphenyls in the environment. The discussion in the following sections outlines the transformations of PCBs caused by these reactions.

3.1 Physio-chemical transformations

PCBs are non-polar compounds. Their non-polar nature makes them only slightly soluble in water. In general, the water solubility of a PCB compound decreases as the degree of chlorination increases (Table 5). However, there are some exceptions to this rule; for instance, decachlorobiphenyl is about twice as soluble as 2,2',3,3',4,4',5,5'-octachlorobiphenyl. Within a group of chlorobiphenyls containing the same number of chlorine atoms, the solubility depends on the positions of the chlorine atoms on the biphenyl ring.

The solubility of PCBs is also influenced by the environment as these compounds or preparations show a strong affinity for sediment and organic fractions. Sorption of PCBs on suspended and bottom sediment in an aqueous environment would result in

lower concentrations of PCBs in water. Sorption on the dissolved organic fraction, on the other hand, will probably enhance the concentration of PCBs in water (Sawhney, 1986). PCBs have been shown to adsorb relatively rapidly onto plastic and glass containers (Hutzinger *et al.*, 1974).

Owing to their low solubility's in water, PCBs are often associated with the solid fraction (*e.g.*, particulate matter, sediments) of the aquatic and terrestrial environments. The sorption reactions of PCBs in aquatic and terrestrial systems play an important role in determining their fate and transport in the environment. In general, sorption of PCBs increases with the degree of chlorination (Haque and Schmedding, 1979), the surface area (Hiraizumi *et al*., 1979), and the organic carbon content of the sorbents (Karickhoff *et al.*, 1979; Weber *et al.*, 1983).

PCBs exhibit low vapour pressure which decreases with increased chlorination. The factors which influence the vaporisation of PCBs include surface area of the sorbent, organic matter content, clay type, and pH (Sawhney, 1986; Moore and Walker, 1991).

TABLE 5

Water solubility of chlorobiphenyl compounds and formulations (Hutzinger *et al.*, 1974; U.S. EPA, 1980)

Compound	% Chlorine by weight	Solubility (µg/L)	Compound	% Chlorine by weight	Solubility (µg/L)
Monochlorobiphenyls	18.8		2,3',4,4'-		58
2-		5 900	2,3',4',5-		41
3-		3 500	3,3',4,4'-		175
4-		1 190	Pentachlorobiphenyl	54.3	
Dichlorobiphenyls	31.8		2,2',3,4,5'-		22
2,4-		1 400	2,2',4,5,5'-		31
2,2'-		1 500	Hexachlorobiphenyl	58.9	
2,4'-		1 880	2,2',4,4',5,5'		8.8
4,4'-		80	Octachlorobiphenyl	66.0	
Trichlorobiphenyls	41.3		2,2',3,3',4,4',5,5'-		7.0
2,4,4'-		85	Decachlorobiphenyl	71.2	15
2',3,4-		78	Aroclor 1221	20.5-	200

				21.5	
Tetrachlorophenyls	48.6		Aroclor 1016	41	225-250
2,2',5,5'-		46	Aroclor 1242	42	240
2,2',3,3'-		34	Aroclor 1248	48	54
2,2',3,5'		170	Aroclor 1254	54	12
2,2',4,4'-		68	Aroclor 1260	60	2.7

Haque *et al.* (1974) noted that about 60% of Aroclor 1254 sorbed by Ottawa sand was lost by vaporisation in a 4-week period, while no significant loss occurred from Woodburn soil in the same period.

PCBs are chemically inert compounds; as a result they are resistant to chemical degradation reactions in the environment. However, photochemical breakdown of PCBs has been noted by several investigators. In studying the photolysis of hexachlorobiphenyl in methanol, Andersson *et al.* (1973) identified a series of *ortho*-methoxy PCBs and methoxy-substituted chlorodibenzofurans among the 80 compounds formed. The photolysis of 2,5-dichlorobiphenyl and 2,2',5,5'-tetrachlorobiphenyl in aqueous suspension yielded low quantities (~0.2%) of 2-chlorodibenzofuran; also, the higher molecular weight polychlorinated dibenzofurans could be the primary photochemical products, undergoing photo-reduction in water. In aqueous environments, photochemical degradation is limited to the uppermost layers of the water column.

The biphenyl molecule exhibits two absorption maxima; the main band at a wavelength of 202 nm and the k band at a wavelength of 242 nm. Chlorine substitution on the biphenyl molecule produces a shift on the k band; the shift is much greater for the *para* -substituted than for the *meta* -substituted biphenyls. For more highly substituted chlorinated biphenyls, both the main and k bands are shifted towards the visible region with increasing chlorination. This implies that higher chlorinated biphenyls photo-degrade faster than lower chlorinated biphenyls (Moore and Ramamoorthy, 1984).

3.2 Biological transformations

A considerable variety of biota are capable of metabolising lower chlorinated biphenyls (LCBPs) of up to 6 Cl atoms into polar metabolites. The metabolic degradation of PCB in animal tissues (*e.g.*, rats, birds, cows, and fish) is, therefore, characterised by the disappearance or reduction in the concentration of LCBPs. The breakdown of PCBs may yield hydroxylated products (with or without the arene oxide intermediary) which may differ with species (Moore and Ramamoorthy, 1984).

Isomerization and dechlorination reactions have been implicated in the metabolism of higher chlorinated biphenyls (HCBPs) (McKinney, 1976; Hutzinger *et al*., 1974). However, the identification of potentially toxic dibenzofuran structures in some metabolites, and lower ratios of PCBs to polychlorinated dibenzofurans (PCDFs) in liver tissue have caused concern regarding the metabolic formation and accumulation of PCDFs in the liver (Kuratsune *et al.*, 1976).

Micro-organisms are assumed to play a major role in the breakdown of environmental chemicals. Studies have shown that mono-, di-, and trichlorobiphenyls are significantly biodegraded and volatilised, whereas PCBs with 5 Cl atoms tend to sorb to suspended particulates and sediments, and resist biodegradation (Clark, 1979; Tulp *et al.*, 1978). Among commercial mixtures, Aroclors 1221 and 1232 showed significant biodegradation (Tabak *et al.*, 1981), whereas Aroclor 1248 and 1260 showed virtually no biologically induced breakdown at concentrations of 5 mg/L or 10 mg/L. The availability of C-H bonds in PCB determines the extent of hydroxylation and, in turn, biodegradation.

A faster degradation rate has been reported for commercial PCB mixtures than their single components. It has also been shown that HCBPs such as Aroclor 1254 exhibit enhanced degradation in the presence of LCBPs such as Aroclor 1221 (the process of co-metabolism) (Baxter *et al.*, 1975). Emulsification of PCB mixtures by sodium lignosulfonate greatly enhances the microbial degradation of PCB mixtures from Aroclor 1221 (lower chlorinated) to Aroclor 1254 (higher chlorinated). This is due to the increase in the surface area of the substrate, which is the limiting factor in the biodegradation process.

4. OCCURRENCE IN THE ENVIRONMENT

PCBs are synthetic chemical compounds which have been used widely in industrial applications. Because their hazardous nature has only recently been understood, PCBs have been routinely disposed of over the years without any precautions being taken. As a result, large volumes of PCBs have been introduced into the environment through open burning or incomplete incineration; by vaporisation from paints, coatings and plastics; by direct entry or leakage into sewers and streams; by dumping in non-secure landfill sites and municipal disposal facilities; and by other routes (e.g., ocean dumping). Despite regulations, some PCBs have been illegally dumped through ignorance, through negligence, or wilfully. Accidental spills and leaks, while of local significance, have been relatively minor sources of PCB contamination of the global

environment (CCREM, 1986). The following sections outline levels found in the environment.

4.1 Waters

PCBs are only slightly soluble, resulting in low dissolved levels in water. Using 1975 to 1983 water quality data obtained from the National Water Quality Data Bank (NAQUADAT), Strachan (1988) noted a wide regional disparity in the number of those samples in which quantifiable amounts of PCBs were measured (0 to 35% based on detection limits of 0.009 to 0.02 µg/L). In samples with positive results, the mean concentration of total PCBs was found to range from 0.015 (in the Atlantic region) to 0.04 µg/L (in the Provinces of Quebec and Ontario). None of the water samples from western Canada contained PCBs at quantifiable levels, despite their appearance in areas other than those reported in NAQUADAT.

In British Columbia, PCBs have not been detected in the vast majority of freshwater samples collected (Garrett, 1983; SEAM, 1989). However, detectable levels were observed in the McLeese Lake area (up to 0.2 µg/L in Cuisson Lake in December 1976), in Coldstream Creek (<0.2 to 5 µg/L in June 1979) in the Okanagan, and in Lime Creek (0.1 to 4.8 µg/L in March 1980) near the Amax Kitsault Mine. These high levels were attributed to accidental spillage or disposal of PCB-containing materials (Garrett, 1983).

PCB levels ranging from <0.0002 to 0.09 µg/L have been measured in marine and estuarine waters (Garrett, 1983). Maximum residues seldom exceed 0.002 µg/L and may fall below 0.0002 µg/L in offshore marine areas; however, unfiltered water samples containing particulate matter often bear much higher residues (Moore and Ramamoorthy, 1984). No data are available for marine waters off the British Columbia coast.

4.2 Sediments and soils

Unless indicated otherwise, the PCB concentrations in sediments in the following discussion are expressed on a dry weight basis.

Sediments are the primary sink for PCBs. The concentrations of PCBs in sediment depend upon the characteristics of the sediments and their proximity to the source. Frank *et al.* (1981) reported sediment residues of 0.010-0.020 µg/g in Lake Michigan near Chicago, Milwaukee, and Green Bay; by contrast the whole lake average was 0.0097 µg/g. In the vicinity of waste outfalls, residues may range from 2 to > 500 µg/g (Elder *et al.*, 1981). The mean and 95% confidence limits, respectively, for the Niagara

River sediment at Niagara-on-the-Lake were 0.580 and ±0.103 µg/g; somewhat lower levels were detected in the Ontario portion of the St. Lawrence River (mean concentration of 0.179 µg/g) and in the Atlantic Region (mean concentration of 0.247 µg/g) (Strachan, 1988).

In British Columbia, total PCB concentrations in freshwater sediments are generally below the detection limits (0.02 and 0.01 µg/g) (SEAM, 1989; Swain and Walton, 1988 and 1990). However, elevated levels of PCBs have been measured in sediments off certain industrial facilities; e.g., up to 1.0 µg/g was measured in sediments adjacent to the Belkin Paperboard paper recycling plant in Burnaby (Garrett, 1983).

Inshore marine areas of industrial zones are often highly contaminated. PCB levels in sediments of the Southern California Bight (Los Angeles) generally exceeded 1.0 µg/g and in some areas reached 10 µg/g (Young and Heeson, 1978). In British Columbia, PCB concentrations in marine sediments have been measured up to (a) 16.8 µg/g off the Bayshore Inn in Coal Harbour, (b) 17 µg/g in the vicinity of Burrard Yarrow Shipyards in Burrard Inlet, (c) 6.9 µg/g under the Granville Street Bridge in the False Creek area of Vancouver, and (d) 3.6 µg/g in Victoria's Inner Harbour (Garrett, 1983).

More recently, Goyette and Boyd (1989) found that mean PCB concentration in sediments from Vancouver Harbour (based on 1985/86 data) ranged from < 0.02 µg/g to 0.90 µg/g. The maximum concentration (0.90 µg/g) was recorded for the site at Burrard Yarrow on the north shore. Overall mean level for the Vancouver Inner Harbour (44 sites) was 0.17 ± 0.20 µg/g. Sediment PCB concentration in Port Moody Arm in 1985/86 (13 sites) averaged 0.06 µg/g (range, 0.02 - 0.18 µg/g), and in 1987 (33 sites) the average was 0.13 µg/g (range, 0.03 to 0.32 µg/g).

Offshore sediments such as those in the Mediterranean and Baltic seas, usually contain much lower residues of PCBs (<0.005 µg/g) (Basturk *et al.*, 1980).

PCBs do not generally occur in soil environments, except when spills, industrial releases, atmospheric transport, or application of sewage occur. Carey and Gowen (1976) reported that agricultural soils in the U.S. rarely contain detectable levels of PCBs; although PCBs were frequently detected in soils from urban areas, concentrations were <1.0 µg/g. In a study on Ontario soils, Weber *et al.* (1983) reported a range of 0.007 to 0.025 µg/g with a mean value of 0.013 µg/g PCBs for ten sites.

4.3 Biota

PCB concentrations in animal tissues in the following sections are on a wet weight basis unless reported otherwise.

4.3.1 Aquatic organisms

PCBs are readily sorbed from water by aquatic organisms; although variable, bio-concentration factors up to 300 000 have been reported for freshwater and marine organisms (Defoe *et al.*, 1978; Scura and Theilacker, 1977).

Johnston *et al.* (1975) reported PCB levels in fish collected in 1972/73 from the Fraser River. Although concentrations in most species were generally between 0.1 and 0.9 µg/g, certain coarse fish species showed levels in their muscle tissue which were equivalent to or in excess of the Health and Welfare guideline of 2 µg/g. One large-scale sucker contained over 3 µg/g and tissue levels in two northern squawfish approached 2 µg/g. Singleton (1983) reported measurable PCB levels in 11 of 253 fish (muscle tissue) samples collected from the Fraser River in 1980; detectable values (0.3 µg/g) ranged from 0.4 to 0.8 µg/g. Fish muscle tissue analyses carried out in 1988 by Swain and Walton (1989) had PCB levels between 0.003 and 0.26 µg/g (mean concentrations in the fish ranged from 0.02 to 0.06 µg/g),which were lower than those measured in the 1972/73 and 1980 surveys.

PCBs are extremely lipophilic; hence, maximum concentrations in fish are usually associated with fat deposits. The liver is another site of PCB accumulation. Residues are generally lowest in the muscle but are dependent on the fat content of the tissue. Removal of skin and adipose (fatty) tissue significantly decreases the level of PCBs in the trimmed fillet of fish (White *et al.*, 1985; Sanders and Haynes, 1988). Overall, concentrations in fat are a good tool for monitoring PCB contamination and should be measured in conjunction with edible muscle tissue determinations.

PCB levels in fish can vary with season. Three- to seven-fold seasonal differences in PCB residues were noted in perch and roach collected from a bay on the Baltic coast (Edgren *et al.*, 1981). Similarly, residues in whiting from the Medway Estuary (UK) peaked at 0.16 µg/g in April, declining to 0.01 µg/g by October (van den Broek, 1979). Such variability stems from the interplay of nutritional, reproductive, and activity cycles of the fish.

Sewage discharges have been shown to cause a significant increase in PCB levels in the marine environment. McDermott *et al.* (1975) showed that Dover sole collected near large sewage outfalls in the South California Bight contained much higher PCB concentrations than sole collected away from the outfalls. The median levels in the muscle tissue of the fish collected near three of the larger outfalls ranged from 0.7 to 1.9 µg/g in 1971-72 and 0.6 to 2 µg/g in 1974-75.

In British Columbia, PCB concentration was measured in rainbow trout caught from Okanagan Lake. The mean PCB concentration (on wet weight basis) in the muscle tissues of the fish was 1.57 µg/g (range, 0.37 to 4.06 µg/g) in 1974, 0.03 µg/g (range, 0.1 to 0.4 µg/g) in 1975, 0.50 µg/g (range, < 0.1 to 1.83 µg/g) in 1976, 0.24 µg/g (range, < 0.1 to 0.6 µg/g) in 1988, and 0.27 µg/g (range, 0.13 to 0.37 µg/g) in 1990 (Bryan and Jensen, 1991).

High concentrations of PCBs have been detected in the sediments from Coal Harbour. Crab collected from this area contained a mean concentration of 0.2 µg/g. The same species from False Creek contained PCB levels about 10-fold lower (Garrett, 1983).

Mussels collected under the Burrard and Granville bridges contained very low concentrations (0.014 - 0.017 µg/g) of PCBs. However, mussels collected in Alice Arm near the Kitsault Mine contained relatively high PCB levels (up to 1.7 µg/g). Limited data indicated that water samples collected in this area (Alice Arm) also had elevated PCB levels (0.24 to 4.8 µg PCBs/L) (Garrett, 1983).

4.3.2 Wildlife

Certain wildlife species, particularly fish-eating birds and aquatic mammals, accumulate high levels of PCBs.

Populations of double-crested cormorants (*Phalacrocorax auritus*) from Lake Huron are recovering rapidly, presumably due to a decrease in PCBs and other contaminant residues in their eggs. PCB levels between 10.3 and 25.6 µg/g (fresh weight) measured in the eggs of the cormorant population in 1972 were higher than in other Canadian cormorant populations (Weseloh *et al.*, 1983).

Average PCB concentrations of 14.9 and 21.4 µg/g (fresh weight) were measured in Great Blue Heron eggs from the Coquitlam and the University of British Columbia populations in 1977-78. The concentration in heron eggs collected in the Kootenay River area in 1969 ranged from 0.036 to 25.6 µg/g (fresh weight) with a mean value of 13 µg/g. These levels are higher than those that cause embryonic deformities and mortalities in poultry. However, no impacts on heron colonies in British Columbia traceable to PCBs have been observed (Garrett, 1983).

Residues of PCBs in birds are modified by numerous biotic factors including fat content, tissue specificity, sex, and developmental stage. Sexual differences in PCB content are pronounced due to the female's ability to shed a significant portion of the PCB burden into eggs. Also, PCB levels are reduced from egg to fledgling. Residues in the brain appear to be good indicators of PCB stress in birds. Concentrations greater

than 300 µg/g of PCB in the brain (fresh weight) were consistently recorded in dead or dying ring-billed gulls (*Larus delawarensis*) and ring-necked pheasants (*Phasianus colchicus*) poisoned by PCBs (Eisler, 1986).

5. BIOLOGIC AND TOXIC EFFECTS OF PCBS

The discussion in these sections is primarily based on the reviews by Parkinson and Safe (1987), Hansen (1987), and McFarland and Clarke (1989). The aspects considered in these sections include (a) molecular structure versus toxicity relationships for individual congeners, (b) toxicity of PCB mixtures, and (c) environmental significance of individual PCBs.

Because of their low solubility's in water, sublethal and chronic toxic effects of PCB contamination in the environment are more likely than acute and lethal effects. Growth, moulting, and reproduction are primary functions that have been shown to be affected by exposure of aquatic organisms to PCBs. The ability of organisms to eliminate foreign organic compounds or endogenous waste products may also be affected. In both fish and higher vertebrates, the metabolic activities such as steroid biosynthesis and the degradation and bio-transformation of foreign organic compounds are strongly influenced by terminal oxidase activities of the microsomal cytochrome P-450 systems. Induction of hepatic microsomal enzymes is one of the earliest and most sensitive indicators of PCB response. Some, although not all, PCB congeners are mixed-function oxidase (MFO) inducers in fish, mammals, and birds, and to a lesser extent in aquatic invertebrates.

5.1 Mixed-function oxidase induction

Liver endoplasmic reticulum (microsomes) contains a family of 12 cytochrome P-450 isozymes (*i.e.*, group of enzymes which are chemically distinct but functionally alike; also referred to as mixed-function oxidase or MFO systems). The function of these isozymes or hemoproteins is to catalyse the bio-transformation of lipophilic xenobiotics (*e.g.*, by hydroxylating, epoxidating, dealkylating or oxygenating, and in some cases by dehalogenating and reducing) to metabolites that are more readily eliminated from the body. However, bio-transformation of xenobiotics by cytochrome P-450 is not always a beneficial process; there are many cases where the metabolites are more toxic or biologically active than the parent compound.

Several cytochrome P-450 isozymes are highly induced by various xenobiotics (*e.g.*, phenobarbital (PB), 3-methylcholanthrene (3-MC), etc.). Frequently, the cytochrome P-

450 systems are characterised (*e.g.*, PB-type, 3-MC-type, etc.) by reference to model chemicals that stimulate (induce) or inhibit the production of these enzymes. The group of microsomal cytochrome P-450-dependent enzyme systems that catalyse oxidative bio-transformations of aromatic ring-containing compounds falls in the category of mixed function oxidases (MFOs). The MFOs that are induced by the industrial mixtures of PCBs in vertebrates are characterised as phenobarbital-type (PB-type), 3-methylcholanthrene-type (3-MC-type), or possessing catalysing properties of both (mixed-type). Both PB-type and 3-MC-inducible enzymes have the potential for producing toxicity through bio-activation. However, the potential for contributing to toxicity is greatest with the pure 3-MC-type and mixed-type inducers while PB-type inducers are considered potentially more toxic than weak inducers and non-inducers. The MFOs of fish and apparently of invertebrates, are qualitatively similar to the 3-MC-inducible MFOs of vertebrates. The PB-type induction has been reported in fish (*e.g.*, mummichog, rainbow trout, and carp) only in a few investigations. The enzymes, aryl hydrocarbon hydroxylase (AHH) and ethoxyresorufin O-deethylase (EROD) which are examples of MFO systems, are characteristic in fish as well as in 3-MC-induced mammals (McFarland and Clarke, 1989).

5.2 Molecular structure and PCB toxicity

The toxic reactions of PCBs have been studied with regard to their receptor binding avidities and several receptor-mediated responses, including body weight loss, thymic atrophy, and the induction of the cytochrome P-450-dependent mono-oxygenases, AHH, and EROD. The specificity for MFO induction (and correlatively, for potential toxicity) of the individual PCB isomers and congeners differs greatly and can be related to how closely the PCB isomers approach the molecular spatial configuration and distribution of forces (i.e., are isosteres) of 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD). The dioxin 2,3,7,8-TCDD is one of the most potent synthetic environmental toxicants, and is regarded as a standard for comparison for other organic toxicants that are more or less iso-steric. The cytosolic receptor that binds 2,3,7,8-TCDD is a soluble protein produced by the Ah (aryl hydrocarbon) gene locus. Translocation of the inducer-receptor complex to the nuclear Ah locus is considered to initiate the synthesis of AHH, EROD, and related enzymes that may be involved in either bio-transformation, conjugation and removal, or bio-activation of certain lipophilic foreign compounds.

Quantitative structure-activity relationship (QSAR) studies have shown that PCB congeners with chlorine substitution at both *para* (4 and 4') and two or more *meta* (3,3',5, and 5') positions (see Figure 1) are very potent mimics of 2,3,7,8-TCDD both in cytochrome P-450 induction and toxic effects, which are mediated through initial binding to the Ah (aryl hydrocarbon) receptor. These laterally-substituted congeners (a total of 6 congeners; *i.e.*, PCB # 15, 37, 77, 81, 126, and 169- see Table 1) are true

coplanar PCBs as they lack the bulky *ortho*-chloro substituent that restricts free rotation about the phenyl-phenyl bond. Three of the so-called coplanar PCBs, namely 3,3',4,4'-tetra (PCB 77), 3,3',4,4',5-penta (PCB 126), and 3,3',4,4',5,5'-hexachlorobiphenyl (PCB 169) are the most toxic PCBs known. With the exception of PCB # 81, the non-*ortho*-coplanar congeners are potent inducers of AHH and EROD activities in *in vitro* rat hepatoma cell preparations.

The addition of a single *ortho* -chloro substituent to the coplanar PCBs decreases the co-planarity between the two phenyl rings and hence the toxicity of the chlorinated derivatives of coplanar PCBs. This group of mono-*ortho* coplanar PCBs includes congeners 105, 114, 118, 123, 156, 157, 167, and 189. The loss of toxic potency is accompanied by a qualitative shift in cytochrome P-450 induction; all of the mono-*ortho* -substituted PCBs are mixed-type inducers (PB-type + 3-MC-type) in contrast to the parent coplanar PCBs, which are predominantly 3-MC-type inducers (except PCB # 81 which is a mixed-type inducer). Among the mono-*ortho* -chloro substituted PCBs, the group having *ortho*-chloro adjacent to a *meta*-chloro substituent (as in 2,3,4- and 2,3,4,5-substituted PCBs) is more toxic than the group with *ortho*-chloro adjacent to a *meta*-hydrogen (as in the 2,4- and 2,4,5-substituted PCBs).

The trend toward phenobarbital (PB)-type characteristics and away from 3-methylcholanthrene (MC)-type characteristics continues as two or more *ortho* -chloro substituents are added to the coplanar PCBs. The di-*ortho* coplanar PCB congeners (i. e., PCB 128, 137, 138, 153, 158, 166, 168, 170, 180, 190, 191, 194, and 205) are less potent than their non-*ortho* coplanar and mono-*ortho* coplanar congeners.

Although there have been few systematic studies, toxicities other than those associated with the Ah locus do not appear to follow structure/activity patterns. Toxic responses unrelated to Ah locus effects and less intensive than those for Aroclor 1254 have been reported for PCB congeners 4, 28, 31, 49, 52, 84, 95, 110, 136, and 153 (Hansen, 1987).

The presence of the most toxic PCB congeners #77, #126, and #169 has been reported in various commercial PCB preparations and tissue samples from a wide range of terrestrial and aquatic (freshwater and marine) organisms, including humans (Tanabe *et al.*,1987; Niimi and Oliver, 1989; Kannan *et al.*, 1988). However, the concentration of these congeners in water and animals was low, ranging from the non-detectable level (<0.01 ng/L) to (a) 1.0 ng/L in water samples from the Hudson River, N.Y. (Bush *et al.*, 1985), and (b) 0.007 µg/g (wet weight) in the muscle tissue of lake trout (*Salvelinus namaycush* ; Niimi and Oliver, 1989) or 0.055 µg/g in the fat tissue (wet weight) of killer whale (Tanabe *et al.*,1989).

Several receptor-mediated responses, as noted above, have been used to study the structure/activity relationships. Table 6 shows the ED_{50} and EC_{50} values, respectively, for *in vivo* inhibition of body weight gain in the immature male Wistar Rat and *in vitro* induction of AHH and EROD enzyme activities in the rat hepatoma cell cultures (Sawyer and Safe, 1982; Safe, 1987). Both the *in vivo* mammalian toxicity response and the *in vitro* induction of the enzyme systems were positively correlated. The results in Table 6 show that 3,3',4,4',5 pentachlorobiphenyl (PCB 126) is the most toxic congener with a potential for toxicity (expressed as TEF or toxic equivalent factor) approaching that of the dioxin, 2,3,7,8-TCDD. Toxic equivalent factors have been used to assess the toxic potential of (a) coplanar PCBs in the environment relative to dioxins and furans (Niimi and Oliver, 1989; Olafsson and Bryan, 1987; Olafsson *et al.*, 1988) and (b) PCB formulations (Kannan *et al.*, 1988). At present, however, the AHH and/or EROD induction data cannot be used for setting water quality guidelines for PCBs. The reasons being:

(i) the data are specific to Wistar rats and are scant on other organisms (e.g., aquatic life),
(ii) the concentrations (in µg/L) and doses (in µg/g body weight) used in these experiments are much higher than those to which organisms are subjected to in the terrestrial and aquatic environments,
(iii) the difficulty in translating AHH or EROD response to a no adverse effect concentration, which is usually obtained from chronic effects or bioaccumulation information. It has been suggested that the increase in the hepatic microsomal AHH activity of fish may be used as an indicator of environmental contamination; but, should a concentration of PCBs in water, however small, be considered toxic (or undesirable) if it induces the enzymatic response in the organism? The linkage between chronic effects and AHH induction is currently unknown,
(iv) lack of relevant information on PCB congeners other than those shown in Table 6. Should other congeners which are structurally unlike coplanar PCBs or 2,3,7,8-TCDD

TABLE 6

A summary of *in vivo* biological and toxic effects and *in vitro* AHH and EROD induction potency of a TCDD and several PCBs.
(From Sawyer and Safe, 1982 and 1985; Niimi and Oliver, 1989) *

Test chemical	*in vivo* ED50							
	Inhibition of body weight gain in immature male Wistar rats					AHH induction		
	µmol/kg	µg/g	molar	µg/L	molar	µg/L		

			conc.		conc.			
2,3,7,8-TCDD	0.05	0.016	9.60×10^{-11}	0.031	8.02×10^{-11}	0.026	1.0	1.0
PCB #126	3.3	1.1	2.40×10^{-10}	0.078	2.48×10^{-10}	0.081	0.32	0.16 - 0.4
PCB #169	15	5.4	6.01×10^{-8}	21.7	2.41×10^{-8}	8.7	33×10^{-4}	$12 \times - 16 \times 10^{-4}$
PCB #77			3.51×10^{-8}	10.2	8.85×10^{-8}	25.8	9.1×10^{-4}	$1 \times - 27 \times 10^{-4}$
PCB #105	750	245	8.75×10^{-8}	28.6	1.20×10^{-7}	39.2	6.7×10^{-4}	$8 \times - 11 \times 10^{-4}$
PCB #123	370	121	9.73×10^{-7}	318	5.65×10^{-7}	184	1.4×10^{-4}	$2 \times - 9.9 \times 10^{-5}$
PCB #74								4.8×10^{-6}
PCB #153								1×10^{-5}
PCB #156	180	65	2.07×10^{-6}	747	8.96×10^{-7}	323	8.9×10^{-5}	$3 \times - 4.6 \times 10^{-5}$
PCB #114	180	59	3.91×10^{-6}	1276	1.11×10^{-6}	362	7.2×10^{-5}	$2.5 \times - 7 \times 10^{-5}$
PCB #157	220	79	7.11×10^{-7}	256	1.26×10^{-6}	455	6.4×10^{-5}	$5 \times - 140 \times 10^{-6}$
PCB #81			1.11×10^{-5}	3241	1.92×10^{-6}	561	4.2×10^{-5}	8.6×10^{-6}
PCB #189			1.13×10^{-5}	4467	7.81×10^{-6}	3087	1.0×10^{-5}	8.5×10^{-6}
PCB #118			1.15×10^{-5}	3754	8.86×10^{-6}	2892	9.1×10^{-6}	$6 \times - 8.3 \times 10^{-6}$
PCB #167			1.33×10^{-5}	4800	9.00×10^{-6}	3248	8.9×10^{-6}	7.2×10^{-6}
Aroclor 1254			3.79×10^{-6}	1239	4.61×10^{-6}	1507	1.7×10^{-5}	$1.3 \times - 3 \times 10^{-5}$
Aroclor 1260								8×10^{-6}

* EC_{50} (or ED_{50}) represents half the concentration (or dose) of the toxic congener required to produce the maximum effect; TEF-AHH is the toxic equivalent factor based on AHH induction and is the ratio of the EC_{50} value for the 2,3,7,8-TCDD divided by that of the PCB congener. TEF-EROD is calculated in a similar manner.
+ The range in AHH-based TEFs indicate that several sources were used to derive these factors.

that do not induce AHH or EROD activities, be considered non-toxic? The discussion in section 7 on Aquatic Life suggests they should not be considered non-toxic, and

(v) lack of relevant information on the toxicity of isomers and individual congeners to organisms using accepted chronic and acute tests. This type of information along with toxic equivalent factors could be useful in the development of criteria for those congeners which behave alike (*i.e.*, showing structure/activity relationships).

5.3 Mixtures of PCB isomers and congeners

Industrial PCB mixtures contain several mono- and di-*ortho* derivatives of coplanar PCBs. The abilities of these derivatives to induce several forms of rat liver microsomal cytochrome P-450 and to produce a toxic syndrome similar to that caused by TCDD intoxication suggests that the mono- and di-*ortho* derivatives of the coplanar PCBs contribute substantially to the biologic and toxic properties of industrial PCB mixtures. The presence of relatively more toxic coplanar PCBs such as # 77, 126, and 169 also has been reported in various commercial PCB preparations and tissue samples from a wide range of organisms including marine mammals and humans (Tanabe *et al.*,1987; Niimi and Oliver, 1989; Kannan *et al.*, 1988).

The environment is contaminated with mixtures of PCB congeners. The variation in the biologic and toxic properties of PCB congeners necessitates a careful assessment of the impact of PCBs in mixtures on the environment. The composition of PCB mixtures changes dramatically in the environment, particularly in the food chain. For instance, it has been shown that the mixture of PCBs secreted in human milk differs from the PCBs in the industrial mixtures that contaminate the environment. The mixture of PCBs in breast milk is considerably enriched in many of the mono- and di-*ortho* substituted analogues of coplanar PCBs. The preferential metabolism and subsequent excretion of lower chlorinated PCBs and the poor absorption of higher chlorinated (those having seven to ten chlorines) PCBs likely accounts for this relative enrichment. As a result of this enrichment with mono- and di-*ortho* substituted PCBs, it has been shown that a mixture of PCBs secreted in human breast milk is more biologically active than the industrial PCB mixtures. Obviously, a knowledge of isomeric and con-generic composition in various compartments of the environment would render a more meaningful assessment of the potential risk posed by mixtures of PCBs than total PCB analyses in a single compartment.

There is another important question that must be considered in addressing the toxicity of the mixtures of PCBs; that is, how does one PCB isomer or congener influence the biologic or toxic reaction of another? Two potential mechanisms of synergistic interaction between individual PCBs have been identified. The first involves a sparing effect, whereby a non-toxic PCB congener occupies binding sites (non-specific binding sites, receptors, proteins and/or enzymes), thereby decreasing the removal of a toxic PCB congener. A second possibility involves an interaction at the level of the cytosolic

Ah receptor. Both synergistic and antagonistic effects of PCBs in mixtures have been reported in the literature; more work is, however, needed in this direction. For instance, more than additive response (measured as induction of hepatic microsomal benzo(a)pyrene hydroxylase and EROD activities) was noted in immature male Wistar rats pre-treated with 2,4,5,2',4',5'-hexachlorobiphenyl and followed by an exposure to 3,4,4,3',4'-penta or 3,4,5,3',4',5'-hexachlorobiphenyl (Parkinson and Safe, 1987). On the other hand, less than additive response (based on observed AHH and EROD activities for individual congeners) was noted in Wistar rat hepatoma cells exposed to several environmentally significant reconstituted mixtures of PCBs (Sawyer and Safe, 1985).

Recently, assessments of the effects of PCBs relative to furans and dioxins on aquatic environments have been carried out by several investigators based on AHH or EROD enzyme-inducing capabilities of various toxic congeners (Niimi and Oliver, 1989; Bryan *et al.*, 1987; Kannan *et al.*, 1988). This procedure requires that the AHH-inducing capability of all congeners be known. However, not all PCB congeners are AHH or EROD inducers, and non-inducer congeners seem to be very toxic as well (Bridgham, 1988). Also, some results of the recent investigations have indicated that the PCB mixture Aroclor 1254 is a dioxin antagonist in rat hepatoma H-4-II E cells, *in vitro,* and in C57BL/6J mice, *in vivo*, if administered at non-effective (below toxic threshold) doses (McFarland and Clarke, 1989).

5.4 Carcinogenic and mutagenic effects

Evidence relating to the carcinogenicity of PCBs can be arbitrarily considered in three main categories: (i) influence on mutagenicity and initiation of carcinogenesis, (ii) influence on promotion and progression of carcinogenesis, and (iii) epidemiological evidence for carcinogenesis in naturally exposed populations. The majority of evidence comes from experimental studies, and its value depends upon the quality of various studies and the conceptual framework within which the studies have been designed and interpreted.

A number of rats exposed to dietary levels of 25 to 100 µg/g PCBs (e.g., Aroclor 1254 and 1260) were found to develop various types of cancer (Kimbrough *et al.*, 1975; NCI, 1977; Weltman and Norback, 1981; Kimbrough, 1985). The US Food and Drug Administration (FDA) has used this information to determine the risk to humans from consuming fish contaminated with PCBs. However, the use of such information for risk analysis has been criticised for several reasons which include: (a) the FDA methodology employed high doses (25 to 100 µg/g in diet) in experiments on animals and extrapolated the observed rates of certain types of cancer at these elevated doses to the low doses found in human diets, and (b) the FDA assumed that humans and test

animals are equally sensitive to PCB ingestion when measured on a µg/g in diet basis. Extrapolation on an equivalent consumption per unit of body weight is preferred and results in much lower health risks (Maxim and Harrington, 1984).

Kimbrough (1985) reviewed laboratory and human studies on PCBs and related compounds. It was noted that three attempts were made to determine whether exposure to PCBs (in capacitor and refinery plants) caused an increased incidence of cancer. In all cases, the results were inconclusive for reasons of (a) small sample size, (b) interference from other chemicals, and (c) short-term exposures. It was concluded that, in humans, adequate studies were not conducted to judge if the long-term exposure to PCBs was associated with cancer and reproductive impairment (in exposed females).

In reviewing the literature on carcinogenicity of PCBs, Hayes (1987) reached the following conclusions:

PCBs appear to be, at the worst, very weak genotoxicants or initiators of carcinogenesis in various systems. Their well established activity at moderately high levels as promoters of hepatocarcinogenesis (or liver cancer) in rodents should not necessarily be accepted as clear predictive evidence for a similar effect in humans. Many xenobiotics with no known hepatocarcinogenic effects in human are, like PCBs, strong promoters of liver tumour growth in rodents that appear inherently highly susceptible to this response. Evidence that PCBs enhance genotoxicity and mutagenicity of many other xenobiotics in various *in vitro* test systems is in direct contrast to their protective role against carcinogenicity of many genotoxic carcinogens *in vivo*.. Furthermore, PCBs should not be universally regarded as liver tumour promoters because they strongly prevent the promoting activity of other environmental carcinogens such as dimethylnitrosamine, diethylnitrosamine, azo dyes, 2-acetylaminofluorene, and aflatoxin B1.

Collectively, the evidence suggests that PCBs may potentially be carcinogenic under some specific conditions. However, under natural exposure circumstances, PCBs are perhaps more likely to prevent carcinogenesis than enhance it. Accordingly, the risk estimates based on the worst-case analysis of the potentially carcinogenic effects are likely to be substantial overestimates of the real risk.

While PCBs are clearly toxic and hazardous to humans exposed accidentally to high levels, there is a lack of clear epidemiological evidence for carcinogenicity of environmental PCBs in humans and animals. However, the number of thorough epidemiological studies conducted is small. More discriminating studies of PCBs and

other potential carcinogenic risk factors are necessary before it would be reasonable to conclude that PCBs are not human carcinogens.

5.5 Environmental significance of PCB congeners

McFarland and Clarke (1989) selected 36 PCB congeners which were considered to be of environmental significance based on three factors: (a) potential for toxicity (inferred by MFO induction; PB-type inducers were considered less toxic than pure 3-MC-type and mixed-type inducers, but more toxic than weak and non-inducers), (b) frequency of occurrence in environmental samples, and (c) relative abundance in animal tissue (determined from a PCB congener database developed from information reported in the scientific literature) (Table 7). Group 1 contains the highest priority congeners which are most likely to contribute to adverse biological effects. The subgroup 1A contains the three most potent congeners (pure 3-MC-type inducers: PCB #77, #126, and #169); although these congeners have been reported rarely in the environment, they have been identified as components of technical formulations. Subgroup 1B congeners (PCB #105, #118, #128, #138, #156, and #170) are mixed-type inducers that (except PCB #105) have been reported frequently in the environment. The seven congeners in Group 2 are PB-type inducers with high relative abundance in avian and mammalian tissue, but they are found to a lesser extent (as little as 1.5%) in fish and invertebrates. Except for congeners #77 (a tetrachlorobiphenyl) and #194 (an octachlorobiphenyl), all of the congeners in Group 1 and 2 are member of the penta-, hexa-, and heptachlorobiphenyl isomer groups.

TABLE 7
PCB congeners of highest concern as environmental contaminants
(From McFarland and Clarke, 1989)

Highest Concern IUPAC Number Lowest Concern			
Group 1A	**Group 2**	**Group 3**	**Group 4**
77*+	87*+	18*	37+
126+	99+	44*+	81
169+	101*+	49*+	114*+
Group 1B	153*+	52*+	119
105*+	180*+	70+	123
118*+	183*+	74+	157
128*+	194*	151*	158+
138*+		177	167

156*+		187*	168
170*+		201*	189*+

* Congeners included in Canadian Standard CLB-1, developed under the Marine Analytical Standards Program by the Atlantic Research Laboratory, NRC, Halifax, Nova Scotia. The remaining congeners making up CLB-1 are nos. 15, 31, 40, 54, 60, 86, 103, 121, 129, 137, 141, 143, 154, 159, 171, 173, 182, 185, 191, 195, 196, 200, 202, 203, 205, 206, 207, 208, and 209.
+ Congeners suggested for inclusion in human foodstuff and tissue analyses by Jones (1988). Other congeners listed are 8, 28, 60, 66, 82, 166, 179, and 187.

Group 3 congeners are weak or non-inducers but occur frequently, particularly in fish and invertebrate tissues. This group spans the isomer groups from tri- through octachlorobiphenyl. Group 4 congeners are mixed-type inducers. Although scarce in the environment, they are considered to be of possible concern because of their potential toxicity. McFarland and Clarke suggested that toxicologically relevant evaluations of PCB-contaminated environmental materials can be better accomplished by analysing samples for specific congeners in the four groups as compared to total PCB or as Aroclor equivalent. The congeners in Groups 1 and 2 were considered to be the most environmentally threatening.

In studying PCB congener levels in the Lake Ontario ecosystem, Oliver and Niimi (1988) observed that twelve PCB congeners (#153, #101, #84, #138, #110, #180 #, #87, #97, #149, #187, #182, and #105) constituted over half the PCBs in fish. These congeners were suggested as a focus for research on health effects. The authors also concluded that congener-specific PCB analysis is not required for all applications, but that it is vital for pathway research.

Working with a data set which was largely different from that of McFarland and Clarke (1989), Jones (1988) derived a list of 32 congeners which were considered to be environmentally important. With the exception of PCB 194, the list included all of the congeners assigned to Groups 1 and 2 in Table 7. Jones' list also included 10 of the 20 congeners assigned to Groups 3 and 4 in Table 7.

5.6 Congener-specific analysis of PCBs

The discussion in this section is based on material presented in McFarland and Clarke (1989) and Safe *et al.* (1987).

In general, the analysis of PCBs in environmental and human samples is dependent on gas chromatograph (GC) peak- or pattern-matching of commercial mixtures with the

specific PCB extract under investigation. However, the composition of PCB extracts from diverse environmental matrices can vary widely and such extracts do not resemble any commercial mixture. Therefore, the results obtained from the pattern-matching technique are at best a semi-quantitative estimate of the PCBs present in a sample, and yield very little indication of the relative concentrations of the individual congeners.

Isomeric- or congener-specific PCB analyses have been performed using the high-resolution capillary gas chromatography technique. The individual peaks are identified by using synthetic standards and/or by retention index addition methods (Ballschmiter and Zell, 1980). The latter technique relied on the relative retention times (RRTs) that have been determined for the limited number of available synthetic PCB standards. However, the accurate quantitation of the individual PCB components in a mixture can only be accomplished by comparing the observed relative retention time (RRT) and peak height (or area) data for a PCB-containing extract and the RRT and molar (or weight) response factor of all the PCB standards.

Recently, the unambiguous synthesis and chromatographic properties of the 209 PCBs have been reported by Mullin *et al*.(1985). These workers successfully separated 187 of the 209 PCB congeners by capillary gas chromatography. Eleven pairs (PCB 94/61, 70/76, 95/80, 60/56, 145/81, 144/135, 140/139, 133/122, 163/160, 202/171, and 203/196) exhibited similar retention times using a SE-54 coated glass capillary column. Some of these pairs, however, can be resolved chromatographically despite their comparable RRT values. Also, some of these compounds were not present in commercial PCBs.

An analytical instrument calibration standard for PCB congener-specific analysis by capillary column gas chromatography has been developed under the Marine Analytical Standards Program by the Atlantic Research Laboratory, National Research Council of Canada, Halifax, Nova Scotia (McFarland and Clarke, 1989). The Canadian Standard Mixture, CLB-1, contains 51 congeners which include 13 of 16 congeners in Groups 1 and 2, and 9 of 20 congeners in Groups 3 and 4 (Table 7). Not included are PCBs 126 and 169, two of the three most toxic congeners in Group 1A.

6. DRINKING WATER SOURCES

The criteria for PCBs in drinking water supplies from various jurisdictions are shown in Table 8. However, drinking water criteria for PCBs were not set in this document for two reasons. The primary reason behind this inaction was the fact that PCBs are under

review for possible addition to the Guidelines for Canadian Drinking Water Quality, a document which is published and periodically updated by Health and Welfare Canada (1989). It was also noted that raw drinking water supplies are not a significant source of PCB body burden in humans. The results in Table 9 lend support to this argument.

The contribution of various sources to the PCB body burden of an adult male was estimated, based on recommended concentrations and objectives for the contaminants in food, air, and in ambient waters for the protection of aquatic life and its consumers (Table 9). The results show that food and air together comprise most of the tolerable daily intake (TDI) of 60 µg PCBs (for an average adult weighing 60 kg) recommended by Health and Welfare Canada (Grant, 1983). A raw drinking water supply, in which the maximum allowable PCB concentration may be determined by the most sensitive water use (*e.g.*, aquatic life), is an insignificant source of PCBs. Assuming that other factors (*i.e.*, intake from food and air) remain the same, the advisory concentration of 0.5 µg total PCBs/L by U.S. EPA (1988) may be acceptable for drinking water (*i.e.*, using this concentration, the maximum allowable body burden for PCBs was calculated to be 59.9 µg/person-d, which is slightly less than the tolerable daily intake).

The results (Table 9) also suggest that fish is the main source of PCB body burden in humans. The section of the population whose diet contains significantly higher amounts of fish and shellfish (above the average daily consumption shown in Table 9) will be especially vulnerable.

TABLE 8

PCB CRITERIA FOR DRINKING WATER SUPPLIES

Criteria Statements	Criteria Values	Jurisdiction	Date	Reference
	(µg/L)			
Desirable objective; Recommended maximum concentration	undetectable 3.0	Saskatchewan	1985	Chan, 1985
Recommended maximum concentration	3.0	Ontario	1983	OME, 1983
Recommended standard	0.1	Quebec	1984	Trépanier, 1984
Recommended maximum concentration	3.0	Nova Scotia	1985	Environment Canada, 1986

1-d EPA Suggested No Adverse Response Level (SNARL) for child; 10-d EPA-SNARL for a child	125 12.5	EPA	1981	US EPA, 1981
1-d NAS-SNARL for an adult; 7-d NAS-SNARL for an adult;	350 50	NAS	1981	US EPA, 1981
Advisory drinking water criteria: -at cancer risk level of 10-4 -at cancer risk level of 10-6	0.5 0.005	EPA	1988	US EPA, 1988

TABLE 9

Theoretical maximum allowable daily body burden of PCBs from various sources

FOOD				
Category	Daily intake# (g/person-d)	Average fat content (%)	Recommended concentration (µg PCBs/g)a	PCBs intake (µg/person-d)
meat	194	15	0.2 (fat basis)	5.8
eggs	44	15	0.1 (whole less shell)	0.44
poultry	27	15	0.5 (fat basis)	2.0
fish	20	-	2.0 (edible portion)	40.0
dairy products ##	420	5	0.2 (fat basis)	4.2
Total (Food)				52.4
AIR				
Category	Daily intake (m^3/person-d)		Recommended concentration (µg/m^3) b	PCBs intake (µg/person-d)
Adult engaged in light physical activity	15		0.45 (0.5-h average)	6.75
DRINKING WATER				
Category	Daily intake (L/person-d)		Recommended concentration (µg/L) c	PCBs intake (µg/person-d)

Adult	1.5		0.001	0.0015

a Health and Welfare guidelines (Grant, 1983);
b Ontario ambient air quality criteria (OME, 1984a);
c CCREM (1987) guideline for aquatic life;
Nutrition Canada (1975);
~ 5.5 % cheese (22-39 year old adults; Nutrition Canada, 1975)

7. AQUATIC LIFE

7.1 Effects

7.1.1 Freshwater environment

(a) Lethal and acute toxicity to aquatic animals

The lethal toxicity (2- to 30-d LC_{50}) of PCBs to freshwater organisms varies with several factors which include the PCB formulation, the organism species and stage of development, and the test conditions employed (*e.g.*, length of exposure, static versus flow-through tests, etc.) (Table 10; Figures 2 through 6). Aroclors containing 42 to 54% chlorine appear to be the most toxic formulations of PCBs; for instance, a static test with *Daphnia magna* exposed to Aroclors A-1221, A-1242, A-1248, A-1254, A-1260, A-1262, and A-1268 displayed 504-h LC50s of 180, 67, 25, 31, 36, 43, and 253 µg/L, respectively (Nebeker and Puglisi, 1974; Table 10). Similar observations can be made from tests with rainbow trout (*Oncorhynchus mykiss* formerly classified as *Salmo gairdneri*) exposed to several PCB formulations in a continuous flow-through system (Johnson and Finley, 1980; Mayer *et al.*, 1977; Table 10).

The toxicity of PCBs is more severe when aquatic organisms are exposed to them in a continuous flow-through system rather than in a static system; also, longer exposure periods yield lower LC_{50}s (Nebeker and Puglisi, 1974; Stalling and Mayer, 1972; Table 10). The lowest concentrations causing lethal toxic effects were: 23 µg/L (720-h LC_{50}, Aroclor 1016), 5 µg/L (240-h LC_{50}, Aroclor 1242), 2.6 µg/L (336-h LC_{50}, Aroclor 1248), 0.45 µg/L (504-h LC_{50}, Aroclor 1254), and 3.3 µg/L (720-h LC_{50}, Aroclor 1260). In general, the data in Table 10 and Figure 2 through 6 appear to suggest that invertebrates and fish are equally sensitive to PCBs.

(b) Sublethal and chronic toxicity to aquatic organisms

The information obtained from the literature on sublethal and chronic effects of PCBs to freshwater aquatic organisms is tabulated in Tables 11 and 12. The data were also plotted in Figures 7 and 8. The lowest observed adverse effect level for algae was 1.0 µg/L Aroclor 1242, but, in general, aquatic animals appear to be more sensitive to PCBs than algae and plants. The minimum concentration of commercial PCBs (e.g., Aroclors A-1242 and A-1254) causing chronic effects (e.g., inhibitory effects on the ATPase activity of brain and kidney tissues of fathead minnow) was recorded to be 0.31 µg/L by Koch *et al.*, 1972 and Cutkomp *et al.*, 1972. These investigators also found that the effect on ATPase activity of the fish tissues was not consistent with the toxicant concentration; for instance, in several instances the enzyme activity at a higher exposure level of 8.3 µg/L of the toxicant was comparable to the control. In a 30-d chronic exposure to 0.1 µg/L Aroclor 1248, DeFoe *et al.* (1978) found that both the first- (F1) and the second-generation (F2) fathead minnow (*P. promelas*) fry and larvae survival, weight, or length were not affected.

TABLE 10

48- to 720-h LC50s for freshwater aquatic animals exposed to commercial PCBs.

Organisms	PCB	LC_{50}	Duration	System	Reference
		(µg/L)	(hours)	*	
Stonefly (*P. badia*)	A-1016	424-878	96	S	Johnson and Finley, 1980
Hydra (*H. oligactis*)	A-1016	5 000	72	U	Adams & Haileselassie, 1984
Rainbow trout (*O. mykiss*)	A-1016	114-159	96	S	Johnson & Finley, 1980
Blue gill (*L. macrochirus*)	A-1016	390-540	96	CF	Johnson & Finley, 1980
Channel catfish (*I. punctatus*)	A-1016	340-560	96	S	Johnson & Finley, 1980
Atlantic salmon (*S. salar*)	A-1016	113-159	96	CF	Johnson & Finley, 1980
Brook trout (*S. fontinalis*)	A-1016	> 800	96	CF	Johnson & Finley, 1980
Brown trout (*S. trutta*)	A-1016	109-175	96	CF	Johnson & Finley, 1980
Lake trout (*S.*	A-	386-1154	96	S	Johnson & Finley,

namaycush)	1016				1980
Longnose sucker (*C. catostomus*)	A-1016	222-490	96	CF	Johnson & Finley, 1980
White sucker (*C. commersoni*)	A-1016	325-582	96	CF	Johnson & Finley, 1980
Yellow perch (*P. flavescens*)	A-1016	153-376	96	S	Johnson & Finley, 1980
Fathead minnow (*P. promelas*)	A-1016	23	720	U	Veith, 1976
Water flea (*D. magna*)	A-1221	180	504	S	Nebeker & Puglisi, 1974
Cutthroat trout (*Salmo clarki*)	A-1221	957-1430	96	S	Johnson & Finley, 1980
Cutthroat trout (*Salmo clarki*)	A-1232	1720-3080	96	S	Johnson & Finley, 1980
Water flea (*D. magna*)	A-1242	67	504	S	Nebeker & Puglisi, 1974
Scud (*G. pseudolimnaeus*)	A-1242	10	96	CF	Stalling & Mayer, 1972
Stalling & Mayer, 1972	A-1242	5	240	CF	Stalling & Mayer, 1972
Crayfish (*O. nais*)	A-1242	30	168	CF	Stalling & Mayer, 1972
Scud (*G. pseudolimnaeus*)	A-1242	73	96	CF	Nebeker & Puglisi, 1974
Dragonfly (*Macromia sp.*)	A-1242	800	168	S	Johnson & Finley, 1980
Scud (*G. pseudolimnaeus*)	A-1242	10	96	CF	Johnson & Finley, 1980
Crayfish (*O. nais*)	A-1242	30	168	S	Johnson & Finley, 1980
Damselfly (*I. verticalis*)	A-	400	96	CF, M	Mayer *et al.*, 1977

	1242				
Blue gill (*L. macrochirus*)	A-1242	150	120	CF	Stalling & Mayer, 1972
Blue gill (*L. macrochirus*)	A-1242	72	240	CF	Stalling & Mayer, 1972
Blue gill (*L. macrochirus*)	A-1242	54	360	CF	Stalling & Mayer, 1972
Blue gill (*L. macrochirus*)	A-1242	125	120	CF	Johnson and Finley, 1980
Blue gill (*L. macrochirus*)	A-1242	84	720	CF	Mayer *et al.*, 1977
Channel catfish (*I. punctatus*)	A-1242	174	240	CF	Stalling & Mayer, 1972
Channel catfish (*I. punctatus*)	A-1242	107	360	CF	Stalling & Mayer, 1972
Channel catfish (*I. punctatus*)	A-1242	87	720	CF	Mayer *et al.*, 1977
Cutthroat trout (*S. clarki*)	A-1242	3820-7680	96	S	Johnson and Finley, 1980
Rainbow trout (*O. mykiss*)	A-1242	67	120	CF	Johnson and Finley, 1980; Mayer et al., 1977
Rainbow trout (*O. mykiss*)	A-1242	12	600	CF	Mayer *et al.*, 1977
Yellow perch (*P. flavescens*)	A-1242	> 150	96	CF	Johnson and Finley, 1980
Fathead minnow (*P. promelas*)	A-1242	15-300	96	CF, M	Nebeker *et al.*, 1974
Fathead minnow (*P. promelas*)	A-1242	28	720		Veith, 1976
Scud (*G. pseudolimnaeus*)	A-1248	52	96	S	Stalling & Mayer, 1972
Cladoceran (*D. magna*)	A-1248	25	504	S	Nebeker & Puglisi, 1974
Scud (*G. pseudolimnaeus*)	A-1248	52	96	CF	Nebeker & Puglisi, 1974

Cladoceran (*D. magna*)	A-1248	2.6	336	CF	Nebeker & Puglisi, 1974
Blue gill (*L. macrochirus*)	A-1248	10	480	CF	Stalling & Mayer, 1972
Blue gill (*L. macrochirus*)	A-1248	76	360	CF	Stalling & Mayer, 1972
Blue gill (*L. macrochirus*)	A-1248	136	120	CF	Mayer *et al.*, 1977
Channel catfish (*I. punctatus*)	A-1248	225	240	CF	Stalling & Mayer, 1972
Channel catfish (*I. punctatus*)	A-1248	127	360	CF	Stalling & Mayer, 1972
Channel catfish (*I. punctatus*)	A-1248	75	720	CF	Mayer *et al.*, 1977
Cutthroat trout (*S. clarki*)	A-1248	5100-6480	96	S	Johnson and Finley, 1980
Rainbow trout (*O. mykiss*)	A-1248	54	120	CF	Johnson and Finley, 1980
Rainbow trout (*O. mykiss*)	A-1248	3.4	600	CF	Mayer *et al.*, 1977
Yellow perch (*P. flavescens*)	A-1248	> 100	96	CF	Johnson & Finley, 1980
Fathead minnow (*P. promelas*)	A-1248	4.7	720	U	Defoe *et al.*, 1978
Glass shrimp (*P. kadiakensis*)	A-1254	3	168	CF	Stalling & Mayer, 1972
Scud (*G. pseudolimnaeus*)	A-1254	2 400	96	S	Stalling & Mayer, 1972
Crayfish (*O. nais*)	A-1254	100	168	S	Stalling & Mayer, 1972
Crayfish (*O. nais*)	A-1254	80	168	CF	Stalling & Mayer, 1972
Water flea (*D. magna*)	A-1254	24	336	S	Maki & Johnson, 1975

Water flea (*D. magna*)	A-1254	31	504	S	Nebeker & Puglisi, 1974
Cladoceran (*D. magna*)	A-1254	1.8	336	CF	Nebeker & Puglisi, 1974
Cladoceran (*D. magna*)	A-1254	1.3	504	CF	Nebeker & Puglisi, 1974
Midge (*T. dissimilis*) (larvae)	A-1254	0.65	504	CF	Nebeker & Puglisi, 1974
(pupae)	A-1254	0.45	504	CF	Nebeker & Puglisi, 1974
Damselfly (*I. verticalis*)	A-1254	200	96	CF, M	Mayer *et al.*, 1977
Glass shrimp (*P. kadiakensis*)	A-1254	3.0	168	CF	Johnson & Finley, 1980
Dragonfly (*Macromia sp.*)	A-1254	800	168	S	Johnson & Finley, 1980
Cladoceran (*D. magna*)	A-1254	1.8 - 24	336	U	EPA, 1980
Cladoceran (*D. magna*)	A-1254	1.3	504	U	EPA, 1980
Hydra (*H. oligactis*)	A-1254	20 000	72	U	Adams & Haileselassie, 1984
Blue gill (*L. macrochirus*)	A-1254	2 740	96	S	Stalling & Mayer, 1972
Blue gill (*L. macrochirus*)	A-1254	200	360	CF	Stalling & Mayer, 1972
Blue gill (*L. macrochirus*)	A-1254	140	480	CF	Stalling & Mayer, 1972
Blue gill (*L. macrochirus*)	A-1254	54	600	CF	Stalling & Mayer, 1972
Blue gill (*L. macrochirus*)	A-1254	177	720	CF	Mayer *et al.*, 1977
Channel catfish (*I. punctatus*)	A-1254	139	720	CF	Mayer *et al.*, 1977
Channel catfish (*I. punctatus*)	A-1254	741	360	CF	Stalling & Mayer, 1972

Channel catfish (*I. punctatus*)	A-1254	300	480	CF	Stalling & Mayer, 1972
Cutthroat trout (*S. clarki*)	A-1254	38700-46700	96	S	Johnson & Finley, 1980
Rainbow trout (*O. mykiss*)	A-1254	142	120	CF	Johnson & Finley, 1980
Rainbow trout (*O. mykiss*)	A-1254	8	240	CF	Stalling & Mayer, 1972
Rainbow trout (*O. mykiss*)	A-1254	27	600	CF	Mayer *et al.*, 1977
Yellow perch (*P. flavescens*)	A-1254	> 150	96	CF	Johnson & Finley, 1980
Fathead minnow (*P. promelas*)	A-1254	7.7	96	CF, M	Nebeker *et al.*, 1974
Water flea (*D. magna*)	A-1260	36	504	S	Nebeker & Puglisi, 1974
Channel catfish (*I. punctatus*)	A-1260	296	480	CF	Stalling & Mayer, 1972
Channel catfish (*I. punctatus*)	A-1260	535	240	CF	Mayer *et al.*, 1977
Channel catfish (*I. punctatus*)	A-1260	482	360	CF	Mayer *et al.*, 1977
Channel catfish (*I. punctatus*)	A-1260	512	480	CF	Mayer *et al.*, 1977
Channel catfish (*I. punctatus*)	A-1260	465	560	CF	Mayer *et al.*, 1977
Channel catfish (*I. punctatus*)	A-1260	433	720	CF	Mayer *et al.*, 1977
Channel catfish (*I. punctatus*)	A-1260	137	720	CF	Stalling & Mayer, 1972
Blue gill (*L. macrochirus*)	A-1260	400	720	CF	Mayer *et al.*, 1977
Blue gill (*L. macrochirus*)	A-1260	151	720	CF	Stalling & Mayer, 1972

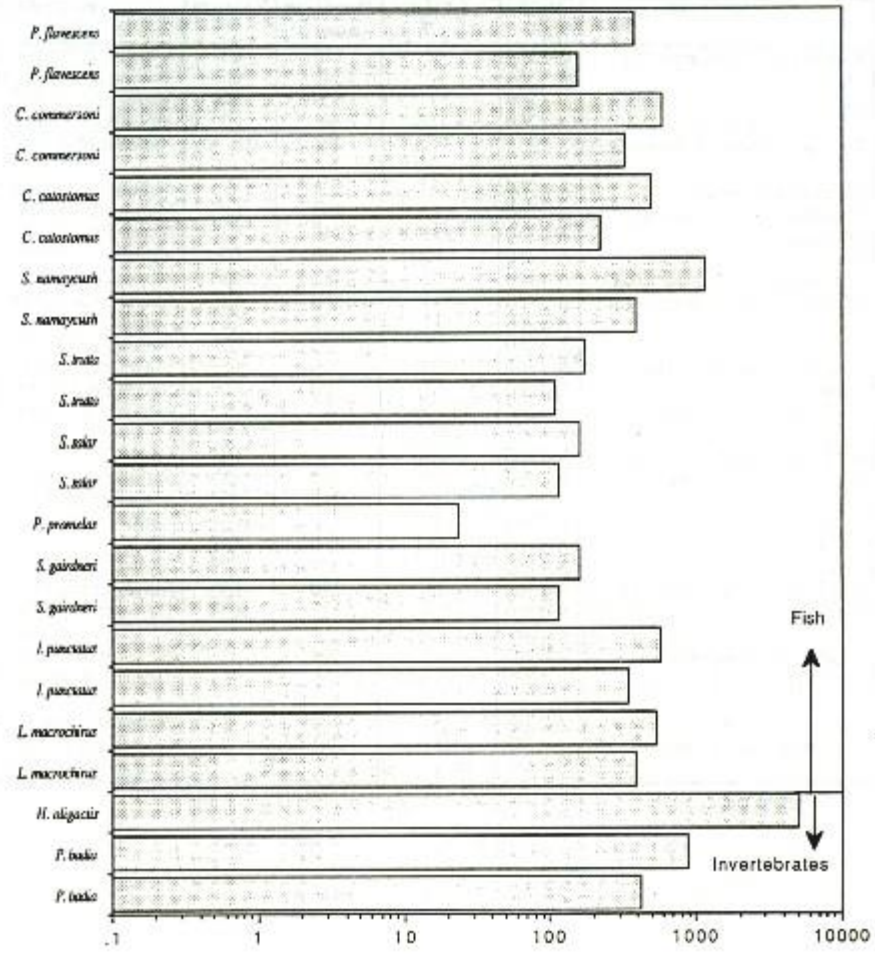
Cutthroat trout (*S. clarki*)	A-1260	55400-67000	96	S	Johnson & Finley, 1980
Yellow perch (*P. flavescens*)	A-1260	> 200	96	CF	Johnson & Finley, 1980
Rainbow trout (*O. mykiss*)	A-1260	21	480	CF	Stalling & Mayer, 1972
Rainbow trout (*O. mykiss*)	A-1260	326	240	CF	Mayer *et al.*, 1977
Rainbow trout (*O. mykiss*)	A-1260	143	360	CF	Mayer *et al.*, 1977
Rainbow trout (*O. mykiss*)	A-1260	78	480	CF	Mayer *et al.*, 1977
Rainbow trout (*O. mykiss*)	A-1260	49	600	CF	Mayer *et al.*, 1977
Rainbow trout (*O. mykiss*)	A-1260	51	720	CF	Mayer *et al.*, 1977
Fathead minnow (*P. promelas*)	A-1260	3.3	720	CF	Defoe *et al.*, 1978
Water flea (*D. magna*)	A-1262	43	504	S	Nebeker & Puglisi, 1974
Cutthroat trout (*S. clarki*)	A-1262	> 50 000	96	S	Johnson & Finley, 1980
Water flea (*D. magna*)	A-1268	253	504	S	Nebeker & Puglisi, 1974
Cutthroat trout (*S. clarki*)	A-1268	> 50 000	96	S	Johnson & Finley, 1980

* S = Static, CF = Continuous flow, M = Measured concentration; U = Unknown

Figure 2

FIGURE 2

72- to 720-h LC50s for freshwater organisms exposed to Aroclor 1016



LC50 (Aroclor 1016) - µg/L

Figure 3

FIGURE 3

96- to 720-h LC50s for freshwater organisms exposed to Aroclor 1242

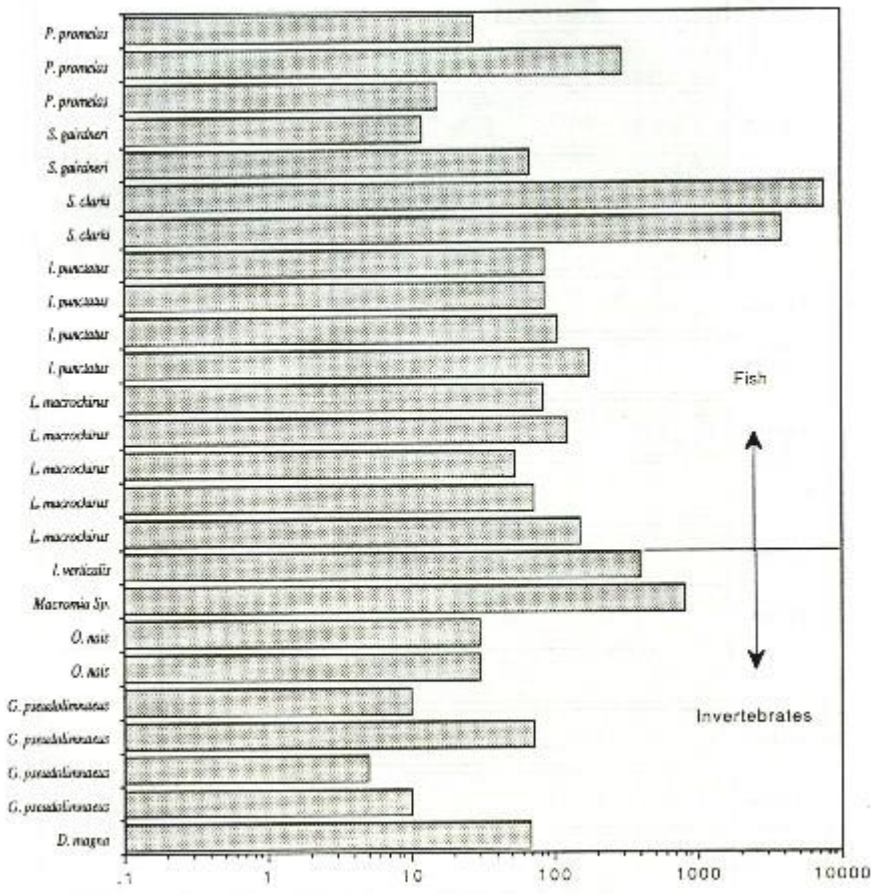
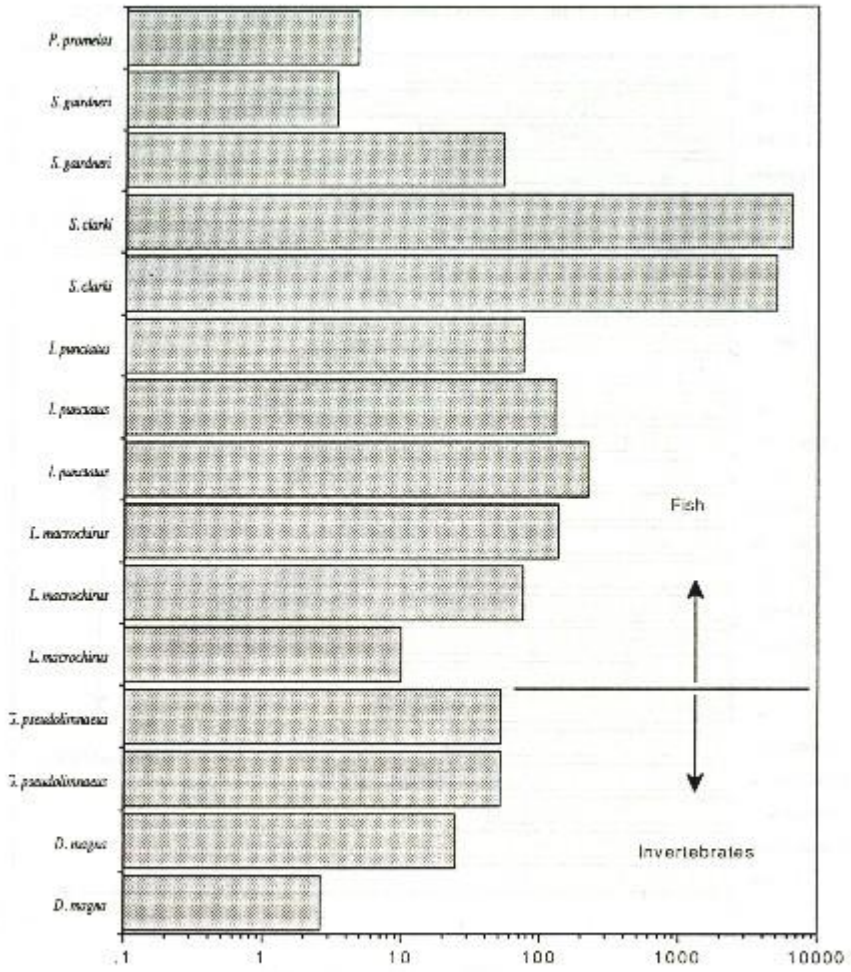


Figure 4

FIGURE 4

96- to 720-h LC50s for freshwater organisms exposed to Aroclor 1248



LC50 (Aroclor 1248) - µg/L

Figure 5

FIGURE 5

96- to 720-h LC50s for freshwater organisms exposed to Aroclor 1254

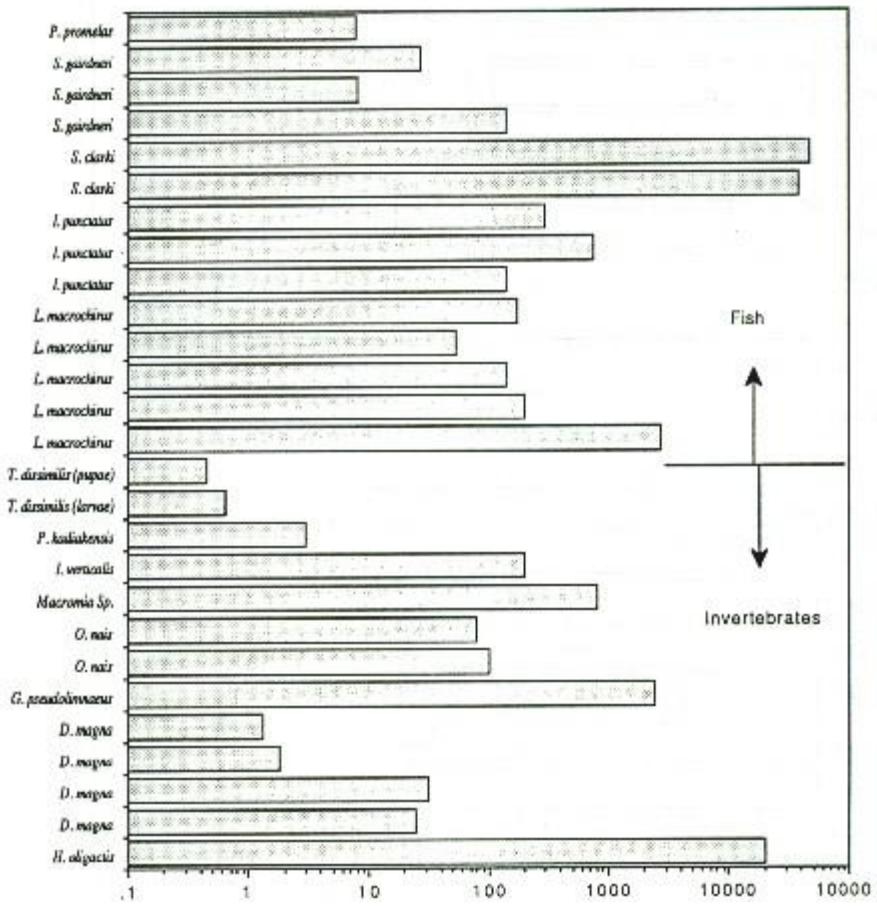
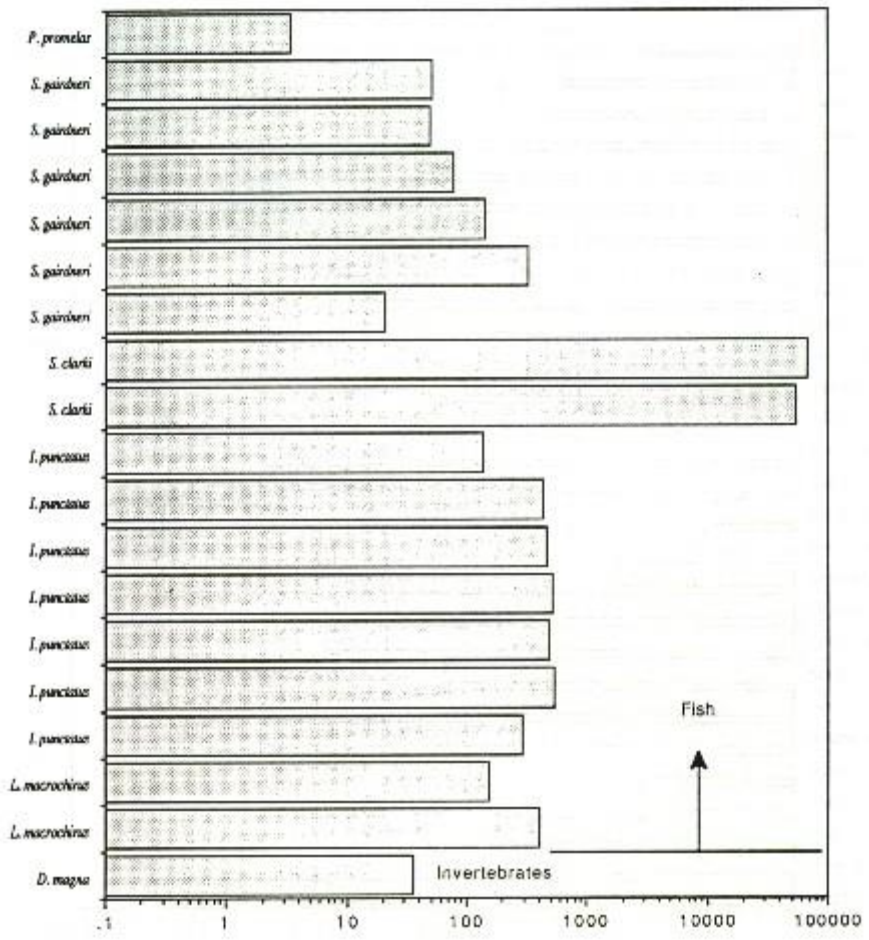


Figure 6

FIGURE 6

96- to 720-h LC50s for freshwater organisms exposed to Aroclor 1260



LC50 (Aroclor 1260) - µg/L

TABLE 11

Chronic and Sublethal Toxicity of Commercial PCB Formulations to freshwater algae and aquatic plants

Organisms	PCB	Conc. (µg/L)	Effects	Reference

Green algae (*C. pyrenoidosa*)	A-1232	100-1 000	Transient growth reduction	Hawes *et al.*, 1976
Fungus (*A. flavus*)	A-1232	5 000	Depressed growth	Murado *et al.*, 1976
E. coli	A-1242	10	Stimulated growth	Keil *et al.*, 1972
Green algae (*C. pyrenoidosa*)	A-1242	100-1 000	Transient growth reduction	Hawes *et al.*, 1976
Diatom (*C. closteria*)	A-1242	10	No notable effect	Keil *et al.*, 1971
-ditto-	A-1242	100	Sharply reduced growth	-ditto-
Green algae (*Euglena*)	A-1242	10 000	Depressed growth	Bryan & Olafsson, 1978
Alga (*S. obtusiusculus*)	A-1242	300	Growth inhibition	Larsson & Tillberg, 1975
Freshwater diatom (*S. acus*) & Green algae (*A. falcatus*)	A-1242	1	Decrease in cell number in 9 days	Glooschenko & Glooshenko, 1975
Green algae (*S. quadricauda*)	A-1242	5	Decrease in cell number in 9 days	-ditto-
Green algae (*A. falcatus*)	A-1242	5	Little effect on photosynthetic activity even after 2 d	-ditto-
Fungus (*A. flavus*)	A-1254	5 000	Depressed growth	Murado *et al.*, 1976
Green algae (*C. pyrenoidosa*)	A-1254	100-1 000	Transient growth reduction	Hawes *et al.*, 1976
Fungus (*A. flavus*)	A-1260	25 000	Induction (aldrin epoxidase)	Murado *et al.*, 1976
Green algae (*C. pyrenoidosa*)	A-1268	100-1 000	Transient growth reduction	Hawes *et al.*, 1976

Planktonic algae	various	10-100	Decreased growth	Laake, 1984
Centric diatoms	various	10-100	Decreased numbers	Laake, 1984
Dinoflagellates	various	10-100	Increased numbers	Laake, 1984
Blue-green alga (*Phormidium*)	C-A30	50	Inhibited growth	Zullei and Benecke, 1978
-ditto-	C-A60	100	No effect on growth	-ditto-

TABLE 12

Chronic toxicity of Commercial PCB Formulations to freshwater aquatic animals.

Organisms	PCB	Conc. (µg/L)	Effects	Reference
Fathead minnow (*P. promelas*)	A-1016	44	significant mortality (in 30 days)	Hermanutz & Puglisi, 1976
Amphipod (*H. azteca*)	A-1242	17.6-27.1	no effect on survival, growth, or reproduction	Borgmann *et al.*, 1990
Amphipod (*H. azteca*)	A-1242	51-56	complete mortality of *Hyalella*	Borgmann *et al.*, 1990
Scud (*G. pseudolimnaeus*)	A-1242	2.8	Good survival & reproduction of young after 60 d.	Nebeker & Puglisi, 1974
Fathead minnow (*P. promelas*)	A-1242	15	No spawning	Nebeker *et al.*, 1974
Fathead minnow (*P. promelas*)	A-1242	5	Reduced spawning	Nebeker *et al.*, 1974
Fathead minnow (*P. promelas*)	A-1242	23	Significant mortality (in 30 days)	Hermanutz & Puglisi, 1976
Fathead minnow (*P. promelas*)	A-1242	0.31	Inhibition of ATPase activity	Cutkomp *et al.*, 1972

Daphnia magna	A-1248	5	Decreased reproduction	Stalling & Mayer, 1972
Cladoceran (*D. magna*)	A-1248	1.0	16% reproductive impairment in 2 weeks	Nebeker & Puglisi, 1974
Scud (*G. pseudolimnaeus*)	A-1248	2.2	Good survival & reproduction of young after 60 d.	Nebeker & Puglisi, 1974
Fathead minnow (*P. promelas*)	A-1248	0.1	No effect on F1 or F2 generations	Defoe *et al.*, 1978
Fathead minnow (*P. promelas*)	A-1248	0.54	Wt. & survival unaffected	Nebeker *et al.*, 1974
Flagfish (*J. florodae*)	A-1248	0.54	Survival & wt. unaffected	Nebeker *et al.*, 1974
Protozoan (*T. pyriformis*)	A-1254	1	Reduced population growth	Nimmo *et al.*, 1975
Protozoan (*T. pyriformis*)	A-1254	0.48-1.0	16% reproductive impairment in 2-3 weeks	Nebeker & Puglisi, 1974
Grass shrimp (*P. pugio*)	A-1254	15	Killed larvae	Roesijadi *et al.*, 1976
Grass shrimp (*P. pugio*)	A-1254	3	Delayed larval development	Roesijadi *et al.*, 1976
Oyster (*C. virginica*)	A-1254	1	No effect	Nimmo *et al.*, 1975
Oyster (*C. virginica*)	A-1254	5	Reduced growth, tissue changes	Nimmo *et al.*, 1975
Brook trout (*S. fontinalis*)	A-1254	0.7-1.5 < 0.43	No effect levels for growth & mortality; Backbone composition unaffected	Mauck *et al.*, 1978
Brook trout (*S. fontinalis*)	A-1254	0.94	No effect on survival, growth, reproduction	Snarski & Puglisi, 1976
Fathead minnow (*P. promelas*)	A-1254	0.31	Inhibition of ATPase activity	Koch *et al.*, 1972
Pinfish (*Lagodon*)	A-1254	5	Increased disease susceptibility	Hansen *et al.*, 1971

Spot (*Leistomus*)	A-1254	5	Increased disease susceptibility	Hansen *et al.*, 1971
Fathead minnow (*P. promelas*)	A-1254	1.8	Spawning significantly lower than control	Nebeker *et al.*, 1974
Fathead minnow (*P. promelas*)	A-1260	0.7	Highest no observable adverse effect level	Defoe *et al.*, 1978
Fathead minnow (*P. promelas*)	A-1260	2.1	Lowest observable adverse effect level	Defoe *et al.*, 1978
Fathead minnow (*P. promelas*)	A-1260	1.2	Maximum adverse toxicant concentration	Defoe *et al.*, 1978
Rainbow trout (*O. mykiss*) A1254:A1260 = 1:2	Tech. grade	1.5	No effect on survival & growth in 90 d	Mayer *et al.*, 1985
Rainbow trout (*O. mykiss*) A1254:A1260 = 1:2	Trans-former oil	0.43	No effect on survival & growth in 90 d	Mayer *et al.*, 1985

Figure 7

FIGURE 7

Chronic toxicity of PCBs to freshwater animals

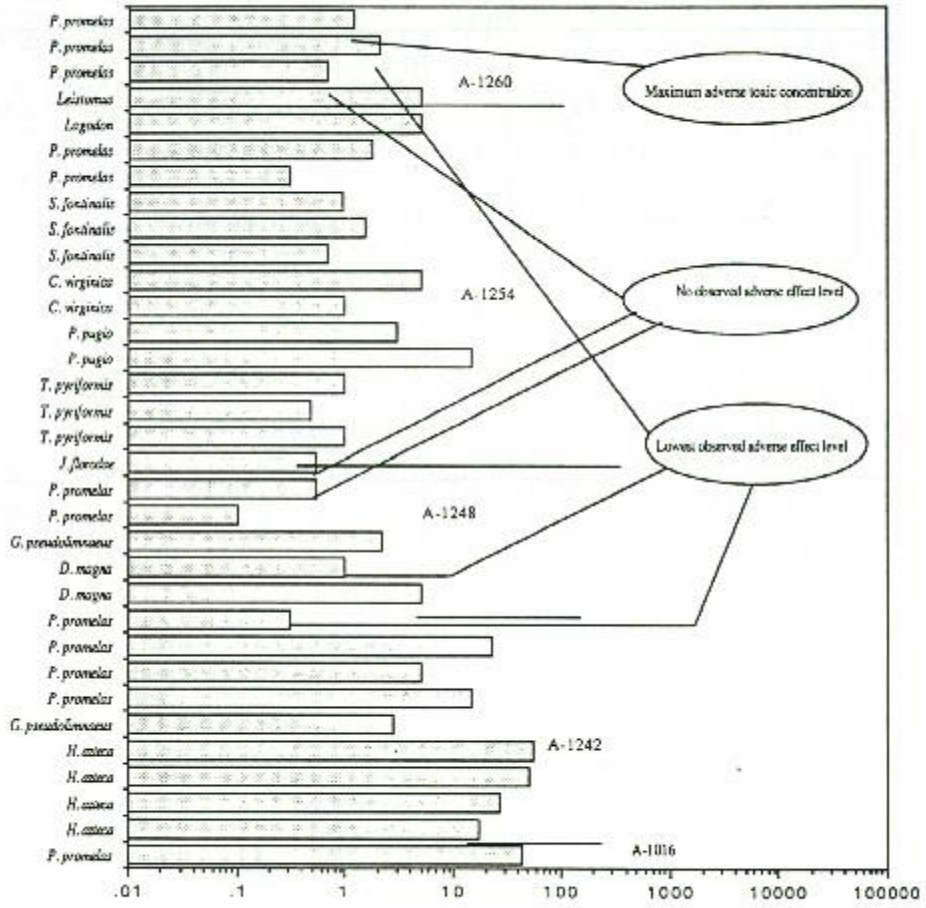
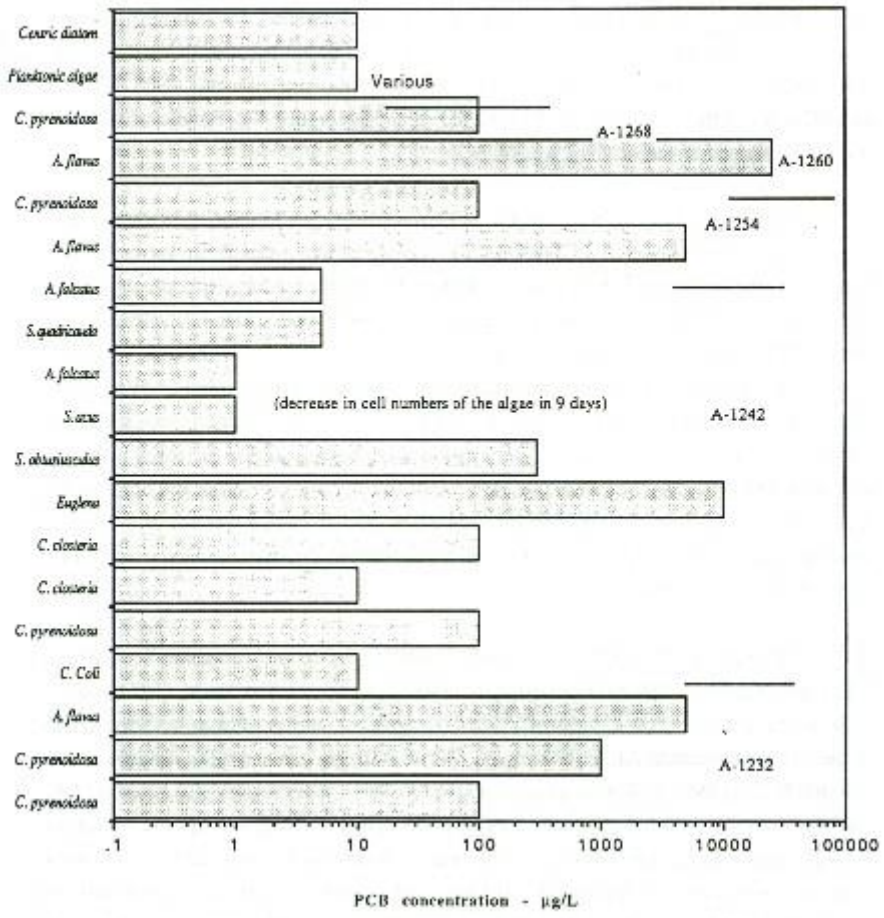


Figure 8

FIGURE 8

Toxicity of PCBs to freshwater algae and aquatic plants



Addison *et al.* (1978) found that trout (*S. fontinalis*) fed Aroclor 1254 to produce a tissue (fillet) concentration of 39 µg PCB/g wet weight showed a significant increase in the activity of ethoxycoumarin O-deethylase (ECOD). During the feeding experiment, one fish out of the six died after 17 days on the PCB diet. Also, there was substantial variation in the sensitivity of various MO (mono-oxygenase) activities to xenobiotics. For instance, (i) EROD activity increased in rainbow trout, carp, and channel catfish treated with single intra-peritoneal injections of 1.0 µg Aroclor 1254/g body weight (bw) (Melancon and Lech, 1983; Ankley *et al.*, 1986); (ii) ECOD activity in rainbow trout was also increased by a dose of 1.0 µg Aroclor 1254/g body weight. However, ECOD activity in catfish did not increase until treated with a dose of 10 µg Aroclor 1254/g bw, and, in carp, it was not stimulated by doses as high as 200 µg Aroclor 1254/g bw (Melancon *et al.*, 1981; Melancon and Lech, 1983; Ankley *et al.*, 1986).

Using groups of eight rainbow trout (*O. mykiss*) fed rations containing 0, 1, 10 and 100 µg/g Aroclor 1254 over a period of 330 days, Nestel and Budd (1974) found pathological changes in the kidneys of 13 fish. The greatest number of cases occurred in fish at the 10 µg/g concentration. Fish fed 1 and 10, and 100 µg/g diet had mean residues of 1.4, 2.3, and 80.1 µg PCB/g (wet weight), respectively. These investigators also noted that the rainbow trout survived an oral intake of PCBs at the rates of 0.04 µg, 0.4 µg, and 4.0 µg per g body weight (bw) for 330 days. The results of the Nestle and Budd (1974) study combined with those of Melancon and Lech (1983) above, appear to suggest that the induction of EROD activity at 1.0 µg PCB/g bw may not necessarily mean initiation of toxic effects (including death).

(c) Toxicity of PCB isomers or congeners

Industrial PCBs are mixtures of several isomers and congeners. The structure-activity relationships have shown that several PCB congeners are similar to 2,3,7,8-TCDD in structure and in induction of aryl hydrocarbon hydroxylase (AHH) and ethoxyresorufin O-deethylase (EROD) activity in rat hepatoma cells. Based on these structure-activity relationships, it was found that the true coplanar PCBs (#77, #126, and #169) and coplanar PCBs which are additionally halogenated in meta or para positions, are among the most toxic PCBs. Among 15 congeners tested, 3,3',4,4',5 pentachlorobiphenyl (PCB #126) was the most potent inducer of the AHH and EROD activities and was followed in the induction response by 3,3',4,4',5,5' hexachlorobiphenyl (PCB #169) and 3,3',4,4' tetrachlorobiphenyl (PCB #77) (Table 6; Safe, 1987; Sawyer and Safe, 1982).

The congener-specific toxicity data for aquatic life are limited, especially with respect to the relatively more toxic coplanar congeners. Also, the available data show that a non-coplanar PCB congener (e.g., PCB #52) can be more severely toxic to amphipod *Hyalella. azteca* than a coplanar congener (e.g., PCB #77) (Table 13; Figure 9). The most severe chronic toxicity to a PCB congener was displayed by *D. pulicaria* exposed to PCB #4 at 0.05 µg/L (Bridgham, 1988; Figure 9).

7.1.2 Marine environment

(a) Lethal and sublethal toxicity

The lethal and sublethal toxic effects of PCB formulations on marine organisms are shown in Tables 14 and 15 and Figures 10 through 13. The data suggest that PCBs are as toxic to marine organisms as they are to freshwater organisms. In flow-through chronic tests with Aroclor 1254, Hansen *et al.* (1973, 1974) found that the survival of fry and the hatching of embryos from exposed sheepshead minnow adults were affected

by 0.1 µg/L PCB (measured concentration). Adverse effects were also observed on the growth of a marine diatom (*Rhizosolenia setigera*) exposed to 0.1 µg Aroclor 1254/L; the growth was more severely reduced at a lower temperature (10 C) than at a higher temperature (15 C) (Fisher and Wurster, 1973) .

Cosper *et al.* (1987) found that the marine diatom *Ditylum brightwellii*, pre-treated with sublethal concentrations of 10 to 30 µg/L over a period of 30 days, developed a resistance to PCBs. The PCB-resistant strain exhibited greater tolerance to PCB than the PCB-sensitive strain under all environmental conditions. However, PCB resistance decreased the tolerance of the strain to lower salinities and nitrogen limitation, but increased its tolerance to lower temperatures.

TABLE 13

Toxicity of PCB congeners to freshwater aquatic organisms.

Organisms	PCB Congener	Conc. (µg/L)	Effects	Reference
Amphipod (*G. fasciatus*)	22	70	96-h LC_{50}	Mayer *et al.*, 1977
Amphipod (*G. fasciatus*)	15	100	96-h LC_{50}	Mayer *et al.*, 1977
Amphipod (*G. fasciatus*)	8	120	96-h LC_{50}	Mayer *et al.*, 1977
Amphipod (*G. fasciatus*)	155	150	96-h LC_{50}	Mayer *et al.*, 1977
Amphipod (*G. fasciatus*)	101	210	96-h LC_{50}	Mayer *et al.*, 1977
Daphnia magna	1	710	48-h LC_{50}	Dill *et al.*, 1982
Daphnia magna	2	430	48-h LC_{50}	Dill *et al.*, 1982
Daphnia magna	3	420	48-h LC_{50}	Dill *et al.*, 1982
Daphnia magna	47	30	48-h LC_{50}	Dill *et al.*, 1982
Blue-green algae *(Phormidium)*	1, 2, 3, 4, 5, 6, 8, 9, 15, 28, & 37	100 µg/ 2.1µg algae (dry weight)	Inhibited growth	Zullei and Benecke, 1978

Blue-green algae *(Phormidium)*	7, 18, 52, 141, & 209	100	No effect on growth	Zullei and Benecke, 1978
Daphnia pulicaria	4	0.05-0.1 10	Significant mortality and inhibition of reproduction; Inhibition at 10 µg/L occurred only after continuous exposure for 3 generations	Bridgham, 1988
Amphipod (*H. azteca*)	52	6.5-10.4	no effect on survival, growth, or reproduction	Borgmann *et al.*, 1990
Amphipod (*H. azteca*)	52	37.8	complete mortality of *Hyalella*	Borgmann *et al.*, 1990
Amphipod (*H. azteca*)	77	2 700	No toxic effect	Borgmann *et al.*, 1990

Figure 9

FIGURE 9

Toxicity of PCB congeners to freshwater organisms

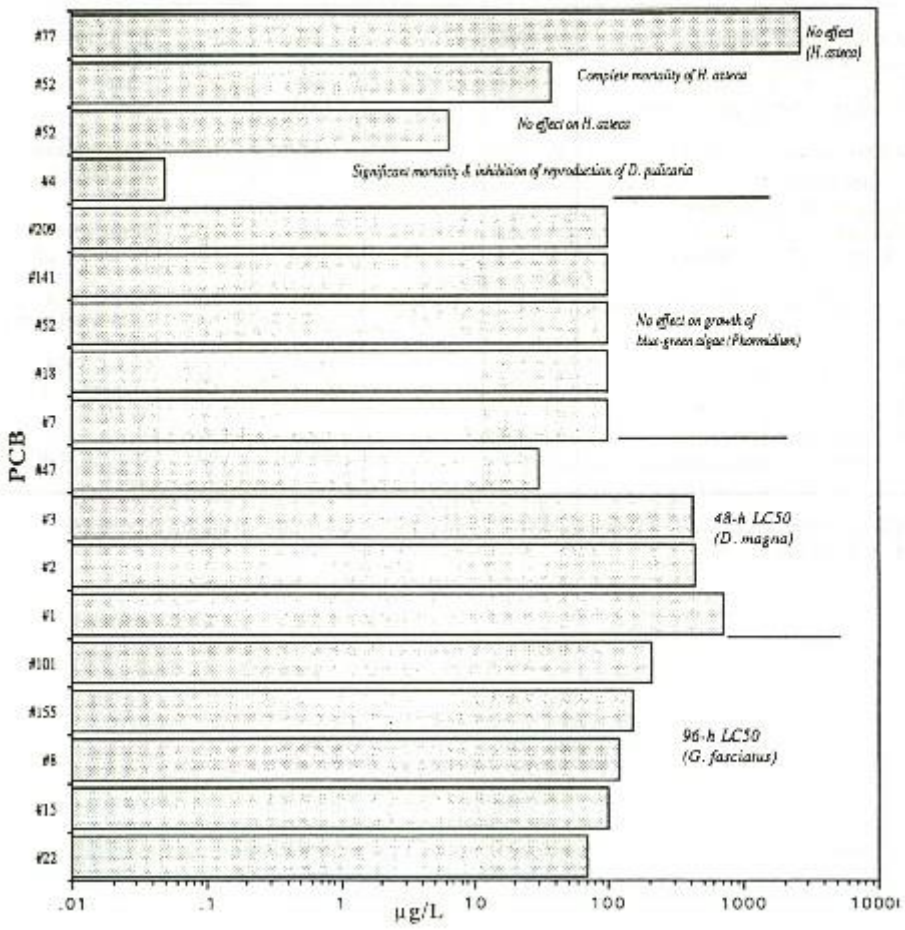


TABLE 14

Lethal Toxicity of PCBs to Marine Aquatic Animals

Organisms	PCB	LC50 (µg/L)	Duration (hours)	System *	Reference
Grass shrimp (*P. pugio*)	A-1016	12.5	96	CF, U	EPA, 1980
Brown shrimp (*P. aztecus*)	A-1016	10.5	96	CF, U	EPA, 1980

Eastern Oyster (*C. virginica*)	A-1016	10.2	96	CF, U	EPA, 1980
Pinfish (*L. rhomboides*)	A-1016	21	1 008		EPA, 1980
Decapod (*L. adespersus*) at 50 o/oo salinity 30 o/oo & 10 o/oo salinities	A-1254	10-100 100-1 000	96	NK	Dalla Venezia and Fossato, 1984
Grass shrimp (*P. pugio*)	A-1254	6.1-7.8	96	NK	Ernst, 1984
Pink shrimp (*P. duorarum*)	A-1254	0.94	360	CF	Nimmo *et al.*, 1971
Sheepshead minnow (C*. variegatus*)	A-1254	0.1-0.32	504	NK	Ernst, 1984
Sheepshead minnow (C*. variegatus*)	A-1254	0.9	504	NK	Schimmel *et al.*, 1974
Spot (*L. xanthurus*)	A-1254	0.5	912	NK	Ernst, 1984
Spot (*L. xanthurus*)	A-1254	5	480-1 080	CF	Hansen *et al.*, 1971
Pinfish (*L. rhomboides*)	A-1254	0.5	288	NK	Ernst, 1984
Pinfish (*L. rhomboides*)	A-1254	5	336-840	CF	Hansen *et al.*, 1971

* CF = Continuous flow; U = Unmeasured (i.e., concentration not measured during experiment);
NK = Not known

TABLE 15

Sublethal and Chronic Toxicity of PCBs to Marine Aquatic Plants and Animals.

Organisms	PCB	Conc. (µg/L)	Effects	Reference
Purple sea urchin (*A. punctulata*)	A-1016	500	Reduced fertilisation efficiency in eggs	Adams & Slaughter-Williams, 1988
Sheepshead minnow (*C. variegatus*)	A-1016	0.1-3.2	Early life stage test; no effect on eggs, fry, juvenile, or adult fish	Hansen *et al.*, 1975

Phytoplankton communities	A-1242	>1.0	Reduced carbon uptake	Moore & Harriss, 1972
Diatom (*T. pseudonana* 3H)	A-1254	0.1	Reduced growth rate	Fisher & Wurster 1973; Fisher *et al.*, 1974
Diatom (*R. setigera*)	A-1254	0.1	Reduced growth rate at 10 C for first 192 h	Fisher & Wurster 1973
Diatom (*T. pseudonana*)	A-1254	1.0	Reduced cell division by day 3	Harding & Phillips, 1978
Heptophyceae (algae) (*I. galbana*)	A-1254	1.0	Reduced cell division by day 3	Harding & Phillips, 1978
Chlorophyceae (algae) (*D. tertiolecta*)	A-1254	1.0-50	No effect	Harding & Phillips, 1978
Chlorophyceae (algae) (*D. tertiolecta*)	A-1254	100	Increased cell division	Harding & Phillips, 1978
Diatom (*S. costatum*)	A-1254	10	Reduced cell division	Harding & Phillips, 1978
Chrysophyceae (algae) (*M. lutheri*)	A-1254	10	Reduced cell division	Harding & Phillips, 1978
Diatom (*C. socialis*)	A-1254	10	Reduced cell division	Harding & Phillips, 1978
Diatom (*N. longissima*)	A-1254	25	Reduced cell division by day 4	Harding & Phillips, 1978
Phytoplankton communities	A-1254	>1.0	Reduced carbon uptake	Moore & Harriss, 1972
Phytoplankton communities	A-1254	1.0-10	Reduced biomass and size	O'Connors *et al.*, 1978
Diatom (*S. costatum*)	A-1254	10	Reduced growth	Mosser et al., 1972
Protozoa (*T. pyriformis* W)	A-1254	1.0	Reduced growth rate in 96 h	Cooley *et al.*, 1972
Purple sea urchin (*A.*	A-	1 000	Fertilisation efficiency in	Adams &

punctulata)	1254		eggs unaffected	Slaughter-Williams, 1988
Eastern Oyster (*C. virginica*)	A-1254	5	Reduced growth in 24 weeks	Lowe *et al.*, 1972
Eastern Oyster (*C. virginica*)	A-1254	1	No effect on growth in 30 wk.	Lowe *et al.*, 1972
Pink shrimp (*P. duorarum*)	A-1254	0.6-19	Min. affecting conc.=0.9 µg/L	Nimmo *et al.*, 1975
Grass shrimp (*P. pugio*)	A-1254	0.2-12.5	Min. affecting conc.=1.3 µg/L	Nimmo *et al.*, 1975
Brown shrimp (*P. aztecus*)	A-1254	0.1-1.4	Min. affecting conc.=1.4 µg/L	Nimmo *et al.*, 1975
Longnose killifish (*F. similis*)	A-1254	1.-100	Min. affecting conc.=1.0 µg/L	Nimmo *et al.*, 1975
Pinfish (*L. rhomboides*)	A-1254	5.0	Min. affecting conc.=5.0 µg/L	Nimmo *et al.*, 1975
Spot (*L. xanthurus*)	A-1254	1.0-5.0	Min. affecting conc.=5.0 µg/L	Nimmo *et al.*, 1975
Sheepshead minnow-fry (*C. variegatus*)	A-1254	0.06 0.16	No effect maximum conc. Minimum affecting conc.	Schimmel *et al.*, 1974
Sheepshead minnow-fry (*C. variegatus*)	A-1254	10	Lethargy, fin rot, mortality	Hansen *et al.*, 1971
Sheepshead minnow-fry (*C. variegatus*)	A-1254	0.1	Affected reproduction or hatching of embryos from exposed adults	Hansen *et al.*, 1973 and 1974

Figure 10

FIGURE 10

Lethal toxicity for marine organisms exposed to Aroclor 1016

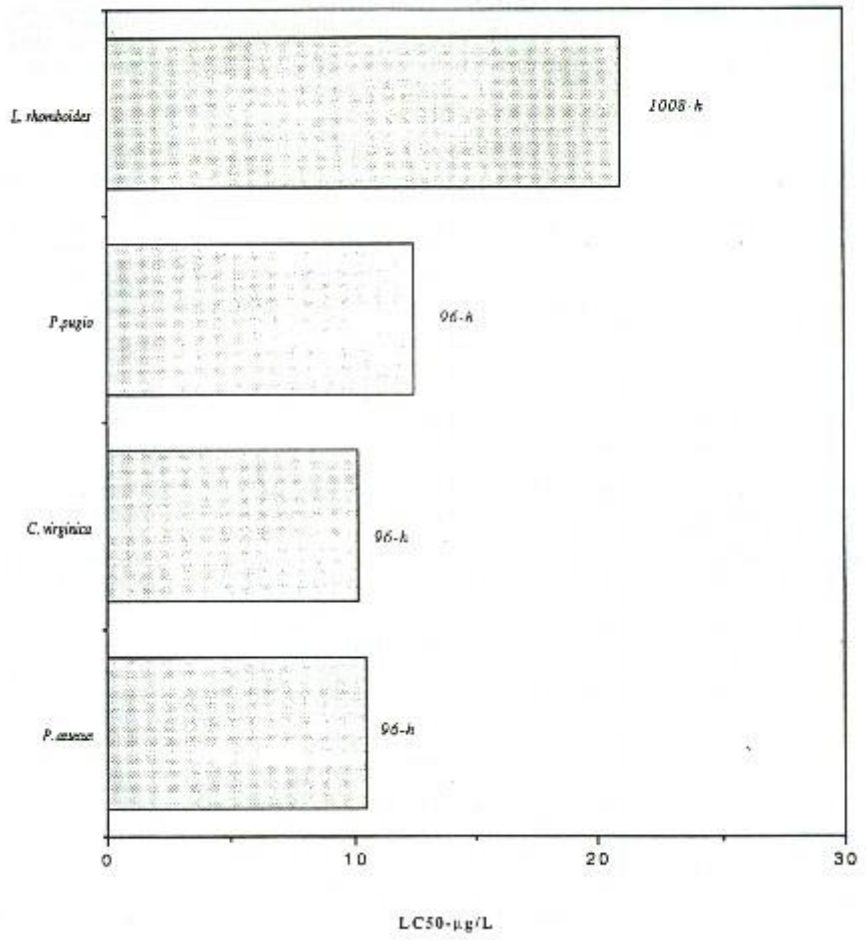


Figure 11

FIGURE 11

Lethal toxicity for marine organisms exposed to Aroclor 1254

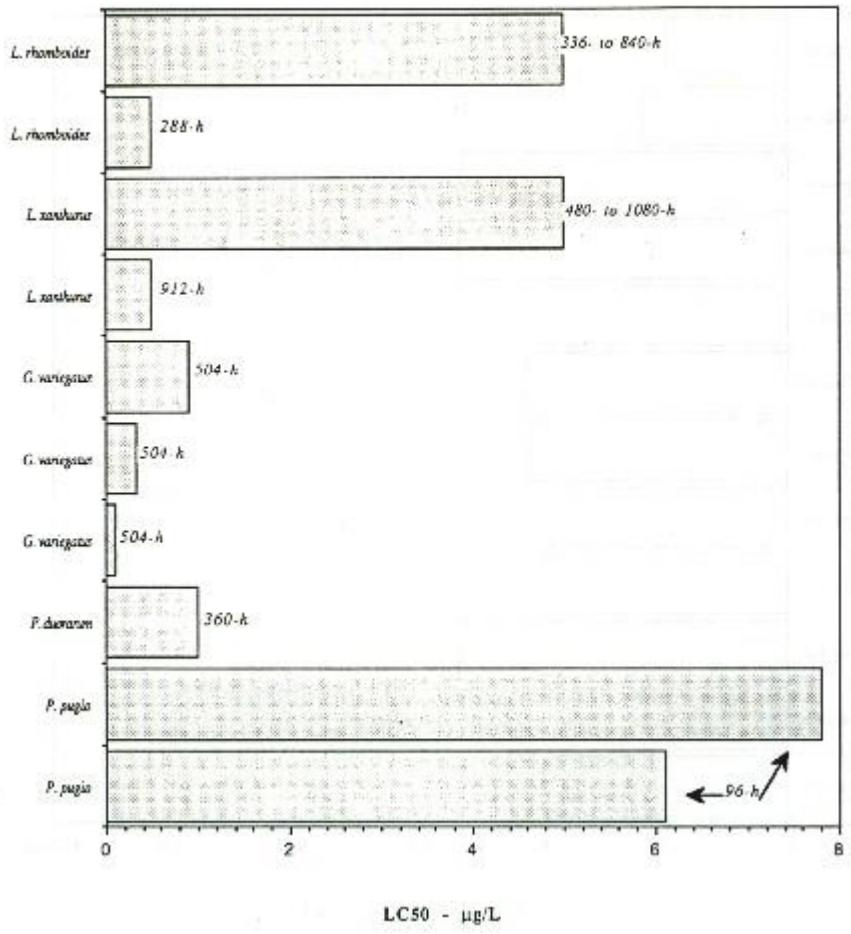


Figure 12

FIGURE 12

Sublethal toxicity of Aroclor 1254 to marine animals

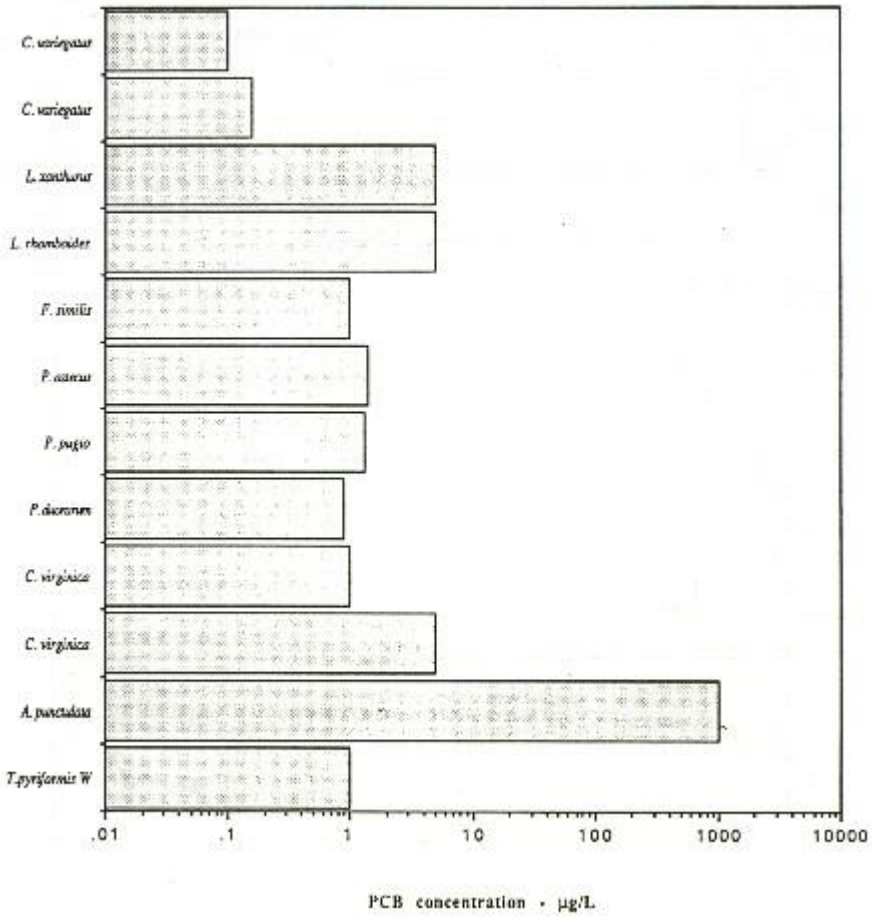
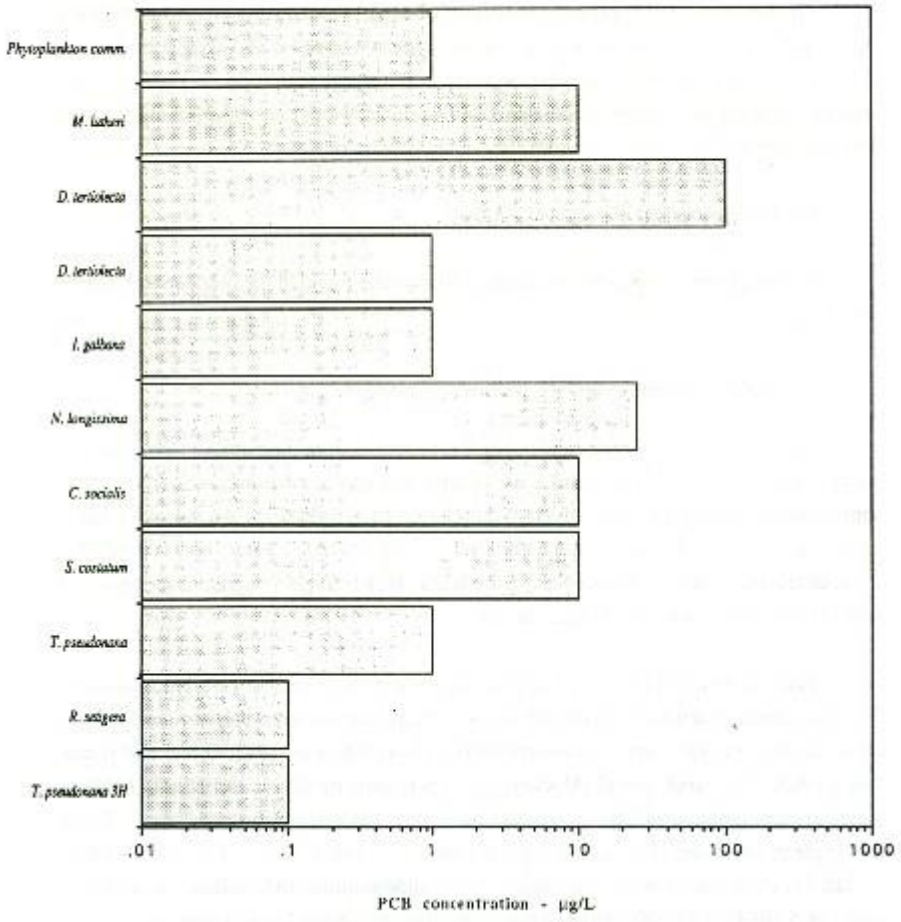


Figure 13

FIGURE 13

Chronic toxicity of Aroclor 1254 to marine algae and plankton



Gruger *et al.* (1977) reported induction of hepatic aryl hydrocarbon hydroxylase (AHH) activity in coho salmon (*Oncorhynchus kisutch*) exposed to 1.0 µg/g of Aroclor 1242 in diet (wet weight). No adverse effects of the concentration of PCBs in fish, however, were reported by the investigators. These observations concur with those reported for freshwater fish in section 7.1.1 (b).

(b) Toxicity of PCB isomers or congeners

No congener-specific toxic reactions to PCBs were found in the literature for marine organisms.

7.1.3 Bioaccumulation of PCBs in aquatic organisms

Accumulation of PCBs in aquatic organisms results from their uptake from food and water. This type of residue buildup in aquatic organisms through water and food is appropriately defined in terms of bioaccumulation factor (BAF). On the other hand, accumulation of contaminants in tissues of aquatic organisms from water alone is defined in terms of bio-concentration or bio-concentration factor (BCF). Table 16 contains the results of several freshwater and marine residue studies.

Fish exposed to PCBs in water alone have shown an excellent correlation between BCF (*i.e*, contaminant concentration in fish ÷ contaminant concentration in water) and the contaminant's octanol-water partition coefficient (K_{ow}) (Mackay, 1982; Oliver and Niimi, 1983, 1985). One such model (Mackay, 1982) was used by Oliver and Niimi (1988) to study accumulation of PCBs in salmonids in the Lake Ontario ecosystem. These investigators concluded that: (a) contaminated food was a major source of the PCB residues in fish, (b) PCB uptake from water alone would underestimate fish residues by at least a factor of 5 (BCF of 780 000 was estimated for salmonids in the Lake Ontario ecosystem), and (c) a bioaccumulation factor of 3 900 000 (the highest value for freshwater aquatic life in Table 16) was found for total PCB in fish.

TABLE 16

Bio-concentration (as BCF) of PCBs in Aquatic Organisms.

Organisms	PCB	Expo. conc.	Duration	Tissue	BCF	Reference
		(µg/L)	(days)			
Freshwater aquatic life						
Fathead minnow (*P. promelas*)	A-1016	8.7	32	Whole	42 500	Veith *et al.*, 1979
Fathead minnow (*P. promelas*)	A-1242	0.86	255	Whole	107 000-274 000	Nebeker *et al.*, 1974
Fathead minnow (*P. promelas*)	A-1242	2.9	255	Whole	32 000-37 000	Nebeker *et al.*, 1974
Fathead minnow (*P.*	A-1242	5.4	255	Whole	63	Nebeker *et al.*,

promelas)					000-81 000	1974
Fathead minnow (*P. promelas*)	A-1248	3.0	250	Whole	60 000	DeFoe *et al.*, 1978
Fathead minnow (*P. promelas*)	A-1248	4.0	32	Whole	70 500	Veith *et al.*, 1979
Daphnid (*D. magna*)	A-1254	1.1	4	Whole	47 000	NAS, 1979
Phantom midge (*C. punctipennis*)	A-1254	1.3	4	Whole	23 000	NAS, 1979
Phantom midge (*C. punctipennis*)	A-1254	1.3	14	Whole	25 000	NAS, 1979
Scud (*G. pseudolimnaeus*)	A-1254	1.6	4	Whole	24 000	NAS, 1979
Scud (*G. pseudolimnaeus*)	A-1254	1.6	21	Whole	27 000	NAS, 1979
Mosquito larvae (*C. tarsalis*)	A-1254	1.5	4	Whole	18 000	NAS, 1979
Crayfish (*O. nais*)	A-1254	1.2	4	Whole	1 700	NAS, 1979
Crayfish (*O. nais*)	A-1254	1.2	21	Whole	5 100	NAS, 1979
Glass shrimp (*P. kadiakensis*)	A-1254	1.3	4	Whole	12 000	NAS, 1979
Glass shrimp (*P. kadiakensis*)	A-1254	1.3	21	Whole	17 000	NAS, 1979
Protozoan (*T. pyriformis*)	A-1254	1.0	4	Whole	60	EPA, 1980
Cichlid (*C. facetum*)	A-1254		3	Spleen	1 862	Gooch & Hamdy, 1983
Cichlid (*C. facetum*)	A-1254		3	Fins	268	Gooch & Hamdy, 1983
Cichlid (*C. facetum*)	A-1254		3	Liver	173	Gooch & Hamdy, 1983
Cichlid (*C. facetum*)	A-1254		3	Muscle	164	Gooch & Hamdy, 1983

Fathead minnow (*P. promelas*)	A-1254	4.3	32	Whole	100 000	Veith *et al.*, 1979
Fathead minnow (*P. promelas*)						
(6 month old fish)	A-1260	1.0	32	Whole	194 000	Veith *et al.*, 1979
(male - adult)	A-1260	2.1	250	Whole	160 000	Defoe *et al.*, 1978
(female - adult)	A-1260	2.1	250	Whole	270 000	Defoe *et al.*, 1978
Fathead minnow (*P. promelas*)	A-1260	0.23	240	Whole	196 000-235 000	Nebeker *et al.*, 1974
Fathead minnow (*P. promelas*)	A-1260	0.52	240	Whole	156 000-201 000	Nebeker *et al.*, 1974
Salmonids	Total	0.0011	-	Whole	3.9 x 106	Oliver & Niimi, 1988
Sheepshead minnow (*C. variegatus*) (juvenile)	A-1016	1.0-10	28	Whole	10 000 -30 000	Hansen *et al.*, 1975
Green algae (*Dunaliella* sp.)	A-1254	0.008	45	Lipid	477 000	Scura & Theilacker, 1977
Green algae (*Dunaliella* sp.)	A-1254	0.008	45	Dry tissue	30 000	Scura & Theilacker, 1977
American oyster (*C. virginica*)	A-1254	5.0	168	Soft parts	85 000	Ernst, 1984
American oyster (*C. virginica*)	A-1254	5.0	168	Whole	85 000	Lowe *et al.*, 1972
American oyster (*C. virginica*)	A-1254	1.0	175	Whole	101 000	Lowe *et al.*, 1972
Rotifer (*B. plicatilis*)	A-1254	0.008	45	Lipid	340 000	Scura & Theilacker, 1977

Rotifer (*B. plicatilis*)	A-1254	0.008	45	Dry tissue	51 000	Scura & Theilacker, 1977
Northern anchovy (larva) (*E. mordax*)	A-1254	0.002	45	Lipid	13 x 106	Scura & Theilacker, 1977
Pinfish (*L. rhomboides*)	A-1254	5.0	35	Whole	21 800	Ernst, 1984
Spot (*L. xanthurus*)	A-1254	1.0	56	Whole	27 800	Ernst, 1984
Spot (*L. xanthurus*)	A-1254	1.0	28	Whole	37 000	Hansen *et al.*, 1971
Spot (*L. xanthurus*)	A-1254	1.0	42	Muscle	7 600	Hansen *et al.*, 1971
Juvenile Sole (*Solea solea*)	16 PCB congeners	0.00001 - 0.00094	42	Whole (less liver)	44 000 - 370 000	Boon and Duinker, 1985

In most cases, the results shown in Table 16 (except Oliver and Niimi, 1988) were obtained from organisms exposed to the contaminants in water alone under controlled conditions; hence, they represent bio-concentration factors (BCFs) rather than bio-accumulation factors (or BAFs). The maximum BCF for marine life was found to be 13 000 000 (expressed on lipid basis) for Northern anchovy larva, which would translate to 975 000 when expressed on whole fish basis (lipid content of the fish was 7.5%) (Scura and Theilacker, 1977).

It is evident from Table 16 and the above discussion that PCBs in freshwater and marine organisms bio-concentrate to a similar degree.

7.1.4 Sediment toxicity

Sediments act as a sink for contaminants such as PCBs. The bio-availability of sediment-associated contaminants is central to whether the toxic compounds (e.g., PCBs) present in the sediment will have deleterious effects on aquatic species or will become part of the food chain.

In studying PCB availability to marine animals from the spiked sediments (containing 1.0 µg/g PCBs), McLeese *et al.* (1980) found that the bioaccumulation of PCBs in polychaetes (*Nereis virens*) and shrimp (*Crangon septemspinosa*) was directly related to the concentration of the contaminants in the sediment and inversely related to animal size; the sediment produced no toxicity among the test organisms. A bioaccumulation

factor ranging from 1.6 to <0.2 was found for polychaetes (*Nereis virens*) exposed to natural sediment containing PCBs; accumulation in clams (*Mercenaria mercenaria*) and shrimp (*Palaemonetes pugio*), on the other hand, was much less (Rubinstein *et al.*, 1983). Stein *et al.* (1987) compared the accumulation of PCBs in a benthic fish (English sole, *Parophrys vetulus*) exposed for up to 108 days to a test (2.2 µg PCBs/g dry weight) and a reference (PCBs at non-detectable level of <4.9 ng/g dry weight) sediment. English sole exposed to the test sediment had a hepatic concentration of 1.4 ± 0.6 µg PCBs/g wet weight, which was eight times greater than that for the reference sediment.

The bio-availability of PCBs from sediment is a function of several factors including concentration of the contaminants in the sediment, exposure time, species, and sediment characteristics (e.g., particle size, organic carbon, etc.). Lynch and Johnson (1982) demonstrated that increased organic matter content and a larger particle size of the natural sediment reduced the concentration of 2,2',4,4',5,5'-hexachlorobiphenyl (hexaCBP; PCB #153) in water and in the freshwater benthic amphipod (*Gammarus pseudolimnaeus*). Their results also indicated that substrate characteristics, especially the organic matter content, control the availability of hexaCBP to the overlying water and, in turn, to the aquatic organisms.

Tatem (1986) exposed freshwater prawns (*Macrobrachium rosenbergii*) and clams (*Corbicula fluminea*) to sand and sediment mixtures, containing 10%, 50%, and 100% of the sediment 5-80 (61.1 µg PCBs/g) in one set and the same proportions of the sediment 11-80 (2.3 µg PCBs/g) in the other. (The sediments 5-80 and 11-80 were dredged materials obtained, respectively, in May and November 1980 from one site in Sheboygan River, Mississippi). The mixtures containing 10% sediment produced the maximum bioaccumulation of PCBs in the organisms. The maximum values for bioaccumulation factor (BAF) were determined to be 0.9 (for Aroclor 1242 in sediment 5-80) and 2.4 (for Aroclor 1254 in sediment 5-80) for prawns, and 12.52 (for Aroclor 1242 + Aroclor 1254 in sediment 11-80) for clams.

To assess the likelihood or potential for adverse biological effects of sediment-associated toxicants (*e.g.*, PCBs) to biota, Long and Morgan (1990) assembled data derived from a wide variety of methods and approaches (*e.g.*, the equilibrium partitioning approach, the spiked-sediment bioassay approach, etc.). The data were evaluated to identify informal guidelines for use for sediments under the National Status and Trends Program. The contaminant concentrations observed or predicted by various methods to be associated with biological effects were sorted. The lower 10th percentile (*i.e.*, Effects Range-Low or ER-L, defined as a concentration at the low end of the range in which effects had been observed) and median (*i.e.*, Effects Range-Median or ER-M, defined as a concentration approximately midway in the range of

reported values associated with biological effects) values were identified. These investigators obtained ER-L and ER-M values, respectively, of 0.05 µg PCBs/g sediment and 0.4 µg PCBs/g sediment from the data used. The ER-L obtained by Long and Morgan was supported by the apparent effects threshold (*i.e.*, AET, defined as the sediment concentration of a contaminant above which statistically significant biological effects, such as depression in abundance of benthic infauna or elevated incidence of mortality in sediment toxicity tests, are always expected) for bivalve larvae for San Francisco Bay. Furthermore, the ER-M value was observed to be similar to the mean level (0.368 µg PCBs/g) in the Commencement Bay (Washington) sediment samples which were highly toxic to oyster larvae and the mean concentration (0.4 µg PCBs/g) in southern California sediments with moderate species richness.

7.2 Criteria from the literature

7.2.1 Ambient water

Criteria, objectives, and standards to protect aquatic life from the harmful effects of PCBs are shown in Table 17. The recommended criterion varied with jurisdiction. The most stringent criteria (0.0079 - 0.79 ng/L) were proposed by the U.S. Environmental Protection Agency (1980) for the protection of humans against cancer risk from consuming PCB contaminated water and aquatic organisms.

In developing its criterion based on bio-magnification of PCBs in fish, the Ontario Ministry of the Environment assumed a tolerance level of 2 µg/g wet weight in the edible tissue. This upper limit of 2 µg PCBs/g wet weight was established by Health and Welfare Canada as an action level for the sale and export of fish for human consumption. A similar approach was used by the International Joint Commission (1977), but it recommended a concentration of total PCBs in fish tissue (whole fish) not exceeding 0.1 µg/g wet weight to protect fish-consuming birds and animals. The recommended concentration of 1 ng PCBs/L in water by IJC was based on (i) Platonow and Karstad (1973) studies on commercial ranch mink where the lowest dietary concentration observed to cause deleterious effect was 0.64 µg/g Aroclor 1254, (ii) an application of a safety factor of 5, and (iii) a bio-concentration factor of 100 000. The criterion of 1 ng/L was also recommended by CCREM, Ontario, Indiana, Ohio, and Pennsylvania.

The U.S. EPA (1980) criteria for ambient waters were developed to protect freshwater aquatic life, marine aquatic life, and human health. The concentrations of 14 ng/L for freshwater and 30 ng/L for marine environments were considered too high by the EPA as they were based on bio-concentration factors measured in laboratory studies (BCFs for fish from field studies are at least 10 times higher). It was, therefore, recognised that

these criteria would provide adequate protection only against acute effects of PCBs. The Province of Manitoba adopted the U.S. EPA criterion of 14 ng/L for freshwater.

TABLE 17

Freshwater and Marine water PCB Criteria from the Literature

Criteria Statements	Criteria Values (ng/L)	Jurisdiction	Date	Reference
FRESHWATER				
Surface quality objective to protect aquatic life	14	Manitoba	1983	Williamson, 1983
Ambient water quality objective for unfiltered sample	1	Ontario	1984	OME, 1984
Water quality objective estimated to meet the recommended level in fish and aquatic life of 0.1 µg/g wet weight	1	IJC-Great Lakes	1977	IJC, 1977
Water quality guideline to protect freshwater aquatic life	1	Canada-CCREM	1987	CCREM, 1987
Water quality criteria for protection of freshwater aquatic life	14	U.S. EPA	1980	U.S. EPA, 1980
Water quality criteria for protection of human health at cancer risks of: 1:105 1: 106 and 1: 107	0.79, 0.079, 0.0079	U.S. EPA	1980	U.S. EPA, 1980
Water quality criteria for protection of aquatic life	1	Indiana, Ohio, Pennsylvania	1985	IJC, 1985

MARINE				
Saltwater quality criteria for protection of saltwater aquatic life	30	U.S. EPA	1980	U.S. EPA, 1980
Water quality criteria for protection and maintenance of marine aquatic life	10	Canada, CCME	1991	CWQG, 1991

For the protection of human health from potential carcinogenic effects of PCBs ingested from the use of contaminated water and contaminated aquatic organisms, the ambient water concentration of PCBs should be zero according to the U.S. EPA (1980). The U.S. EPA, however, recognised that this level may not be attainable at this time. As a result, PCB levels in water were recommended considering the increased risk of developing cancer in a lifetime. Based on the consumption of 2 L/d of contaminated water and 6.5 g/d of fish taken from the contaminated water, it was recommended that PCB levels in water should not exceed 0.79 ng/L, 0.079 ng/L, and 0.0079 ng/L for increased cancer risks of 1 in 100 000, 1 in 1 000 000, and 1 in 10 000 000, respectively.

Recently, the Canadian Council of Environment Ministers (CCME), formerly known as CCREM, recommended a concentration of 10 ng/L in saltwater to protect marine aquatic life (CWQG, 1991).

7.2.2 Fish and/or shellfish

The International Joint Commission (1977) recommended that the concentration of total PCBs in fish tissue (whole fish) should not exceed 0.1 µg/g wet weight to protect fish-consuming birds and animals. Health and Welfare Canada (1975) recommended the maximum tolerance level of 2 µg/g wet weight in fish (edible portion) to protect humans. This guideline was based on several factors which include maximum residue levels in all foods other than fish, economic impact to the fishing industry, and the recommended 'tolerable daily intake' of 1.0 µg/kg body weight/d for PCBs in Canada (Grant, 1983). Similar levels (2µg PCBs/g wet weight) in fish were adopted by the Ontario Ministry of Environment (1985) and the U.S. Food and Drug Administration (1984).

The objective of 0.5 µg/g (wet weight) PCBs in fish tissue was recommended for the Fraser River and Burrard Inlet in British Columbia (Swain and Holms, 1984; Nijman and Swain, 1990).

7.2.3 Sediments

The sediment criteria from various jurisdictions are shown in Table 18. A comparison among the jurisdictions is difficult to make since the guidelines are not expressed in consistent units (i.e., they are not normalised to organic carbon content). The lowest value

TABLE 18

Sediment Quality Criteria for PCBs.
(From Chu, 1989)

Criteria Statements	Values (µg PCBs/g)*	Jurisdiction	Date	References
FRESHWATER				
Interim guideline based on equilibrium partitioning(normalised to 1.0% organic carbon content)	0.03	CCREM	1989	Chu, 1989
Screening Level Concentration (Great Lakes)-interim guideline	0.036	Ontario	1988	Beak Consultants Ltd., 1988
Objective level for Fraser River (Based on background level)	0.03	British Columbia	1985	Swain and Holms, 1985
Interim guideline for in-water dredge material disposal	0.05	Wisconsin	1988	Sullivan *et al.*, 1988
Guideline for open-water dredge spoil disposal	0.05	Ontario	1976	OME, 1976
Interim median guideline normalised to 1.0% organic carbon content	0.195	EPA	1989	U.S. EPA, 1989
Guideline for Great Lakes Harbour (Region V)	10.0	EPA	1977	U.S. EPA, 1977
MARINE				

Objective level for Fraser River (Based on background level)	0.03	British Columbia	1985	Swain and Holms, 1985
Interim median guideline normalised to 1.0% organic carbon content	0.42	EPA	1989	U.S. EPA, 1989
Lowest Apparent Effects Threshold (LAET)	0.13	Washington	1988	WDE, 1988
(includes Mictotox AET); Screening Level	0.1	Washington	1987	WDE, 1988
Concentration (SLC) for	0.13	Washington	1987	WDE, 1988
Puget Sound;	0.1	Washington	1987	WDE, 1988
Apparent Effects Threshold: Puget Sound; Guideline determined using Triad method;				

* Dry weight basis

of 0.03 µg/g for total PCBs is an objective for the lower Fraser River and Estuary, Boundary Bay, and Burrard Inlet, set by the British Columbia Ministry of Environment based on measurements for uncontaminated sites. The highest value of 10 µg/g total PCBs is set by Region V of the U.S. EPA (1977); this guideline will likely be superseded by the U.S. EPA (1989) sediment criteria currently under development.

The State of Washington criteria are given in terms of Screening Level Concentration (SLC, i.e., estimated highest concentration of a non-polar contaminant that co-occurs with approximately 95% of the infauna) and AET (Table 18). The recommended SLC (0.1 µg PCBs/g sediment) and AET (0.13 µg PCBs/g sediment) for Puget Sound are similar, but twice the Effects Range-Low (ER-L) value of 0.05 µg PCBs/g sediment obtained by Long and Morgan (1990) or the AET recommended for San Francisco Bay (see Section 7.1.4). Since organic carbon content of the sediments was not stated in these references, a direct comparison is difficult to make between the criteria recommended by the State of Washington (Table 18) and the ER-L and ER-M proposed by Long and Morgan. Note that the availability of PCBs in sediment is strongly dependent upon its organic carbon content.

7.3 Recommended Criteria

7.3.1 Freshwater and marine aquatic life

For the protection of freshwater and marine aquatic life and consumers of fish and shellfish (e.g., wildlife), it is recommended that the total PCB concentration in water should not exceed 0.1 ng/L. Additionally, it is recommended that the concentration of some selective PCB congeners (e.g., PCB congeners 77, 105, 126, and 169) should not exceed levels shown in Table 19.

The recommended guidelines by CCREM (1987) and CCME (CWQG, 1991) for freshwater and marine water, respectively, are 1 ng PCBs/L and 10 ng PCBs/L.

7.3.2 Fish and shellfish

To protect wildlife dependent on aquatic life for food, it is recommended that the concentration of PCBs in fish and/or shellfish should not exceed 0.1 µg/g (wet weight).

TABLE 19

PCB congener concentration in water and PCB formulations, and recommended water quality criteria for the protection of aquatic life and consumers of fish.++

PCB congener	Concentration in PCB formulations+ µg/g (% Total PCBs)				Level in water+ng/L (% Total PCBs)	Recommended criteria (ng/L)*
	Aroclor 1254	Aroclor 1260	Aroclor 1248	Aroclor 1242		
#126	46 (0.005%)	8.3 (0.0008%)	62 (0.006%)	17 (0.0017%)		0.00025
#169	0.5 (0.5x10-4%)	0.05 (0.5x10-5%)	0.05 (0.5x10-5%)	0.05 (0.5x10-5%)		0.06
#77	600 (0.06%)	260 (0.026%)	6100 (0.61%)	5200 (0.52%)	1.0 (0.18%)	0.04
#105					0.014 (1.3%)	0.09
#123						1.0
#74					0.01 (0.9%)	21
#153	(6.1%)	(9.6%)			0.050 (4.6%)	10
#156	(0.7%)	(0.45%)			10 (1.8%)	2.2
#114						1.4

#157						0.7
#81					0.0097 (0.9%)	11
#189						12
#118	(9.5%)	(0.5%)			0.034 (3.1%)	12
#167						14
Aroclor 1254						3.0
Aroclor 1260						12.5

* based on 2,3,7,8-TCDD criterion in water of 0.1 pg/L and the maximum value of the TEF-range shown in Table 6.
+ From Oliver and Niimi, 1988; Hansen, 1987; Kannan et al., 1988; Bush et al., 1985
++ The absence of data does not necessarily mean the congener is absent; it may be below the reliable detection limit or the standard may not have been available.

7.3.3 Sediments

To protect aquatic life and consumers of aquatic life (e.g., wildlife), it is recommended that the concentration of PCBs in freshwater and marine sediments containing 1% organic carbon should not exceed 0.02 µg/g sediment (dry weight) (or 2 µg/g organic carbon, when expressed on an organic carbon basis).

7.3.4 Application of criteria

In Section 7.3.1, the recommended criteria for PCBs for fresh and marine waters are given in terms of total PCB concentration as well as some selected PCB congeners. The measurement of total PCB concentration will provide protection against the effects that may be caused by most of the congeners listed in Table 19, as long as the criterion for total PCB is met. However, the criteria recommended for congeners #77, #105, #126, and #169 are more stringent than the total PCB criterion of 0.1 ng/L. Since these coplanar congeners (i.e., PCB #77, #105, #126, and # 169) are present in most of the commercially available PCB formulations, it is recommended that they should also be measured to ensure that the PCB criteria in water are met in all respects. Both the total and congener-specific PCB criteria should be met.

7.4 Rationale

7.4.1 Freshwater and marine aquatic life

The criteria to protect freshwater and marine aquatic life from accumulating undesirable levels of PCBs in their tissue are the same, and were based upon the information presented in Platonow and Karstad (1973), Section 7.1.3, and Table 16. Considering a maximum acceptable toxicant concentration 0.1 µg PCBs/ g (wet weight) in fish consumed by wildlife (see Section 8.4), and a bio-concentration factor of ~1 000 000 for fish from water alone (which appears to be the same for both freshwater and marine environments - see Section 7.1.3), it was calculated that the concentration equal to or lower than 0.1 ng PCBs/L in water (i.e., 0.1 µg/g ÷ 1 000 000 = 0.1 ng PCBs/L) should protect fish from excessive accumulation PCBs in their tissues.

The criteria for toxic PCB congeners were based on toxic equivalent factors shown in Table 6. It was assumed that the maximum level for 2,3,7,8-TCDD in water is not to exceed 0.1 pg/L, the recently set water quality criterion by the Ontario Ministry of Environment (Lupp and McCarty, 1989). The congener-specific criteria (based on toxic equivalent factors) and concentrations in water from various sources are shown in Table 19.

The criterion of 0.1 ng/L total PCBs recommended in this document either exceeds slightly or is several orders of magnitude above the recommended concentrations for congeners #77, #105, #126, and #169 in Table 19. Although these congeners are very toxic, their concentrations are generally low in natural waters as well as in the most common PCB formulations (Table 19). For instance, PCB # 105 constituted, at the maximum level, 1.3% of the total PCB in water. At the recommended level of 0.1 ng/L total PCB, the concentration of congener #105 would be 0.006 ng/L which is at least an order of magnitude lower than the congener criterion derived in Table 19. However, for congener #77, the concentration in water may exceed the criterion for total PCBs.

No rationale, in clear terms, was provided by CCREM (1987) in setting their criterion of 1 ng PCBs/L for the protection of freshwater aquatic life. However, the interim guideline of 10 ng PCBs/L for the protection and maintenance of marine aquatic life was based on the application of a safety factor of 0.1 to the lowest observed effect level (0.16 µg/L) observed with *C. variegatus* in a 21-d study (Schimmel *et al.*, 1974). Bioaccumulation models, as in this document, were not used by the CCME (CWQG, 1991)

7.4.2 Fish and shellfish

The criterion (0.1 µg PCBs/g - wet weight - see Section 8.4) for fish and shellfish was based on adverse effects in mink fed 0.64 µg PCBs/g in their diet (containing meat from cows which had been fed Aroclor 1254 (Platonow and Karstad, 1973). The maximum residue level (*i.e.*, 0.1 µg PCBs/g - wet weight) recommended in this document for the

protection of wildlife is one-sixth the lowest concentration (in the mink diet) used in the Platonow and Karstad study.

The International Joint Commission (1977) guideline of 0.1 µg PCBs/g (wet weight) in whole fish, to protect fish-consuming birds and animals, is also based on the Platonow and Karstad (1973) study on minks. As stated above in this document, an application factor of 5 was applied by the IJC to obtain the recommended guideline of 0.1 µg PCBs/g in fish.

7.4.3 Sediments

Several methods have been proposed in the literature to derive sediment quality guidelines. The sediment criterion recommended in this document is an average value (geometric mean) based on the results obtained using these approaches. The results of equilibrium partitioning (sediment to water and sediment to biota) of PCBs in the environment are shown below. Other approaches include Apparent Effect Threshold (AET), Screening Level Concentration (SLC) and Triad. These approaches employ all relevant data available from the literature. The sediment quality criteria developed by the State of Washington (Table 18) are based on these approaches.

(a) Partitioning of PCBs between Water and Sediment

It was assumed that PCBs sorbed on sediment are inactive and the toxic fraction of PCBs is the one associated with interstitial water. The U.S. EPA (1989) suggested the following relationship between sediment quality criteria (SQC expressed as µg PCB/kg organic carbon) and water quality criteria (WQC expressed as µg PCB /L):

$$SQC = K_{ow}\ WQC$$

where K_{ow} is the octanol-water partition coefficient for PCBs. Given that the average value for K_{ow} for most common PCB formulations (*e.g.*, Aroclors 1016, 1248, 1254 and 1260) is 2.19 x 106 (log K_{ow} = 6.34 - MacKay, 1982) and WQC = 0.0001 µg/L (section 7.3.1), the sediment (for freshwater as well as marine) criterion was calculated to be 0.22 µg PCBs/g organic carbon or 2.2 ng PCBs/g for sediment containing 1% organic carbon.

(b) Partitioning of PCBs between Biota and Sediment

The following results were obtained from partitioning of PCBs between sediment and biota or bio-concentration of PCBs in animal tissues from sediments. The information presented in Tatem (1986) was used in determining the maximum level of PCBs in

sediment, using equilibrium partitioning between sediment and benthic organisms. The maximum BAF for freshwater clams (*Corbicula fluminea*) exposed to PCBs (Aroclor 1254) in the sediment plus sand mixture (containing 0.111 µg PCB/g dry weight and 0.375% organic carbon) was determined to be 12.5. Assuming the maximum desirable concentration of PCBs in fish/shellfish to be 0.1 µg/g wet weight (see Section 7.4.2), it was calculated that the maximum concentration of PCBs in the sediment should not exceed (0.1 µg/g ÷ 12.5) = 0.008 µg/g dry weight. The results, when expressed in terms of sediment containing 1% organic carbon, would yield the maximum desirable concentration of 0.008 ÷ 0.375 = 0.021 µg PCBs/g-sediment or 21 ng PCBs/g-sediment (containing 1% organic carbon).

(c) Discussion

The two partitioning approaches, (a) and (b) above, yielded sediment PCB criteria of 0.0022 and 0.021 µg PCBs/g-sediment; the geometric mean of the two was determined to be 0.007 µg PCBs/g-sediment. The upper limit (*i.e.*, 0.021 µg PCBs/g-sediment containing 1% organic carbon) of the range obtained using the two partitioning models, is similar to the criteria proposed by CCREM (Chu, 1989), and the objectives proposed for Fraser River and tributaries in British Columbia (Swain and Holms, 1985) (Table 18). (Note that later measurement showed that sediment from the lower Fraser River had an organic carbon content of about 1%.). The National Oceanic and Atmospheric Administration (NOAA) arrived at a guideline (ER-L = 0.05 µg PCB/g sediment - see section 7.1.4), based on all available data, which was about 7 times higher than the geometric mean obtained from the partitioning approach (Long and Morgan, 1990); however, no reference to the organic carbon content of sediment was made in their analysis.

A direct comparison between the sediment criteria obtained above (*i.e.*, in Sections 7.4.3a and 7.4.3b) and those proposed by the State of Washington for Puget Sound (Table 18) is difficult to make since the organic carbon content for sediments was not stated in the Washington State criteria. Note, that the State of Washington used several approaches to derive its criteria (*e.g.*, apparent effects threshold, screening level concentration, and triad).

The criterion of 0.02 µg PCBs/g sediment (freshwater and marine), containing 1% organic carbon, was adopted in this document for two reasons: (a) Sediments containing less than or equal to 0.02 µg PCBs/g (dry weight) are not likely to cause adverse effects on aquatic organisms, and (b) this value represents the mean (geometric) of values obtained using different methods (*e.g.*, partitioning approaches outlined in Sections 7.4.3a and 7.4.3b above, equilibrium partitioning approach used by

Chu (Table 18), and the approach used by Long and Morgan (1990) in defining ER-L- see Section 7.1.4).

8. WILDLIFE

8.1 Effects

Most of the literature on toxicity of PCBs to wildlife concerns the consumption of PCBs in their diet. In reviewing data gathered from various sources, Eisler (1986) noted that, as a group, birds were more resistant to acutely toxic effects of PCBs than mammals. LD_{50}s ranging from 604 to more than 6 000 mg Aroclor/kg of diet were reported for various species of birds. Also, for all avian species, PCB residues of 310 mg/kg fresh weight of the bird were associated with an increased likelihood of death from PCB poisoning,

Among mammals, the mink (*Mustela vison*) is the most sensitive wildlife species tested. Diets containing 6.7 mg Aroclor 1254/kg fresh weight and 8.6 mg Aroclor 1242/kg fresh weight killed 50% of the mink in 9 months (Ringer 1983). In comparing primary toxicity (where animals were fed a diet containing a PCB formulation) and secondary toxicity (where the diet contained the same concentrations of the metabolised xenobiotic or PCBs in this case) of Aroclor 1254 to mink, Aulerich *et al.* (1986) found that the mean feed consumption and body weight gains were lower for the animals fed metabolised Aroclor 1254 (secondary toxicity) than for animals that received the same concentrations of the technical grade of Aroclor 1254 (primary toxicity). The tests yielded 28- and 35-day LC_{50} values, respectively, of 79.0 and 48.5 mg Aroclor 1254/kg fresh weight of feed for the primary toxicity test and 47.0 and 31.5 mg/kg fresh weight of feed for the secondary toxicity test.

Signs of PCB poisoning in mink include anorexia, weight loss, lethargy, and un-thrifty appearance. In studying sublethal effects, Aulerich *et al.* (1985) found that diets supplemented with as little as 2 mg Aroclor 1254/kg of feed for 8 months, or 5 mg Aroclor 1254/kg of feed for 4 months, resulted in near reproductive failure with normal breeding and whelping, but a high death rate of kits; reproduction was not affected at dietary levels of 1.0 mg Aroclor 1254/kg. Two of the 12 female minks died in 129 days, when exposed to a PCB level of 0.64 mg/kg fresh weight in a diet containing meat from cows which had been fed Aroclor 1254 (Platonow and Karstad, 1973). Death was not reported in the mink population fed a control commercial diet containing 0.3 mg PCBs/kg. Although poor reproduction at the rate of 1.81 kits/female bred (satisfactory production is considered to be 4 kits/female) was noted by these investigators, lesions

referable to the PCB content of the control ration were not seen in the mink of this herd, which were used for research on aleutian disease during the course of these PCB experiments.

The data relating toxicity of individual PCB congeners to wildlife are limited. Recently, it has been shown that certain hexachlorobiphenyls, such as 3,4,5,3',4',5'-hexachlorobiphenyl (PCB #169), are extremely toxic to mink ; concentrations as low as 0.1 mg/kg fresh weight in their diet killed 50% of the animals exposed to the congener. However, other hexachlorobiphenyls, such as 2,4,5,2',4',5'-hexachlorobiphenyl (PCB #153) and 2,3,6,2',3',6'-hexachlorobiphenyl (PCB #136) were non-fatal to mink under similar conditions, and did not produce adverse reproductive effects (Aulerich *et al.*, 1985). Note that PCB #169 is a coplanar biphenyl while the other two hexachlorobiphenyls are not.

8.2 Criteria from the Literature

The Province of Manitoba recommended a surface water quality objective of 0.014 µg total PCB/L to protect wildlife from the adverse effects of PCBs. This guideline is the same as suggested by the US EPA (1980) for freshwater aquatic life, to provide protection for fish-consuming wildlife against excessive accumulation of PCBs in fish.

Based on an acceptable level of 0.1 µg PCB/g wet weight in whole fish for the protection of fish-consuming birds (and a bio-concentration factor of 1 x 105), the IJC (1977) estimated that a concentration of 0.001 µg PCBs/L in water would be required to reach the recommended fish tissue level. The safe level of 0.1 µg PCBs/g in whole fish was derived, in part, from the Platonow and Karstad (1973) study, where the lowest dietary concentration observed to cause a deleterious biological effect was 0.64 µg Aroclor 1254/g (see Section 8.1). A safety factor of five was applied to obtain the recommended level for fish tissue (whole fish) of 0.1 µg PCBs/g wet weight. Note that, in latter reviews (Eisler, 1986), birds were found to be more resistant to PCBs than mammals.

No other jurisdiction recommended levels for PCBs in water for the protection of wildlife.

8.3 Recommended Criteria

Criteria for PCBs in waters consumed by wildlife are not recommended at this time.

To protect wildlife (e.g., mink) from harmful effects of PCBs in the diet, it is recommended that the concentration of total PCBs in fish and shellfish should not exceed 0.1 µg/g wet weight in whole fish and shellfish.

8.4 Rationale

Criteria for PCBs in waters consumed by wildlife are not recommended at this time, primarily due to the lack of pertinent information (e.g., the toxicity of PCBs in water to wildlife). As with human drinking water (section 6) and livestock (section 9.2), PCB levels in ambient waters consumed by wildlife were considered to pose an insignificant threat to wildlife.

For waters inhabited by aquatic life, the criterion recommended for the protection of aquatic life and their consumers appears more than adequate to protect wildlife.

The major source of PCBs in wildlife is food (e.g., fish and shellfish). The criteria for PCBs in fish consumed by wildlife are primarily based on the results of the Platonow and Karstad (1973) study with minks. Platonow and Karstad observed some adverse effects in female minks, fed a diet (0.64 µg PCBs/g) containing meat from cows exposed to PCBs (Aroclor 1254). In this document, a rounded figure of 0.1 µg PCBs/g wet weight in whole fish was chosen to be the acceptable level for fish-consuming wildlife (see Section 7.4.2 for more detail). This concentration is more than 5 times lower than the lowest observed effect level (LOEL) noted by Platonow and Karstad (1973) with minks.

The CCREM (1987) did not recommend a guideline for the protection of wildlife.

9. LIVESTOCK WATERING

9.1 Effects

Most of the data on livestock were on the effects of PCBs in their feed. Hansen *et al.* (1975) found adverse effects in third-litter sows which were fed 20 µg Aroclor 1254/g of feed (wet weight) throughout the gestation and nursing periods. Treated sows also had more mummified foetuses. These investigators found that the total PCB levels in blood, brain, kidney, and ovarian tissues were less than 0.5 mg/kg (wet weight), but ranged from 4 to 20 mg/kg (wet weight) in the fat tissue.

Chickens exposed to 2 µg Aroclor 1254/g in their diet for 14 weeks showed a reduction in number of eggs laid (Platonow and Reinhart, 1973). PCBs (*e.g.*, Aroclor 1254) at a concentration as low as 0.1 µg/g in the feed have also been shown to alter enzyme levels in the liver of chicken and quail (Srebocan *et al.*, 1977). Strachan (1988) suggests that these effects are probably reversible and are not necessarily adverse in themselves to the well-being of the individual birds. They are, however, symptomatic of birds under stress and consequently, PCB dosages causing stress should be considered adverse.

No data were found in the literature regarding toxicity of PCBs to livestock from drinking water. The concentration of PCBs in ambient waters generally ranges from low to undetectable (section 4.1.1).

9.2 Criteria from the Literature

No criterion for livestock watering was recommended in the literature.

9.3 Recommended Criteria

Criteria for PCBs in waters consumed by livestock are not recommended at this time.

9.4 Rationale

Criteria for PCBs in waters consumed by the livestock are not recommended at this time for two reasons: (i) the lack of pertinent information (*e.g.*, the toxicity of PCBs in water to livestock), (ii) the concentration of PCBs in ambient waters generally ranges from low to undetectable (section 4.1.1). As a result, it is believed that an exposure to the ambient waters (with PCB concentrations similar to those recommended for the protection of aquatic life) will not contribute significantly to PCB residues in livestock. This argument is further supported by a worst-case scenario presented in Table 20, which suggests that a fairly long-term exposure to contaminated waters is necessary to accumulate PCBs in livestock tissue to the level recommended by Health and Welfare Canada.

TABLE 20

Accumulation of PCBs in livestock tissue from drinking water.

Assumptions:

- Total PCBs concentration in livestock drinking water = 10 ng/L, (This concentration is 10 times higher than the CCREM (1987) guideline for the protection of freshwater aquatic life).
- Maximum tolerable level of PCBs in beef fat = 0.2 µg/g - as recommended by Health and Welfare Canada (Grant, 1983).
- Mean daily water consumption by 450 kg beef cattle = 60 L
- Mean fat concentration in beef cattle = 10% (of live weight)
- The PCBs in drinking water consumed by the livestock are retained in the fat tissue without any loss from the animals.

Daily accumulation of PCBs in the fat tissue = (10 ng/L) x (60 L/d) / (450 kg live weight x 0.10 fat/live weight x 1000 g/kg) = 0.0133 ng PCBs/g/d

Time required to accumulate levels equal to the guideline of 0.2 µg PCBs/g-fat = (0.2 µg/g) x (1000 ng/µg) / (0.0133 ng/g/d) x (365 d/a) = 41 years

10. IRRIGATION

10.1 Effects

PCBs have a strong affinity for soils. The clay and organic matter contents of the soil, as well as the chlorine content and hydrophobicity of the individual PCB isomers, tend to increase PCB adsorption to soils (Fairbanks and O'Connor, 1984).

The most frequently encountered PCBs in soils are similar in composition to Aroclor 1254 and Aroclor 1260 (Richardson and Waid, 1979). The major means of loss of PCBs (e.g., Aroclor 1254) from soils is by volatilisation. Sewage sludge amendments have been shown to reduce volatilisation while increasing the degradation of PCBs (Fairbanks *et al.*,1987).

Plants grown in PCB-amended soils have been shown to accumulate these compounds in their aerial parts (Sawhney and Hankin, 1984; Suzuki *et al.*, 1977). In a recent study, PCB concentrations of 0.01 to 0.2 µg/g (fresh weight) were detected in leaves from purple loosestrife (*Lythrum salicaria*) growing in a contaminated soil (0.12 µg PCBs/g); the major route of entry was via the roots (Bush *et al.*, 1986). However, the mechanism of PCB translocation from soil to the plant foliar tissue is controversial. Several investigators have concluded that the level of PCBs in the foliar tissues of soybean, broad bean, tomato, and cucumber plants is due to vapour transport from the

soil, rather than to translocation through the plant (Fries and Marrow, 1981; Buckley, 1982; Bacci and Gaggi; 1985).

The toxicity of PCBs to terrestrial plants has not been studied extensively. Since PCBs are lipophilic and less mobile, their effects are difficult to monitor in the whole plant. A few studies have shown that PCBs in soils can interfere with the growth of the plant when applied at a concentration much higher than observed in the environment. For instance, Strek *et al.* (1981) noted a significant decrease in height, fresh top weight, and inhibition of cumulative water use in soybean and beet root at 1000 µg/g Aroclor 1254; however, corn and sorghum plants were unaffected.

Livestock can become exposed to PCBs applied to land by ingestion of contaminated soil when grazing. Soil consumption by grazing dairy cows can be as high as 14% of dry-matter intake when the amount of available forage is low and no supplemental feed is used (Healy, 1968; Fries, 1982). Using a worst-case scenario, Fries (1982) noted that 1.0 µg/g of PCBs in surface soil could cause milk-fat residues of 0.7 µg PCBs/g in dairy cattle, and tissue (edible) residues of 0.23 µg PCBs/g in beef cattle. It was assumed that the steady-state milk-fat concentrations are about five times the diet concentrations (dry weight) in dairy cattle, and that the steady-state body fat concentration will be similar in non-lactating animals. Note that the administrative guideline established by Health and Welfare Canada for milk or dairy products and beef is 0.2 µg/g (fat basis) (Grant, 1983).

10.2 Criteria from the Literature

Criteria for PCBs in water used for irrigation were not found in the literature. On the other hand, various jurisdictions have guidelines for cleanup of soils contaminated with PCBs from spills (Table 21). The guidelines shown in Table 21 suggest that the urgency for remediation is a function of PCB concentration in contaminated soils. Furthermore, it would appear that PCBs in soil would pose a minor threat to the environment as long as their concentration is <1.0 µg/g.

10.3 Recommended Criteria

It is recommended that the total PCB concentration in irrigation water should not exceed 0.5 µg/L.

10.4 Rationale

The PCB criterion for irrigation water was designed to limit undesirable accumulation of PCBs in soils in an agricultural environment. The information presented in Table 21 and

the results obtained by Fries (1982) in his study concerning the ingestion of PCB-contaminated soil and its effect on livestock formed the basis of the criterion. A PCB concentration of 0.3 µg/g-soil (dry weight) was assumed to be the safe level in this document.

TABLE 21

PCB Criteria for Contaminated Soil Cleanup

Criteria Statements	Criteria Values (µg PCBs/g - dry weight)	Jurisdiction	Date	Reference
Level considered to be contaminated; Recommended target level for cleanup	> 5.0 1.0	Quebec Quebec	1985 1984	Bernier (1985) QME (1984)
Target level for cleanup	< 5.0	Saskatchewan	1985	Chan (1985)
guideline for further investigation of contamination; guideline for urgent remediation; target level for cleanup of residential area	1.0 10.0 1.0 to 5.0	Holland Holland Holland	1983 1983 1985	NMHPE (1983) NMHPE (1983) Beaulieu (1985)
Investigation level (level A) for residential, recreational, & agricultural land use; Remediation level (level B) for residential, recreational, & agricultural land use; Remediation level (level c) for commercial or industrial land use	0.1 5.0 50.0	British Columbia	1989	BC MOE (1989)
guideline for further investigation;	1.0	France	1985	Beaulieu (1985)

guideline for remediation; guideline for urgent remediation	5.0 10.0			
TSCA regulation for cleanup of spills <1 lb PCBs; TSCA regulation for cleanup of high-conc. spill or low-conc. spill of 1 lb PCBs in outdoor electrical substation; TSCA regulation for cleanup of high-conc. spill of 1 lb PCBs in restricted access areas; TSCA regulation for cleanup of high-conc. spill or spill of 1 lb PCBs in non-restricted access areas	< 1.0 25.0 or 50.0 + notice 25.0 10.0 + excavation of top 25 cm; 1.0 for replacement soil	U.S. EPA	1987	U.S. EPA (1987)

Given (i) the bulk density of a soil to be 1500 kg/m^3, (ii) the concentration of PCBs in irrigation water at 0.5 µg/L , (iii) the irrigation rate of 1.0 $m^3/m^2/a$, and (iv) the PCBs in irrigation water to be retained in the top 0.15 m of the soil, the soil in question will accumulate PCBs at the rate of:

= (0.5 µg PCBs/L) x (1.0 $m^3/m^2/a$) x (1/0.15 m) x (1 m^3/1500 kg-soil) x (1000 L/m^3) = 2.2 µg PCBs/kg-soil/a;

provided there is no loss of PCBs from the soil. At this rate it will take at least (300 µg PCBs/kg-soil) / (2.2 µg PCBs/kg-soil/a) = 136.4 years for the soil to accumulate PCBs to the assumed safe level of 0.3 µg/g.

In practice, PCB levels of this magnitude (*i.e.*, 0.5 µg/L) will not be encountered in irrigation water, and for the majority of cases the much lower criteria for the protection of aquatic life (0.1 ng/L) would apply to waters used for irrigation.

Note that the degradation of PCBs in the soil was not considered in the above calculations. This lack of consideration for degradation combined with the fact that PCB levels in ambient waters will generally be determined by the much lower aquatic life

criterion, will result in a period, for PCBs to accumulate in soil to a safe level, much longer than that shown above (*i.e.*, 136.4 years).

11. RECREATION AND AESTHETICS

11.1 Effects

Besides through food and air, PCBs can enter the bodies of humans and animals through skin contact (Maroni *et al.*, 1981; Fishbein, 1974). However, the data quantifying the contribution of a dermal exposure to PCBs in an occupational environment or in recreational waters are scant in the literature.

Jan and Tratnik (1988) studied the effects on members of the population living in the vicinity of (<1 km from the river; Group A) and away (1 - 3 km from the river; Group B) from the Krupa River (Slovenia, Yugoslavia) contaminated with PCBs (0.3 µg/L). They concluded that dermal exposure from bathing and clothing washed in the river water was the primary cause of high PCB levels in the blood of the Group A population (PCBs are trapped on the fibres of underwear during washing, and the underwear acts as a transfer agent for PCBs between the water and skin). Twenty-five months after ceasing to use the river for bathing and washing, the PCB levels declined in human blood from 247 to 15 ng/g (mean of three samples) and in human skin fat from 295 to 12 µg/g (mean of two samples). The PCB concentration in fish caught from the Krupa River was 117 µg/g, but the residents of the area did not eat fish from the river.

In examining the health and PCB levels in the blood of electrical workers, Maroni *et al.* (1981) found that mean PCB (trichlorobiphenyl) concentrations were 215 ng/g (range 77 - 407 ng/g) in the workers with abnormal liver findings and 92 ng/g (range 13 - 345 ng/g) in those without abnormal liver findings. Based on these data, one would expect ill effects in some residents near the Krupa River (see above). However, note that evaluation of hepatic findings (i.e., abnormal liver findings) in the Maroni study were complicated by: (a) the small number of individuals showing hepatic abnormality (16 cases out of 80), (b) lack of association between severity of the hepatic effects and duration of exposure, and (c) unrelated health problems that may have contributed to the hepatic effects in three of the workers (US PHS, 1989).

Other concerns for PCBs in waters used for recreational purposes are expected to be the same as those for drinking water.

11.2 Criteria from the Literature

The criteria of 0.1 µg/L PCBs and 0.001 µg/L PCBs for recreational water were recommended by the Province of Quebec in Canada and the State of Indiana in U.S.A., respectively (Trépanier, 1984; IJC, 1985). Criteria from other jurisdictions, including the CCREM, were not found in the literature.

The criterion of 0.001 µg PCBs/L for recreational waters recommended by the State of Indiana pertains to providing protection for fish-eating birds, rather than humans swimming in it. The recreational water criterion of 0.1 µg PCBs/L quoted for Quebec (Trépanier, 1984) is out-dated; currently, a PCB criterion for recreational waters has not been recommended by Quebec.

11.3 Recommended Criteria

Criteria for PCBs in waters used for recreational purposes are not recommended at this time.

11.4 Rationale

Criteria for PCBs in waters used for recreational activities are not recommended in this document primarily for the lack of pertinent information.

In practice, the PCB criterion recommended for the protection of aquatic life (0.1 ng/L) would also apply to waters used for recreation.

12. RESEARCH and DEVELOPMENT NEEDS

Several research needs, as noted below, were identified during preparation of this document.

1. PCBs have been shown to be carcinogenic. However, in the literature, the doses used to study carcinogenicity of these chemicals in animals are relatively much larger than those to which animals may be exposed in the environment. Furthermore, it is not clear from the studies conducted to date how the information (*e.g.*, carcinogenicity to animals) generated using rodents can be used to predict carcinogenicity in humans. More discriminating studies are required to study these aspects.

2. Commercial PCB formulations are mixtures of PCB isomers and congeners. Several of these congeners (co-planars as well as non co-planars) are very toxic to terrestrial

and aquatic animals. Research on congener-specific toxicity, bioaccumulation, and environmental levels is needed for establishing congener-specific guidelines or criteria.

3. Toxicity equivalency (with respect to 2,3,7,8-TCDD) for various PCB coplanar congeners has been established using AHH and EROD enzyme induction in hepatoma cell cultures of terrestrial animals. The toxicity equivalency factors, based on the enzyme induction in terrestrial animal cells, need to be checked for their application in aquatic environments.

4. Several organochlorines, including PCBs, furans and dioxins, have shown a good correlation between *in vitro* induction of the AHH and EROD enzyme activity in rat hepatoma cells and several receptor-mediated *in vivo* responses (*e.g.*, body weight loss, thymic atrophy, etc.). The big question, however, still remains to be answered; *i.e.*, should a PCB concentration in water, however small, be considered toxic if it results in the induction of enzyme activity? The relationship between induction of the enzyme (*e.g.*, EROD) activity and long-term toxicity of PCBs to aquatic animals exposed to the same concentration in water needs to be addressed.

5. Recently, an approach based on structure-activity relationships or toxicity equivalency to 2,3,7,8-TCDD has been used to assess the contamination potential of various organochlorines (*e.g.*, PCBs, furans, and dioxins). It is generally assumed that the toxicities (expressed as toxic equivalent of 2,3,7,8-TCDD) of various organochlorines are independent of one another and that the toxicity of all congeners for a given group of organochlorines is additive. Recent investigations have shown that PCB mixtures such as Aroclor 1254 are a dioxin antagonist in rat hepatoma cells. If it is true, then summation of toxic equivalents cannot be a viable approach for estimating the toxic significance of these chemicals in the environment. Interaction between organochlorines and their isomers and/or congeners needs more research.

6. More research is needed on PCB effects (toxicity and accumulation) on humans from primary-contact recreational waters, although this is likely to be of low priority in British Columbia.

13. REFERENCES

Adams, J. A., and Haileselassie. 1984. The effects of polychlorinated biphenyls (Aroclor 1016 and 1254) on mortality, reproduction, and regeneration in *Hydra oligactis*. Arch. Environ. Contam. Toxicol. 13: 493-499.

Adams, J. A., and Slaughter-Williams, S. 1988. The effects of PCB's (Aroclor 1254 and 1016) on fertilisation and morphology in *Arbacia punctulata*. Water, Air, and Soil Pollut. 38: 299-310.

Andersson, K., A. Norstrom, C. Rappe, B. Rasmuson, and H. Swahlin. 1973. Photochemical degradation of PCB's. National Meeting of the American Chemical Society, Aug. 26-31, Chicago, Illinois (Cited from Moore and Ramamoorthy, 1984).

Addison, R. F., M. E. Zinck, and D. E. Willis. 1978. Induction of hepatic mixed-function oxidase (MFO) enzymes in trout (*Salvelinus fontinalis*) by feeding Aroclor 1254 or 3-methylcholanthrene. Comp. Biochem. Physiol. 61C: 323-325.

Ankley, G. T., V. S. Blazer, R. E. Reinert, and M. Agosin. 1986. Effects of Aroclor 1254 on cytochrome P-450-dependent mono-oxygenase, glutathione s-transferase, and UDP-glucuronosyltransferase activities in channel catfish liver. Aquat. Toxicol. 9: 91-103.

Aulerich, R. J., R. K. Ringer, and J. Safronoff. 1986. Assessment of primary vs. secondary toxicity of Aroclor 1254 to mink. Arch. Environ. Contam. Toxicol. 15: 393-399.

Aulerich, R. J., S. J. Bursian, W. J. Breslin, B. A. Olson, and R. K. Ringer. 1985. Toxicological manifestations of 2,4,5,2',4',5'-, 2,3,6,2',3',6'-, and 3,4,5,3',4',5'-hexachlorobiphenyl and Aroclor 1254 in mink. J. Toxicol. Environ. Health 15: 63-79 (Cited from Eisler, 1986).

Bacci, E., and C. Gaggi. 1985. Polychlorinated biphenyls in plant foliage: translocation or volatilisation from contaminated soils? Bull. Environ. Contam. Toxicol. 35: 673-681.

Ballschmiter, K., and M. Zell. 1980. Analysis of polychlorinated biphenyls (PCB) by glass capillary chromatography. Fresenius' Z. Anal. Chem. 302: 20

Basturk, O, M. Dogan, I. Salihoglu, and T. Balkas. 1980. DDT, DDE, and PCB residue in fish, crustaceans and sediments from eastern Mediterranean coast of Turkey. Marine Pollut. Bull. 11: 191-195 (Cited from Moore and Ramamoorthy, 1984).

Baxter, R. A., P. E. Gilbert, R. A. Lidgett, J. H. Mainprize, and H. A. Vodden. 1975. The degradation of polychlorinated biphenyls by micro-organisms. The Science of the Total Environment 4: 53-61 (Cited from Moore and Ramamoorthy, 1984).

Beak Consultants Ltd. 1988. Development of sediment quality guidelines. Phase II - Guideline development. Report to Ontario Ministry of Environment, Toronto, Ontario. Prepared by D. R. Hart, J. Fitchko, and P. M. McKee. (Sept. 88) (Cited from Chu, 1989).

Beaulieu, M. 1985. Ministère de l'environment de Québec, personal communication. (Cited from Environment Canada, 1987).

Bernier, A. 1985. Environment Canada, EPS Quebec Region, personal communication. (Cited from Environment Canada, 1987).

Boon, J. P., and J. C. Duinker. 1985. Kinetics of polychlorinated biphenyl (PCB) components in juvenile sole (*Solea solea*) in relation to concentrations in water and to lipid metabolism under conditions of starvation. Aquat. Toxicol. 7: 119-134.

Borgmann, U., W. P. Norwood, and K. M. Ralph. 1990. Chronic toxicity and bioaccumulation of 2,5,2',5'- and 3,4,3',4'-tetrachlorobiphenyl and Aroclor 1242 in the amphipod *Hyalella azteca*. Arch. Environ. Contam. Toxicol. 19: 558-564.

Bridgham, S. D. 1988. Chronic effects of 2,2'-dichlorobiphenyl on reproduction, mortality, growth, and respiration of *Daphnia pulicaria*. Arch. Environ. Contam. Toxicol. 17: 731-740.

British Columbia Ministry of Environment (BC MOE). 1989. Criteria for managing contaminated sites in British Columbia. Draft (#6) Report. Waste Management Program, Ministry of Environment. No. 21, 1989.

Bryan, A. M., and P. G. Olafsson. 1978. The effect of polychlorobiphenyls (Aroclor 1242) on bicarbonate-C14 uptake by *Euglena gracilis*. Bull. Environ. Contam. Toxicol.19: 374-381.

Bryan, A. M., W. B. Stone, and P. G. Olafsson. 1987. Disposition of toxic PCB congeners in snapping turtle eggs: Expressed as toxic equivalents of TCDD. Bull. Environ. Contam. Toxicol. 39: 791-796.

Bryan, J. E., and E. V. Jensen. 1991. *In:* Memorandum to N.K. Nagpal from E.V. Jensen, Environmental Protection, Ministry of Environment, Penticton, BC

Buckley, E. H. 1982. Accumulation of airborne polychlorinated biphenyls in foliage. Science 216: 520-522.

Bush, B., K. W. Simpson, L. Shane, and R. R. Koblintz. 1985. Bull. Environ. Contam. Toxicol. 34: 195 (Cited from Hansen, 1987. *In* : S. Safe (ed.) Polychlorinated biphenyls (PCBs): Mammalian and environmental toxicology).

Bush, B., L. Shane, L. R. Wilson, E. L. Barnard, and D. Barnes. 1986. Arch. Environ. Contam. Toxicol. 15: 285 (Cited from Hansen, 1987. *In* : S. Safe (ed.) Polychlorinated biphenyls (PCBs): Mammalian and environmental toxicology).

Canadian Council of Resource and Environment Ministers (CCREM). 1986. The PCB Story. p 1-32. 60 Bloor St. West, Suite 701, Toronto, Ontario, Canada, M4W3B8.

Canadian Council of Resource and Environment Ministers (CCREM). 1987. Canadian water quality guidelines.

Canadian Water Quality Guideline (CWQG). 1991. Canadian Council of Environment Ministers (CCME) recommended guidelines Appendix VII. Updates (April 1991).

Carey, A. E., and J. A. Gowen. 1976. PCBs in agricultural and urban soils. In: Proc. of National Conference on PCBs. Nov. 19-21, 1975, Chicago, Illinois. p 195-198. EPA-560/6-75-004 (Cited from Garrett, 1985)

Chan, V. 1985. Saskatchewan Ministry of Environment, personal communication (Cited from Environment Canada, 1987).

Chu, B. C 1989. Partition-based evaluative models in the development of Canadian sediment quality guidelines. Water Quality Objectives Division, Water Quality Branch, Environment Canada. 20 p.

Clark, R. R. 1979. Degradation of polychlorinated biphenyls by mixed microbial cultures. Appl. Environ. Microbiol. 37: 680-689 (Cited from Moore and Ramamoorthy, 1984).

Cooley, N. R., J. M. Keltner Jr., and J. Forester. 1972. Mirex and Aroclor 1254: Effect on and accumulation by *Tetrahymena pyriformis* Strain W. J. Protozoology. 19: 636-638.

Cosper, E. M., B. J. Snyder, L. M. Arnold, L. A. Zaikowski, and C. F. Wurster. 1987. Induced resistance to polychlorinated biphenyls confers cross-resistance and altered environmental fitness in a marine diatom. Marine Environ. Res. 23: 207-222.

Cutkomp, L. K., H. H. Yap, D. Desaiah, and R. B. Koch. 1972. The sensitivity of fish ATPases to polychlorinated biphenyls. Environmental Health Perspectives. 1: 165-168.

Dalla Venezia, L., and V. U. Fossato. 1984. Effects of PCBs on *Leander adspersus*: toxicity, bioaccumulation, oxygen consumption, osmo-regulation. FAO/UNEP Meet. on the Toxicity and Bioaccumulation of Selected Substances in Marine Organisms, Rovinj (Yugoslavia), 3-9 Nov., 1984. p 39-49.

DeFoe, D. L., G. D. Veith, and R. W. Carlson. 1978. Effects of Aroclor 1248 and 1260 on the fathead minnow (*P. promelas*). J. Fish. Res. Board Can. 35: 997-1002.

Dill, D. C., M. A. Meyers, C. G. Mendoza, G. V. Boggs, and J. A. Emmitte. 1982. Comparison of toxicities of biphenyl, monochlorobiphenyl, and 2,2',4,4'-tetrachlorobiphenyl to fish and Daphnids. *In* : J.G. Pearson, R.B. Foster, and W.E. Bishop (eds) Aquatic Toxicology and Hazard assessment Fifth Symposium, STP 766, Amer. Soc. Test. Mater., Philadelphia. p 245-256 (Cited from Bridgham, 1988).

Edgren, M., M. Olsson, and L. Reutergardh. 1981. A one year study of the seasonal variations of DDT and PCB levels in fish from heated and unheated areas near a nuclear power plant. Chemosphere 10: 447-452 (Cited from Moore and Ramamoorthy, 1984).

Eisler, R. 1986. Polychlorinated biphenyl hazards to fish, wildlife, and invertebrates: A synoptic review. Biological Report 85(1.7), Contaminant Hazard Reviews Report No. 7, Fish and Wildlife Service, U.S. Department of the Interior, 72 p.

Elder, V. A., B. L. Proctor, and R. A. Hites. 1981. Organic compounds found near the dump sites in Niagara Falls, New York. Environ. Sci. Technol. 15: 1237-1243 (Cited from Moore and Ramamoorthy, 1984).

Ernst, W. 1984. Pesticides and technical organic chemicals. *In* : O. Kinne (ed.) Marine Ecology 5(4) : 1617-1709. John Wiley, New York (Cited from Eisler, 1986).

Environment Canada 1985. National Manual for the Management of PCB wastes. Appendix G: Federal and Provincial regulations relating to PCBs. Environmental Protection Service Draft Report (1986) (Cited from Environment Canada, 1987).

Environment Canada 1987. Summary of Environmental Criteria for Polychlorinated Biphenyls (PCBs). Report EPS 4/HA/1. Ottawa, Ontario, K1A 0E7.

Fairbanks, B. C, and G. A. O'Connor. 1984. Effect of sewage sludge on the adsorption of polychlorinated biphenyls by three New Mexico soils. J. Environ. Qual. 13: 297-300.

Fairbanks, B. C, G. A. O'Connor, and S. E. Smith. 1987. Mineralisation and volatilisation of polychlorinated biphenyls in sludge-amended soils. J. Environ. Qual. 16: 18-25.

Fishbein, L. 1974. Toxicity of chlorinated biphenyls. Ann. Rev. Pharmacol. 14: 139-156.

Fisher, N. S., and C. F. Wurster. 1973. Individual and combined effects of temperature and polychlorinated biphenyls on the growth of three species of phytoplankton. Environ. Pollut. 5: 205-212.

Fisher, N. S., E. J. Carpenter, C. C. Remsen, and C. F. Wurster. 1974. Effects of PCB on inter-specific competition in natural and gnotobiotic phytoplankton communities in continuous and batch cultures. Microbial Ecol. 1: 39-50.

Frank, R., R. L. Thomas, H. E. Braun, D. L. Gross, and T. T. Davies. 1981. Organochlorine insecticides and PCB in surficial sediments of Lake Michigan (1975). J. Great Lakes Res. 7: 42-50 (Cited from Moore and Ramamoorthy, 1984).

Fries, G. F. 1982. Potential polychlorinated biphenyl residue in animal products from application of contaminated sewage sludge to land. J. Environ. Qual. 11: 14-20.

Fries, G. F., and G. S. Marrow. 1981. Chlorobiphenyl movement from soil to soybean plant. J. Agric. Food Chem. 29: 757-759.

Fuhremann, T. W., and E. P. Lichtenstein. 1972. Toxicol. Appl. Pharmacol. 22: 628.

Garrett, C. L. 1983. An overview of PCBs and their current status in British Columbia. Regional Program Report: 83-16. Department of Environment, EPS, Pacific and Yukon Region.

Garrett, C. L. 1985. Chemicals in the Environment. Pacific and Yukon Region : III. Polychlorinated Biphenyls.32p.

Glooschenko, V., and W. Glooschenko. 1975. Effect of polychlorinated biphenyl compounds on growth of Great Lakes phytoplankton. Can. J. Bot. 53: 653-659.

Gooch, J. A., and M. K. Hamdy. 1983. Uptake and concentration of Aroclor 1254 in aquatic organisms. Bull. Environ. Contam. Toxicol. 31: 445-452.

Goyette, D., and J. Boyd. 1989. Distribution and environmental impact of selected benthic contaminants in Vancouver Harbour, British Columbia., 1985 to 1987. Regional Program Report: 89-02. Environmental Protection, Conservation and Protection, Environment Canada, Pacific and Yukon Region, West Vancouver, B.C.99p.

Grant, D. L. 1983. Regulation of PCBs in Canada. *In* : PCBs: F. M. D'Itri and M. A. Kamrin (eds.) Human and Environmental Hazards. Butterworth Publishers, Toronto. p.383-392 (Cited from Environment Canada, 1987)

Gruger, E. H., M. M. Wekell, P. T. Numoto, and D. R. Craddock. 1977. Induction of hepatic aryl hydrocarbon hydroxylase in salmon exposed to petroleum dissolved in seawater and to petroleum and polychlorinated biphenyls, separate and together, in food. Bull. Environ. Contam. Toxicol. 17: 512-520.

Hansen, D. J., P. R. Parrish, J. I. Lowe, A. J. Wilson, Jr., and P. D. Wilson. 1971. Chronic toxicity, uptake, and retention of Aroclor 1254 in two estuarine fishes. Bull. Environ. Contam. Toxicol. 6:113-119.

Hansen, D. J., S. C. Schimmel, and J. Forester. 1973. Aroclor 1254 in eggs of sheepshead minnows: Effect on fertilisation success and survival of embryos and fry. Proc. 27th Annu. Conf. S.E. Assoc. Game Fish. Comm. p 420-426.

Hansen, D. J., S. C. Schimmel, and J. Forester. 1974. Effects of Aroclor 1254 on laboratory-reared embryos and fry of sheepshead minnows (*Cyprinodon variegatus*). Trans. Am. Fish. Soc. 103: 582-586.

Hansen, D. J., S. C. Schimmel, and J. Forester. 1975. Effects of Aroclor 1016 on embryos, fry, juveniles, and adults of sheepshead minnows (*Cyprinodon variegatus*). Trans. Am. Fish. Soc. 104: 584-588.

Hansen, L. G. 1987. Environmental toxicology of Polychlorinated biphenyls. *In* : S. Safe (ed.) Polychlorinated Biphenyls (PCBs): Mammalian and Environmental Toxicology.

Environmental Toxin Series 1 (Edited by S. Safe and O. Hutzinger) Springer-Verlag, Berlin Heidelberg. p 15-48.

Hansen, L. G., C. S. Byerly, R. L. Metcalf, and R. F. Bevill. 1975. Effect of a polychlorinated biphenyl mixture on swine reproduction and tissue residues. Amer. J. Vet. Res. 36: 23-26.

Haque, R., and D. W. Schmedding. 1979. Studies on the adsorption of selected polychlorinated biphenyl isomers on several surfaces. J. Environ. Sci. Health. B11: 129.

Haque, R., D. W. Schmedding, and V. H. Freed. 1974. Aqueous solubility, adsorption, and vapour behaviour of polychlorinated biphenyl Aroclor 1254. Environ. Sci. Technol. 8: 139.

Harding, L. W. Jr., and J. H. Phillips Jr. 1978. Polychlorinated biphenyl (PCB) effects on marine phytoplankton photosynthesis and cell division. Marine Biol. 49: 93-101.

Hawes, M. L., J. C. Kricher, and J. C. Urey. 1976. The effects of various Aroclor fractions on the population growth of *Chlorella pyrenoidosa*. Bull. Environ. Contam. Toxicol. 15: 14-18.

Hayes, M. A. 1987. Carcinogenic and mutagenic effects of PCBs. *In* : S. Safe (ed.) Polychlorinated Biphenyls (PCBs): Mammalian and Environmental Toxicology. Springer-Verlag, Berlin Heidelberg. p 77-95.

Health and Welfare Canada. 1975. Temporary guideline established for polychlorinated biphenyls (PCBs) in fish. News release no. 1975-144, 2p. (Cited from Environment Canada, 1987).

Health and Welfare Canada. 1989. Guidelines for Canadian Drinking Water Quality. Fourth Edition. Canadian Govt. Publ. Centre, Supply and Services Canada, Ottawa, Canada K1A 0S9. 25 p.

Healy, W. B. 1968. Ingestion of soil by dairy cows. N.Z. J. Agric. Res. 11: 487-499.

Hermanutz, R., and F. Puglisi. 1976. Comparative toxicity of Aroclor® 1016 and 1242 to flagfish (*Jordanella floridae*) and fathead minnow (*Pimephales promelas*). Duluth, Minnesota (Cited from U.S. EPA, 1980).

Hiraizumi, Y., M. Takahashi, and H. Nishimura. 1979. Adsorption of polychlorinated biphenyl onto sea bed sediment, marine plankton, and other adsorbing agents. Environ. Sci. Technol. 13: 580.

Hutzinger, O, S. Safe, and V. Zitco. 1974. The Chemistry of PCBs. CRC Press, Inc., Cleveland, Ohio. ISBN 0-87819-049-X

International Joint Commission (IJC). 1977. New and revised Great Lakes water quality objectives: Volume II (Cited from Environment Canada, 1987).

International Joint Commission (IJC). 1985. Great Lakes water quality objectives, guidelines, criteria, and standards (Cited from Environment Canada, 1987).

Jan, J., and M. Tratnik. 1988. Polychlorinated biphenyls in residents around the River Krupa, Slovenia, Yugoslavia. Bull. Environ. Contam. Toxicol. 41: 809-814.

Johnson, W. W., and M. T. Finley. 1980. Handbook of acute toxicity of chemicals to fish and aquatic invertebrates. Summaries of toxicity tests conducted at Columbia National Fisheries Research Laboratory, 1965-78. U.S. Fish Wildl. Serv. Resour. Publ. 137. 98p.

Johnston, N. T., L. T. Albright, T. G. Northcote, P. C. Oloffs, and K. Tsumura. 1975. Chlorinated hydrocarbon residues in fishes from the lower Fraser River. Rep. No. 9, Westwater Research Centre, The Univ. of British. Columbia., Vancouver, BC

Jones, K. C. 1988. Determination of polychlorinated biphenyls in human foodstuffs and tissues: Suggestions for a selective congener analytical approach. Sci. Total Environ. 68: 141-159 (Cited from McFarland and Clarke, 1989).

Kannan, N., S. Tanabe, and R. Tatsukawa. 1988. Toxic potential of non-*ortho* coplanar PCBs in commercial PCB preparations: " 2,3,7,8-T4CDD toxicity equivalence factors approach". Bull. Environ. Contam. Toxicol. 41: 267-276.

Karickhoff, S. W., D. S. Brown, and T. A. Scott. 1979. Sorption of hydrophobic pollutants on natural sediments. Water Res. 13: 241.

Keil, J. E., C. D. Graber, L. E. Priester, and S. H. Sandifer. 1972. Polychlorinated biphenyls (Aroclor 1242): Effects of uptake on *E. coli* growth. 1: 175-177

Kimbrough, R. D. 1985. Laboratory and human studies on polychlorinated biphenyls (PCBs) and related compounds. Environ. Health Pers. 59: 99-106.

Kimbrough, R. D., R. A. Squire, R. E. Linder, D. J. Strandberg, R. J. Montali, and V. W. Burse. 1975. Induction of liver tumours in Sherman strain rats by polychlorinated biphenyl Aroclor 1260. J. Natl. Cancer Inst. 55: 1453-1459 (Cited from Kimbrough, 1985).

Koch, R. B., *et al*. 1972. Polychlorinated biphenyls: Effect of long-term exposure on ASTPase activity in fish, *Pimephales promelas* . Bull. Environ. Contam. Toxicol. 7: 87 (Cited from U.S. EPA, 1980).

Kuratsune, M., Y. Masuda, and J. Nagayama. 1976. Some recent findings concerning Yusho. Proceedings of the National Conference on Polychlorinated Biphenyls, Nov. 19-21, 1975, Chicago, Illinois. U.S.E.P.A Publication No. EPA-560/6-75-004, p 14-29 (Cited from Moore and Ramamoorthy, 1984).

Laake, M. 1984. *In:* M.C. Barros, H. Konemann, and R. Visser (eds) PCB Seminar Proceedings, Ministry of Housing, Physical Planning and Environment, The Hague. 410p. (Cited from Hansen, 1987).

Larsson, C. M., and J. E. Tillberg. 1975. Effects of commercial polychlorinated biphenyl mixture Aroclor®1242 on growth, viability, phosphate uptake, respiration and oxygen evolution in *Scenedesmus*. Physiol. Plant. 33: 256-260.

Lichtenstein, E. P., K. R. Schulz, T. W. Fuhremann, and T. T. Liang. 1969. Biological interaction between plasticizers and insecticides. J. Econ. Entomol. 62: 761-765.

Long, E. R., and L. G. Morgan. 1990. The potential for biological effects of the sediment-sorbed contaminants tested in the National Status and Trends Program. National Oceanic and Atmospheric Admin. U.S.D.C., Seattle, Washington. NOAA Tech. Memo. NOS OMA 52.

Lowe, J. I., P. R. Parrish, J. M. Patrick Jr., and J. Forester. 1972. Effects of the polychlorinated biphenyl Aroclor 1254 on the american oyster *Crassostrea virginica*. Marine Biology 17: 209-214.

Lupp, M., and L. S. McCarty. 1989. Development of provincial water quality guidelines for chlorinated dioxins and furans. A report by MacLaren Plansearch Inc. for Ontario Ministry of Environment.

Lynch, T. R., and H. E. Johnson. 1982. Availability of a hexachlorobiphenyl isomer to benthic amphipods from experimentally contaminated natural sediments. *In* : J. G.

Pearson, R. B. Foster, and W. E. Bishop (eds.) Aquatic Toxicity and Hazard Assessment: Fifth Conf. ASTM STP 766. Amer. Soc. Test. Mater. p.273-287.

MacKay, D. 1982. Correlation of bio-concentration factors. Environ. Sci. Technol. 16: 274-278.

Maki, A. W., and H. E. Johnson. 1975. Effects of PCB (Aroclor 1254) and p,p'DDT on production and survival of *Daphnia magna* Strauss. Bull. Environ. Contam. Toxicol. 13: 412-416.

Maroni, N., A. Columbi, S. Cantoni, E. Ferioli, and V. Foa. 1981. Occupational exposure to polychlorinated biphenyls in electrical workers. I. Environmental and blood polychlorinated biphenyls concentrations. Br. J. Ind. Med. 38: 49-54.

Mauck, W. L., P. M. Mehrle, and F. L. Mayer. 1978. Effects of the polychlorinated biphenyl Aroclor 1254 on growth, survival, and bone development in brook trout (*S. fontinalis*). J. Fish. Res. Board Can. 35: 1084-1088.

Mayer, F. L., P. M. Mehrle, and H. O. Sanders. 1977. Residue dynamics and biological effects of polychlorinated biphenyls in aquatic organisms. Arch. Environ. Contam. Toxicol. 5: 501-511.

Mayer, K. S., F. L. Mayer, and A. Witt, Jr. 1985. Waste transformer oil and PCB toxicity to rainbow trout. Trans. Amer. Fish. Soc. 114: 869-886.

Maxim, L. D., and L. Harrington. 1984. A review of the Food and Drug Administration risk analysis for polychlorinated biphenyls in fish. Regul. Toxicol. Pharm.4: 192-219.

McDermott, D. J., D. R. Young, and T. C. Heesen. 1975. Polychlorinated biphenyls in marine organisms off southern California. Southern California Coastal Water Research Project TM 223 (Cited from Garrett, 1983).

McDonald, C. J., and R. E. Tourangeau. 1986. PCBs: Question and answer guide concerning polychlorinated biphenyls. Commercial Chemical Branch, EPS, Environment Canada, Ottawa, Ontario.

McFarland, V. A., and J. U. Clarke. 1989. Environmental occurrences, abundance, and potential toxicity of polychlorinated biphenyl congeners: Considerations for a congener-specific analysis. Environmental Health Perspectives 81: 225-239.

McKinney, J. D. 1976. Toxicology of selected symmetrical hexachlorobiphenyl isomers: correlating biological effects with chemical structure. Proceedings of the National Conference on Polychlorinated Biphenyl, Nov. 19-21, 1975, Chicago, Illinois. U.S.E.P.A Publication No. EPA-560/6-75-004, p 73-76 (Cited from Moore and Ramamoorthy, 1984).

McLeese, D. W., C. D. Metcalfe, and D. S. Pezzack. 1980. Uptake of PCBs from sediment by *Nereis virens* and *Crangon septemspinosa* . Arch. Environ. Contam. Toxicol. 9: 507-518.

Melancon, M. J., and J. J. Lech. 1983. Dose-effect relationship for induction of hepatic mono-oxygenase activity in rainbow trout and carp by Aroclor 1254. Aquat. Toxicol. 4: 51-61 (Cited from Ankley et al., 1986).

Melancon, M. J., C. R. Elcombe, M. J. Vodicnik, and J. J. Lech. 1981. Induction of cytochromes P-450 and mixed-function oxidase activity by polychlorinated biphenyls and ß-naphthoflavone in carp (*Cyprinus carpio*). Comp. Biochem. Physiol. 69C: 219-226 (Cited from Ankley et al., 1986).

Ministry of Housing, Planning and the Environment (MHPE). 1983. The Netherlands. Implementation of the Soil Clean-up (Interim) Act. 12p. (Cited from Environment Canada, 1987).

Moore, D. R. J., and S. L. Walker 1991. Water quality guidelines for polychlorinated biphenyls in coastal and estuarine waters: IWD Scientific Series No. 186, Environment Canada, Ottawa, Ontario.

Moore, J. W., and S. Ramamoorthy. 1984. Organic Chemicals in Natural Waters: Applied Monitoring and Impact Assessment. Springer-Verlag New York Inc. p 168-191.

Moore, Jr., S. A., and R. C. Harriss. 1972. Effects of polychlorinated biphenyl on marine phytoplankton communities. Nature 240: 356-358.

Mosser, J. L., N. S. Fisher, T-C. Teng, and C.F. Wurster. 1972. Polychlorinated biphenyls: Toxicity to certain phytoplankters. Science 174: 191-192.

Mullin, M. D., C. M. Pochini, S. McCrindle, M. Romkes, S. H. Safe, and L. M. Safe. 1985. Environ. Sci. Technol. 18:468 (Cited from Safe et al., 1987).

Murado, M. A., M. C. Tejedor, and G. Baluja. 1976. Bull. Environ. Contam. Toxicol. 15: 768. (Cited from Hansen, 1987).

National Academy of Sciences (NAS). 1979. Polychlorinated Biphenyls. A report prepared by the Committee on the Assessment of Polychlorinated biphenyls in the Environment. 182p.

National Cancer Institute (NCI). 1977. Bioassay of Aroclor 1254 for possible carcinogenicity. CAS No. 27323-18-8. National Technical Information Service, Springfield, VA, PB-279 624/IGA, NCI-CGTR-38. DHEW/Pub. NCI, Washington, DC. NIH-78-838 (Cited from Kimbrough, 1985).

Nebeker, A. V., and F. A. Puglisi. 1974. Effect of polychlorinated biphenyls (PCBs) on survival and reproduction of *Daphnia, Gammarus*, and *Tanytarsus*. Trans. Amer. Fish. Soc. 103: 722-728.

Nebeker, A. V., F. A. Puglisi, and D. L. DeFoe. 1974. Effect of polychlorinated biphenyl compounds on survival and reproduction of the fathead minnow and flagfish. Trans. Amer. Fish. Soc. 103: 562-568.

Nestel, H., and J. Budd. 1974. Chronic oral exposure of rainbow trout (*Salmo gairdneri*) to a polychlorinated biphenyl (Aroclor 1254): Pathological effects. Can. J. Comp. Med. 39: 208-215.

Niimi, A. J., and B. G. Oliver. 1989. Assessment of relative toxicity of chlorinated dibenzo-p-dioxins, dibenzofurans, and biphenyls in Lake Ontario salmonids to mammalian systems using toxic equivalent factors (TEF). Chemosphere 18: 1413-1423.

Nijman, R, and L. G. Swain. 1990. Coquitlam-Pitt River area, Burrard Inlet water quality assessment and objectives. Technical Appendix. Ministry of Environment, Victoria, British Columbia. 328 p.

Nimmo, D. R., D. J. Hansen, J. A. Couch, N. R. Cooley, P. R. Parrish, and J. I. Lowe. 1975. Toxicity of Aroclor 1254 and its physiological activity in several estuarine organisms. Arch. Environ. Contam. Toxicol. 3: 22-39.

Nimmo, D. R., and L. H. Bahner. 1976. Metals, pesticides and PCBs: Toxicity to shrimp singly and in combination. *In*: M. Wiley (ed.) Estuarine Processes, Vol 1. Uses, Stresses, and Adaptation to the Estuary. Academic Press Inc., New York. p.523-532.

Nimmo, D. R., R. R. Blackman, A. J. Wilson, Jr., and J. Forester. 1971. Toxicity and distribution of Aroclor 1254 in the pink shrimp *Penaeus duorarum*. Marine Biology 11: 191-197.

Nutrition Canada. 1975. Food Consumption Patterns Report. A report from Nutrition Canada by the Bureau of Nutritional Sciences, Health Protection Branch, Department of National Health and Welfare Canada. 248 p.

O'Connors, Jr, H. B., C. F. Wurster, C. D. Powers, D. C. Biggs, and R. G. Rowland. 1978. Polychlorinated biphenyls may alter marine trophic pathways by reducing phytoplankton size and production. Science 201: 737-739.

Olafsson, P. G., and A. M. Bryan. 1987. Toxic congener-specific analysis of PCBs: assessment of toxicity in equivalents of TCDD. Anal. Let. 20: 1719-1727.

Olafsson, P. G., A. M. Bryan, and W. Stone. 1988. Polychlorinated biphenyls and polychlorinated dibenzofurans in the tissue of patients with Yusho or Yu-Chen: Total toxicity. Bull. Environ. Contam. Toxicol. 41: 63-70.

Oliver, B. G., and A. J. Niimi. 1983. Environ. Sci. Technol. 17: 287.

Oliver, B. G., and A. J. Niimi. 1985. Environ. Sci. Technol. 19: 842.

Oliver, B. G., and A. J. Niimi. 1988. Trophodynamic analysis of polychlorinated biphenyl congeners and other chlorinated hydrocarbons in the Lake Ontario ecosystem. Environ. Sci. Technol. 22: 388-397.

Ontario Ministry of Environment (OME). 1976. Evaluating construction activities impacting on water resources. Prepared by D. Persaud and W. D. Wilkins, Water Resources Branch, Toronto, Ontario (Cited from Chu, 1989).

Ontario Ministry of the Environment (OME). 1983. Ontario Drinking Water Objectives. (Cited from Environment Canada, 1987).

Ontario Ministry of the Environment (OME). 1984a. List of tentative standards. Guidelines and provisional guidelines for air contaminants. 6 p. (Cited from Environment Canada, 1987).

Ontario Ministry of the Environment (OME). 1984. Water management goals, objectives, policies and implementation procedures of the Ministry of the Environment (Cited from Environment Canada, 1987).

Ontario Ministry of the Environment (OME). 1985. Guide to eating Ontario sport fish (Cited from Environment Canada, 1987).

Parkinson, A., and S. Safe. 1987. Mammalian biologic and toxic effects of PCBs. *In* : S. Safe (ed.) Polychlorinated Biphenyls (PCBs): Mammalian and Environmental Toxicology. Springer-Verlag, Berlin Heidelberg. p 50-75.

Plapp, F. W., Jr. 1972. Environ. Entomol. 1: 580 (Cited from Hutzinger *et al*., 1974)

Platonow, N. S., and B. S. Reinhart. 1973. The effects of polychlorinated biphenyls (Aroclor 1254) on chicken egg production, fertility and hatchability. Can. J. Comp. Med. 37: 341-346 (Cited from Strachan, 1988).

Platonow, N. S., and L. H. Karstad. 1973. Dietary effects of polychlorinated biphenyls on mink. Can. J. Comp. Med. 37: 391-400.

Québec Ministère de l'Environement (QME). 1984. Déversements d'huile contaminée au BPC: normes à suivre pour le nettoyage et l'élimination. 1p. (Cited from Environment Canada, 1987).

Richardson, B. J., and J. S. Waid. 1979. PCBs in the Port Phillip region. The Environmental significance of polychlorinated biphenyls (PCBs). Ministry for Conservation- Victoria, Environ. Studies Prog. publ. no. 248. Victoria, Australia (Cited from Fairbanks *et al*., 1987).

Ringer, R. K. 1983. Toxicology of PCBs in mink and ferrets. *In* : F. M. D'Itri and M. A. Kamrin (eds.) PCBs: human and environmental hazards. Butterworth Publ., Woburn, MA. p.227-240 (Cited from Eisler, 1986).

Roesijadi, G. S., R. Petrocelli, J. W. Anderson, C. S. Giam, and G. E. Neff. 1976. Toxicity of polychlorinated biphenyls (Aroclor 1254) to adult, juvenile, and larval stages of the shrimp *Palaemonetes pugio* . Bull. Environ. Contam. Toxicol. 15: 297-304 (Cited from Hansen, 1987).

Rubinstein, N. I., E. Lores, N. R. Gregory. 1983. Accumulation of PCBs, mercury, and cadmium by *Nereis virens, Mercenaria mercenaria,* and *Palaemonetes pugio* from contaminated harbour sediments. Aquat. Toxicol. 3: 249-260.

Safe, S. 1987. Determination of 2,3,7,8-TCDD toxic equivalent factors (TEFs): Support for the use of the *in vitro* AHH induction assay. Chemosphere 16: 791-802.

Safe, S. 1987. Polychlorinated biphenyls (PCBs): Mammalian and Environmental Toxicology. Environmental Toxin Series 1. Springer-Verlag.

Safe, S., L. Safe, and M. Mullin. 1987. Polychlorinated biphenyls: Environmental occurrence and analysis. *In* : S. Safe (ed.) Polychlorinated Biphenyls (PCBs): Mammalian and Environmental Toxicology. Springer-Verlag. p 1-13.

Sanders, M., and B. L. Haynes. 1988. Distribution pattern and reduction of polychlorinated biphenyls (PCB) in bluefish *Pomatomus salttatrix* (Linnaeus) fillet through adipose tissue removal. Bull. Environ. Contam. Toxicol. 41: 670-677.

Sawhney, B. L. 1986. Chemistry and properties of PCBs in relation to environmental effects. J. S. Waid (ed.) PCBs and the Environment. Vol. 1, p 47-64.

Sawhney, B. L, and L. Hankin. 1984. Plant contamination by PCBs from amended soils. J. Food. Prot. 47: 232-236.

Sawyer, T, and S. Safe. 1982. PCB isomers and congeners: induction of aryl hydrocarbon hydroxylase and ethoxyresorufin o-deethylase enzyme activities in rat hepatoma cells. Toxicol. Letters. 13: 87-94.

Sawyer, T. W., and S. Safe. 1985. *In vitro* AHH induction by polychlorinated biphenyl and dibenzofuran mixtures: Additive effects. Chemosphere 14: 79-84.

Schimmel, S. C., D. J. Hansen, and J. Forester. 1974. Effects of Aroclor 1254 on laboratory-reared embryos and fry of sheepshead minnows (*Cyprinodon variegatus*). Trans. Am. Fish. Soc. 103: 582-586.

Scura, E. D., and G. H. Theilacker. 1977. Transfer of the chlorinated hydrocarbon PCB in a laboratory marine food chain. Marine Biology 40: 317-325.

SEAM. 1989. Water Quality Data Bank used by the BC Ministry of Environment.

Singleton, H. J. 1983. Trace metals and selected organic contaminants in Fraser River fish. MOE Tech. Paper No. 2. Ministry of Environment, Victoria, BC

Snarski, V. M., and F. A. Puglisi. 1976. Effects of Aroclor® 1254 on brook trout, *Salvelinus fontinalis* . Ecol. Res. Ser. EPA 600/3-76-112. U.S. EPA, Duluth, Minnesota (Cited from U.S. EPA, 1980).

Srebocan, V., J. Pompe-Gotal, V. Brmalj, and M. Plazonic. 1977. Effect of polychlorinated biphenyl Aroclor 1254 on liver gluconeogenic enzyme activities in embryonic and growing chickens. Poult. Sci. 56: 732-735 (Cited from Strachan, 1988).

Stalling, D. L., and F. L. Mayer Jr. 1972. Toxicities of PCBs to fish and environmental residues. Environ. Health Perspectives. 1: 159-164.

Stein, J. E., T. Hom, E. Casillas, A. Friedman, and U. Varanasi. 1987. Simultaneous exposure of English sole (*Parophrys vetulus*) to sediment-associated xenobiotics: Part 2- Chronic exposure to an urban estuarine sediment with added 3H-benzo(a)pyrene and 14C-polychlorinated biphenyls. Mar. Environ. Res. 22: 123-149.

Strachan, W. M. J. 1988. Polychlorinated biphenyls (PCBs) - Fate and effects in the Canadian environment. Report EPS 4/HA/2. Environment Canada, Ottawa, Ontario, K1A0H3.

Strek, H. J., J. B. Weber, P. J. Shea, E. Mrozek, Jr., and M. R. Overcash. 1981. Reduction of polychlorinated biphenyl toxicity and uptake of carbon-14 activity by plants through the use of activated carbon. J. Agric. Food. Chem. 29: 288-293.

Sullivan, J., J. Ball, E. Brick, S. Hausmann, G. Pilarski, and D. Sopcich. 1985. Report of the Technical Subcommittee on determination of dredge material suitability for in-water disposal. Wisconsin Dept. of Natural Resources Report. 44 p.(Cited from Chu, 1989).

Suzuki, M. N. A., G. Okano, and T. Takahashi. 1977. Translocation of polychloro-biphenyls in soil into plants: A study by a method of culture of soybean sprouts. Arch. Environ. Contam. Toxicol. 5: 343-352

Swain, L. G., and D. G. Walton. 1988. Report on the 1987 benthos and sediment monitoring program. Fraser River Estuary Monitoring. 66p.

Swain, L. G., and D. G. Walton. 1989. Report on the 1988 fish monitoring program. Fraser River Estuary Monitoring. 147p.

Swain, L. G., and D. G. Walton. 1990. Report on the 1989 benthos and sediment monitoring program. Fraser River Estuary Monitoring. (in progress).

Swain, L. G., and G. B. Holms. 1985. Fraser-Delta area - Fraser river sub-basin from Kanaka Creek to the mouth: Water quality assessment and objectives. Water Management Division, Ministry of Environment, Victoria, BC

Tabak, H. H., S. A. Quave, C. I. Mashni, and E. F. Barth. 1981. Biodegradability studies with organic priority pollutant compounds. Jour. Water Pollut. Contr. Fed. 53: 1503-1518 (Cited from Moore and Ramamoorthy, 1984).

Tanabe, S., N. Kannan, An. Subramanium, S. Watanabe, and R. Tatsukawa. 1987. Highly toxic coplanar PCBs: Occurrence, source, persistency and toxic implications to wildlife and humans. Environ. Pollut. 47:147-163 (Cited from McFarland and Clarke, 1989).

Tanabe, S., N. Kannan, O. Mitsuhiro, and R. Tatsukawa. 1989. Toxic threat to marine mammals: Increasing potential of non-*ortho* and mono-*ortho* coplanar PCBs from land to ocean. Chemosphere 18: 485-490.

Tatem, H. E. 1986. Bioaccumulation of polychlorinated biphenyls and metal from contaminated sediment by freshwater prawns, *Macrobrachium rosenbergii* and clams, *Corbicula fluminea* . Arch. Environ. Contam. Toxicol. 15: 171-183.

Trépanier, J.-P. 1984. Biphenyles polychlorés: informations générales et situation au Québec. Ministére de l'environment du Québec. 192 p. (Cited from Environment Canada, 1987).

Tulp, M. Th. M., R. Schmitz, and O. Hutzinger. 1978. The bacterial metabolism of 4,4'-dichlorobiphenyl, and its suppression by alternative carbon source. Chemosphere 1: 103-108 (Cited from Moore and Ramamoorthy, 1984).

US Environmental Protection Agency (U.S. EPA). 1973. Water Quality Criteria 1972. EPA-R3-73-033-March 1973. p. 177.

US Environmental Protection Agency (U.S. EPA). 1977. Guidelines for the pollutional classification of Great Lakes Harbour sediments. Region V, Chicago, Illinois (Cited from Che, 1989).

US Environmental Protection Agency (U.S. EPA). 1980. Ambient Water Quality Criteria for PCBs. U.S. EPA, Criteria and Standards Division, Washington, D.C. EPA-440/5-80-068. 200p.

US Environmental Protection Agency (U.S. EPA). 1981. Office of Drinking Water (Draft). Advisory Opinion for Polychlorinated Biphenyls (PCBs) (Cited from Environment Canada, 1987).

US Environmental Protection Agency (U.S. EPA). 1987. Polychlorinated biphenyls Spill Cleanup Policy; Final Rule. Federal Register. 52 (63): 10688-10710 (Cited from Environment Canada, 1987).

US Environmental Protection Agency (U.S. EPA). 1988. Drinking water criteria document for polychlorinated biphenyls (PCBs). ECAO-CIN-414. Final. April 1988 (Cited from U.S. PHS, 1989).

US Environmental Protection Agency (U.S. EPA). 1989. Briefing report to the EPA Science Advisory Board on the equilibrium partitioning approach to generating sediment quality criteria. Office of Water Regulations and Standards, Criteria and Standards, Washington, D.C.

US Food and Drug Administration (U.S. FDA). 1984. Codes of federal regulations. Title 21, Part 109.30. p.21514-21520 (Cited from Environment Canada, 1987).

US Public Health Service (U.S. PHS). 1989. Toxicological profile for selected PCBs (Aroclor-1260, -1254, -1248, -1242, -1232, -1221, and -1016). Prepared by Syracuse Research Corp. under contract No. 68-C8-0004 for Agency for Toxic Substances and Disease Registry (ATSDR), U.S. PHS, in collaboration with U.S. EPA.135p.

van den Broek, W. L. F. 1979. Seasonal levels of chlorinated hydrocarbons and heavy metals in fish and brown shrimps from the Medway estuary, Kent. Environ. Pollut. 19: 21-38 (Cited from Moore and Ramamoorthy, 1984).

Veith, G. 1976. Comparative toxicity of A-1016 and A-1242 to fathead minnow. Environ. Res. Lab. U.S. EPA, Duluth, Minnesota. (Cited from U.S. EPA, 1980).

Veith, G. D., D. L. DeFoe, and B. V. Bergstedt. 1979. Measuring and estimating the bio-concentration factor of chemicals in fish. J. Fish. Res. Board Can. 36: 1040-1048.

Washington Department of Ecology (WDE). 1988. Preamble to draft sediment quality standards. Seattle, Washington, U.S.A. (Cited from Chu, 1989).

Weber, M. D., H. D. Monteith, and D. G. M. Corneau. 1983. Assessment of heavy metals and PCB at sludge application sites. J. Water Pollut. Cont. Fed. 55: 187-.

Weber, W. J., T. C. Voice, M. Pirbazari, G. E. Hunt, and D. M. Ulanoff. 1983. Sorption of hydrophobic compounds by sediments, soils and suspended solids. II. Sorbent evaluation studies. Water Res. 17: 1443.

Weltman, R. H., and D. H. Norback. 1981. Sequential light and electron microscopic analysis of hepatocellular carcinoma induced by Aroclor 1260. The Toxicologist 1: 65 (Cited from Kimbrough, 1985).

Weseloh, D. V., S. M. Teeple, and M. Gilbertson. 1983. Double-crested cormorants of the Great Lakes: egg-laying parameters, reproductive failure, and contaminant residues in eggs, Lake Huron 1972-73. Can. J. Zool. 61: 427-436 (Cited from Eisler, 1986).

Williamson, D. A. 1983. Surface water quality management proposal. Vol. 1. Surface water quality objectives, Water Standards and Studies Report No. 83/2. Manitoba Dept. of the Environment and Workplace Safety and Health, Manitoba (Cited from Environment Canada, 1987).

White, R. J., H. T. Kim, and J. S. Kim. 1985. PCBs in striped bass collected from the Hudson River, New York, during fall, 1981. Bull. Environ. Contam. Toxicol. 34: 883-889.

Young, D. R., and T. C. Heesen. 1978. DDT, PCB, and chlorinated benzenes in the marine ecosystem off Southern California. *In* : R. L. Jolley (ed.) Water Chlorination, Environmental Impact and Health Effects. Vol. 2: p 267-290 (Cited from Moore and Ramamoorthy, 1984).

Zullei, N., and G. Benecke. 1978. Application of a new bioassay to screen the toxicity of polychlorinated biphenyls on blue-green algae. Bull. Environ. Contam. Toxicol. 20: 786-792.