1) page 485: Replace paragraph under Human Health Section for Methylmercury with the following:
EPA is recommending that the Programs and Regions use 0.1 µg/kg/day as an interim RfD for methylmercury until the Agency has had an opportunity to review the work of the National Academy of Science (NAS). NAS is performing an independent assessment of the Agency's reference dose (RfD) for methylmercury (EPA 1999).
[U.S. EPA. 1999. Memo: Transmittal of Interim Agency Guidance on the Use of Methylmercury Reference Dose in Making Risk Management Decisions. From: Peter D. Robertson Acting Deputy Administrator, To: Assistant Administrators, General Counsel, Inspector General, Chief Financial Officer, Associate Administrators, Regional Administrators and Staff Office Directors (April 19, 1999)].

2) pages 7, 23, 35, 45, 61: Add to Human Health: Oral slope factor: 2.0 per mg/kg/d based on environmental mixtures of PCBs in aquatic organisms (EPA 1996)
[U.S. EPA. 1996. *Cancer Dose-Response Assessment for Application to Environmental Mixtures.* EPA/600/P-96/001F. Washington, DC].

3) The table below provides the latest World Health Organization (WHO) toxic equivalent factors (TEFs) for dioxins, furans, and coplanar PCBs. They are more recent than those cited in this document.

Congener	**Toxic Equivalent Factor (TEF)**
2,3,7,8-TCDD	1
1,2,3,7,8-PeCDD	1
1,2,3,4,7,8-HxCDD	0.1
1,2,3,6,7,8-HxCDD	0.1
1,2,3,7,8,9-HxCDD	0.1
1,2,3,4,6,7,8,-HpCDD	0.01
OCDD	0.0001
2,3,7,8-TCDF	0.1
1,2,3,7,8-PeCDF	0.05
2,3,4,7,8-PeCDF	0.5
1,2,3,4,7,8-HxCDF	0.1
1,2,3,6,7,8-HxCDF	0.1
1,2,3,7,8,9-HxCDF	0.1
2,3,4,6,7,8-HxCDF	0.1
1,2,3,4,6,7,8-HpCDF	0.01
1,2,3,4,7,8,9-HpCDF	0.01
OCDF	0.0001
3,4,4′,5-TCB(81)	0.0001
3,3′,4,4′-TCB(77)	0.0001
3,3′,4,4′,5-PeCB(126)	0.1
3,3′,4,4′,5,5′-HxCB(169)	0.01
2,3,3′,4,4′-PeCB(105)	0.0001
2,3,4,4′,5-PeCB(114)	0.0005
2,3′,4,4′,5-PeCB(118)	0.0001
2′,3,4,4′,5-PeCB(123)	0.0001
2,3,3′,4,4′,5-HxCB(156)	0.0005
2,3,3′,4,4′,5-HxCB(157)	0.0005
2,3′,4,4′,5,5′-HxCB(167)	0.00001
2,3,3′,4,4′,5,5′-HpCB(189)	0.0001

Van den Berg, et. al. 1998. Toxic Equivalency Factors (TEFs) for PCBs, PCDDs, PCDFs for Humans and Wildlife. *Environ. Health Perspect.* 106(12):775-792.

APPENDIX

Chemical-Specific Summary Tables

CONTENTS

Chemical Category: POLYNUCLEAR AROMATIC HYDROCARBON (low molecular weight)

Chemical Name (Common Synonyms): ACENAPHTHENE **CASRN:** 83-32-9

Chemical Characteristics

Solubility in Water: Insoluble [1]

Half-Life: No data [1,2]

Log K_{ow}: 3.92 [3]

Log K_{oc}: 3.85 L/kg organic carbon

Human Health

Oral RfD: 6×10^{-2} mg/kg/day [4]

Confidence: Low uncertainty factor = 3000

Critical Effect: Hepatotoxicity

Oral Slope Factor: No data [4]

Carcinogenic Classification: –

Wildlife

Partitioning Factors: Partitioning factors for acenaphthene in wildlife were not found in the literature.

Food Chain Multipliers: Food chain multipliers for acenaphthene in wildlife were not found in the literature.

Aquatic Organisms

Partitioning Factors: The water quality criterion tissue level (WQCTL) for acenaphthene, which is calculated by multiplying the water quality chronic value (710 µg/L) by the BCF (389.05), is 276,222 µg/kg [5].

Food Chain Multipliers: Food chain multipliers for acenaphthene in aquatic organisms were not found in the literature.

Toxicity/Bioaccumulation Assessment Profile

Most polynuclear aromatic hydrocarbons (PAHs) occur in sediment as complex mixtures. The toxicities of individual PAHs are additive and increase with increasing K_{ow}, whereas the bioavailabilities of PAHs decrease as a function of their K_{ow}s. The 10-day LC50s for *Eohaustorius estuarius* and *Leptocheirus plumulosus* in water were 374 µg/L and 678 µg/L, respectively [6]. Both amphipod species were exposed to acenaphthene-spiked sediments with total organic carbon ranging from 0.82 percent to 4.21 percent.

The 10-day LC50s ranged from 1,630 to 4,330 µg/g for *E. estuarius* and from 7,730 µg/g to >23,500 µg/g for *L. plumulosus*.

Bioaccumulation of low-molecular-weight PAHs including acenaphthene from sediments by *Rhepoxynius abronius* (amphipod) and *Armandia brevis* (polychaete) was similar; however, a large difference in tissue concentration between these two species was measured for high-molecular-weight PAHs [12]. Meador et al. [12] concluded that the low-molecular-weight PAHs were available to both species from interstitial water, while sediment ingestion was a much more important uptake route for the high-molecular-weight PAHs. The authors also indicated that bioavailability of the high-molecular weight-PAHs to amphipods was significantly reduced due to their partitioning to dissolved organic carbon.

Summary of Biological Effects Tissue Concentrations for Acenaphthene

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment µmol/g	Water µmol/L	Tissue (Sample Type) µmol/g	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
Invertebrates									
Nereis succinea, Polychaete worm	0.00003 0.001 0.0004 BDL BDL		BDL[4] BDL BDL 0.025 BDL					[7]	F
Corbicula fluminea, Asiatic clam		<0.003 <0.003	<0.0005 <0.0007					[8]	F
Mytilus edulis, Blue mussel						-0.35		[9]	F
Crassostrea virginica, Eastern oyster						-0.03		[9]	F
Macoma balthica, Baltic macoma	0.00003 0.001 0.0004		BDL BDL BDL					[7]	F
Mercenaria mercenaria, Northern quahog						-0.44 -0.09		[9]	F
Mya arenaria, Softshell						0.09		[9]	F

Summary of Biological Effects Tissue Concentrations for Acenaphthene

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment µmol/g	Water µmol/L	Tissue (Sample Type) µmol/g	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
Decapoda	0.034 0.041 0.675		0.001 0.017 0.027					[10]	F
Homarus americanus, American lobster						-0.89		[9]	F
Fishes									
Fundulus spp., Killifish						-0.33		[9]	F
Poecilia reticulata, Guppy		0.14-0.15	0.047 0.027 0.047 0.051					[11]	F
Lepomis sp., Sunfish	0.034 0.041 0.675		0.058 0.038 0.092					[10]	F
Tautogolabrus adspersus, Tautog						-1.22		[9]	F

[1] Concentration units based on wet weight unless otherwise noted.
[2] BCF = bioconcentration factor, BAF = bioaccumulation factor, BSAF = biota-sediment accumulation factor.
[3] L = laboratory study, spiked sediment, single chemical; F = field study, multiple chemical exposure; other unusual study conditions or observations noted.
[4] BDL = below detection limit.

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Chemical Category: POLYCHLORINATED BIPHENYLS

Chemical Name (Common Synonyms): Aroclor 1016 **CASRN:** 1336-36-3

Chemical Characteristics

Solubility in Water: 225-250 µg/L at 25°C [1] **Half-Life:** No data [2,3]

Log K_{ow}: 5.6 [4] **Log K_{oc}:** No data [4]

Human Health

Oral RfD: 7 x 10^{-5} mg/kg-day [5] **Confidence:** Medium [5]

Critical Effect: PCBs have been shown to cause reproductive failure, birth defects, lesions, tumors, liver disorders, and death among sensitive species. Their toxicity is further enhanced by their ability to bioaccumulate and to biomagnify within the food chain due to extremely high lipophilicity [2].

Oral Slope Factor: No data [5] **Carcinogenic Classification:** Unknown [5]

Wildlife

Partitioning Factors: No partitioning factors for Aroclor 1016 were identified for wildlife.

Food Chain Multipliers: For PCBs as a class the most toxic congeners have been shown to be selectively accumulated from organisms at one trophic level to the next [6]. At least three studies have concluded that PCBs have the potential to biomagnify in food webs based on aquatic organisms and predators that feed primarily on aquatic organisms [7,8,9]. The results from Biddinger and Gloss [7] and USACE [9] generally agreed that highly water-insoluble compounds (including PCBs) have the potential to biomagnify in these types of food webs. Thomann's [10] model also indicated that highly water-insoluble compounds (log K_{ow} values 5 to 7) showed the greatest potential to biomagnify. A biomagnification factor of 32 was determined for total PCBs from alewife to herring gull eggs in Lake Ontario [11]. No specific food chain multipliers were identified for Aroclor 1016.

Aquatic Organisms

Partitioning Factors: No partitioning factors for Aroclor 1016 were identified for aquatic organisms.

Food Chain Multipliers: Polychlorinated biphenyls as a class have been demonstrated to biomagnify through the food web. Oliver and Niimi [12], studying accumulation of PCBs in various organisms in the Lake Ontario food web, reported concentrations of total PCBs in phytoplankton, zooplankton, and several species of fish. Their data indicated a progressive increase in tissue PCB concentrations moving

from organisms lower in the food web to top aquatic predators. In a study of PCB accumulation in lake trout (*Salvelinus namaycush*) of Lake Ontario, Rasmussen et al. [13] reported that each trophic level contributed about a 3.5-fold biomagnification factor to the PCB concentrations in the trout. No specific food chain multipliers were identified for Aroclor 1016.

Toxicity/Bioaccumulation Assessment Profile

PCBs are a group (209 congeners/isomers) of organic chemicals, based on various substitutions of chlorine atoms on a basic biphenyl molecule. These manufactured chemicals have been widely used in various processes and products because of the extreme stability of many isomers, particularly those with five or more chlorines [14]. A common use of PCBs was as dielectric fluids in capacitors and transformers. In the United States, Aroclor is the most familiar registered trademark of commercial PCB formulations. Generally, the first two digits in the Aroclor designation indicate that the mixture contains biphenyls, and the last two digits give the weight percent of chlorine in the mixture. The exception to this code is Aroclor 1016, which contains mono- through hexachlorinated homologs with an average chlorine content of 41 percent [4].

As a result of their stability and their general hydrophobic nature, PCBs released to the environment have dispersed widely throughout the ecosystem [14]. PCBs are among the most stable organic compounds known, and chemical degradation rates in the environment are thought to be slow. As a result of their highly lipophilic nature and low water solubility, PCBs are generally found at low concentrations in water and at relatively high concentrations in sediment [15]. Individual PCB congeners have different physical and chemical properties based on the degree of chlorination and position of chlorine substitution, although differences with degree of chlorination are more significant [15]. Solubilities and octanol-water partition coefficients for PCB congeners range over several orders of magnitude [16]. Octanol-water partition coefficients, which are often used as estimators of the potential for bioconcentration, are highest for the most chlorinated PCB congeners.

Dispersion of PCBs in the aquatic environment is a function of their solubility [15] while PCB mobility within and sorption to sediment are a function of chlorine substitution pattern and degree of chlorination [17]. The concentration of PCBs in sediments is a function of the physical characteristics of the sediment, such as grain size [18,19] and total organic carbon content [18,19,20,21]. Fine sediments typically contain higher concentrations of PCBs than coarser sediments because of more surface area [15]. Mobility of PCBs in sediment is generally quite low for the higher chlorinated biphenyls [17]. Therefore, it is common for the lower chlorinated PCBs to have a greater dispersion from the original point source [15]. Limited mobility and high rates of sedimentation could prevent some PCB congeners in the sediment from reaching the overlying water via diffusion [17].

The persistence of PCBs in the environment is a result of their general resistance to degradation [16]. The rate of degradation of PCB congeners by bacteria decreases with increasing degree of chlorination [22]; other structural characteristics of the individual PCBs can affect susceptibility to microbial degradation to a lesser extent [16]. Photochemical degradation, via reductive dechlorination, is also known to occur in aquatic environments; the higher chlorinated PCBs appear to be most susceptible to this process [21].

Toxicity of PCB congeners is dependent on the degree of chlorination as well as the position of chlorine substitution. Lesser chlorinated congeners are more readily absorbed, but are metabolized more rapidly

than higher chlorinated congeners [23]. PCB congeners with no chlorine substituted in the ortho (2 and 2′) positions but with four or more chlorine atoms at the meta (3 and 3′) and para (4 and 4′) positions can assume a planar conformation that can interact with the same receptor as the highly toxic 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) [24]. Examples of these more toxic, coplanar congeners are 3,3′,4,4′-tetrachlorobiphenyl (PCB 77), 3,3′,4,4′,5-pentachlorobiphenyl (PCB 126), and 3,3′,4,4′5,5′-hexachlorobiphenyl (PCB 169). A method that has been proposed to estimate the relative toxicity of mixtures is to use toxic equivalency factors (TEFs) [25]. With this method, relative potencies for individual congeners are calculated by expressing their potency in relation to 2,3,7,8-TCDD. The following TEFs have been recommended [25,26]:

Congener Class	Recommended TEF
3,3′,4,4′,5-PentaCB	0.1
3,3′,4,4′,5,5′-HexaCB	0.05
3,3′,4,4′-TetraCB	0.01
Monoortho coplanar PCBs	0.001
Diortho coplanar PCBs	0.00002

Due to the toxicity, high K_{ow} values, and highly persistent nature of many PCBs, they possess a high potential to bioaccumulate and exert reproductive effects in higher-trophic-level organisms. Aquatic organisms have a strong tendency to accumulate PCBs from water and food sources. The log bioconcentration factor for fish is approximately 4.70 [27]. This factor represents the ratio of concentration in tissue to the ambient water concentration. Aquatic organisms living in association with PCB-contaminated sediments generally have tissue concentrations equal to or greater than the concentration of PCB in the sediment [27]. Once taken up by an organism, PCBs partition primarily into lipid compartments [15]. Thus, differences in PCB concentration between species and between different tissues within the same species may reflect differences in lipid content [15]. PCB concentrations in polychaetes and fish have been strongly correlated to their lipid content [28]. Elimination of PCBs from organisms is related to the characteristics of the specific PCB congeners present. It has been shown that uptake and depuration rates in mussels are high for lower-chlorinated PCBs and much lower for higher-chlorinated congeners [29, 30]. In some species, tissue concentrations of PCBs in females can be reduced during gametogenesis because of PCB transfer to the more lipophilic eggs. Therefore, the transferred PCBs are eliminated from the female during spawning [31,32]. Fish and other aquatic organisms biotransform PCBs more slowly than other species, and they appear less able to metabolize, or excrete, the higher chlorinated PCB congeners [31]. Consequently, fish and other aquatic organisms may accumulate more of the higher chlorinated PCB congeners than is found in the environment [16].

The acute toxicity of PCBs appears to be relatively low, but results from chronic toxicity tests indicate that PCB toxicity is directly related to the duration of exposure [1]. Toxic responses have been noted to occur at concentrations of 0.03 and 0.014 µg/L in marine and freshwater environments, respectively [1]. The LC50 for grass shrimp exposed to PCBs in marine waters for 4 days was 6.1 to 7.8 µg/L [1]. Chronic toxicity of PCBs presents a serious environmental concern because of their resistance to degradation [33], although the acute toxicity of PCBs is relatively low compared to that of other chlorinated hydrocarbons. Sediment contaminated with PCBs has been shown to elicit toxic responses at relatively low

concentrations. Sediment bioassays and benthic community studies suggest that chronic effects generally occur in sediment at total PCB concentrations exceeding 370 µg/kg [34].

A number of field and laboratory studies provide evidence of chronic sublethal effects on aquatic organisms at low tissue concentrations [16]. Field and Dexter [16] suggest that a number of marine and freshwater fish species have experienced chronic toxicity at PCB tissue concentrations of less than 1.0 mg/kg and as low as 0.1 mg/kg. Spies et al. [35] reported an inverse relationship between PCB concentrations in starry flounder eggs in San Francisco Bay and reproductive success, with an effective PCB concentration in the ovaries of less than 0.2 mg/kg. Monod [36] also reported a significant correlation between PCB concentrations in eggs and total egg mortality in Lake Geneva char. PCBs have also been shown to cause induction of the mixed function oxidase (MFO) system in aquatic animals, with MFO induction by PCBs at tissue concentrations within the range of environmental exposures [16].

Summary of Biological Effects Tissue Concentrations for Aroclor 1016

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
Invertebrates									
Crassostrea virginica, Oyster			4 mg/kg (whole body)[4]	Growth, ED10				[38]	L; reduction in shell growth
			32 mg/kg (whole body)[4]	Growth, NA				[38]	L; reduction in shell growth
			95 mg/kg (whole body)[4]	Growth, NA					L; reduction in shell growth
Limulus polyphemus, Horseshoe Crab			11.2 mg/kg (whole body)[4]	Growth, NA				[37]	L; delayed molting; less than 50% molted after 96 days starting with T2-stage crabs
			31.9 mg/kg (whole body)[4]	Growth, NA				[37]	L; delayed molting; less than 50% molted after 96 days starting with T1-stage crabs
			11.2 mg/kg (whole body)[4]	Mortality, NA				[37]	L; less than 50% mortality starting with T2-stage crabs
Fishes									
Lagodon rhomboides, Pinfish			38 mg/kg (muscle)[4]	Mortality, ED50				[38]	L; 50% mortality
			30 mg/kg (muscle)[4]	Mortality, ED50				[38]	L; 50% mortality
			72 mg/kg (muscle and skin)[4]	Mortality, ED50				[38]	L; 50% mortality

Summary of Biological Effects Tissue Concentrations for Aroclor 1016

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
			48 mg/kg (muscle and skin)[4]	Mortality, ED50				[38]	L; 50% mortality
			205 mg/kg (whole body)[4]	Mortality, ED50				[38]	L; 50% mortality
			106 mg/kg (whole body)[4]	Behavior, LOED				[38]	L; erratic swimming, stopped feeding, loss of equilibrium
			38 mg/kg (muscle)[4]	Behavior, LOED				[38]	L; erratic swimming, stopped feeding, loss of equilibrium
			72 mg/kg (muscle and skin)[4]	Behavior, LOED				[38]	L; erratic swimming, stopped feeding, loss of equilibrium
			205 mg/kg (whole body)[4]	Cellular, LOED				[38]	L; liver and pancreatic cell alterations
			30 mg/kg (muscle)[4]	Cellular, LOED				[38]	L; liver and pancreatic cell alterations
			48 mg/kg (muscle and skin)[4]	Cellular, LOED				[38]	L; liver and pancreatic cell alterations
			106 mg/kg (whole body)[4]	Morphology, LOED				[38]	L; darkened coloration
			38 mg/kg (muscle)[4]	Morphology, LOED				[38]	L; darkened coloration
			72 mg/kg (muscle and skin)[4]	Morphology, LOED				[38]	L; darkened coloration

Summary of Biological Effects Tissue Concentrations for Aroclor 1016

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
			205 mg/kg (whole body)[4]	Mortality, LOED				[38]	L; statistically significant increase in mortality
			140 mg/kg (muscle)[4]	Mortality, LOED				[38]	L; statistically significant increase in mortality
			30 mg/kg (muscle)[4]	Mortality, LOED				[38]	L; statistically significant increase in mortality
			180 mg/kg (muscle and skin)[4]	Mortality, LOED				[38]	L; statistically significant increase in mortality
			48 mg/kg (muscle and skin)[4]	Mortality, LOED				[38]	L; 5% mortality in 96 hours
			2.2 mg/kg (whole body)[4]	Mortality, LOED				[38]	L; statistically significant increase in mortality
			620 mg/kg (whole body)[4]	Mortality, LOED				[38]	L; statistically significant increase in mortality
			106 mg/kg (whole body)[4]	Mortality, NA				[38]	L; 18% mortality in 96 hours
			65 mg/kg (whole body)[4]	Cellular, NOED				[38]	L; no incidence of pathology (liver and pancreatic alterations)
			23 mg/kg (muscle)[4]	Cellular, NOED				[38]	L; no incidence of pathology (liver and pancreatic alterations)

Summary of Biological Effects Tissue Concentrations for Aroclor 1016

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
			49 mg/kg (muscle and skin)[4]	Cellular, NOED				[38]	L; no incidence of pathology (liver and pancreatic alterations)
			111 mg/kg (whole body)[4]	Mortality, NOED				[38]	L; no statistically significant increase in mortality
			63 mg/kg (muscle)[4]	Mortality, NOED				[38]	L; no statistically significant increase in mortality
			23 mg/kg (muscle)[4]	Mortality, NOED				[38]	L; no statistically significant increase in mortality
			76 mg/kg (muscle and skin)[4]	Mortality, NOED				[38]	L; no statistically significant increase in mortality
			49 mg/kg (muscle and skin)[4]	Mortality, NOED				[38]	L; no mortality in 96 hours
			21 mg/kg (whole body)[4]	Mortality, NOED				[38]	L; no statistically significant increase in mortality
			170 mg/kg (whole body)[4]	Mortality, NOED				[38]	L; no statistically significant increase in mortality
			111 mg/kg (whole body)[4]	Physiological, NOED				[38]	L; no reduced ability to survive osmotic stress after exposure

Summary of Biological Effects Tissue Concentrations for Aroclor 1016

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
			23 mg/kg (muscle)[4]	Physiological, NOED				[38]	L; no reduced ability to survive osmotic stress after exposure
			49 mg/kg (muscle and skin)[4]	Physiological, NOED				[38]	L; no reduced ability to survive osmotic stress after exposure
			111 mg/kg (whole body)[4]	Mortality, LOED				[38]	L; 33% mortality in 96 hours
			1.1 mg/kg (whole body)[4]	Mortality, NA				[38]	L; 38% mortality in 96 hours
			22 mg/kg (whole body)[4]	Mortality, NA				[38]	L; 93% mortality in 96 hours
			44 mg/kg (whole body)[4]	Mortality, LOED				[38]	L; 8% mortality in 96 hours
			3.8 mg/kg (whole body)[4]	Mortality, NA				[38]	L; 43% mortality in 96 hours
			42 mg/kg (whole body)[4]	Behavior, LOED				[38]	L; uncoordinated swimming, cessation of feeding
			1,100 mg/kg (whole body)[4]	Morphology, LOED				[39]	L; darkened body coloration, body lesions
			1,100 mg/kg (whole body)[4]	Mortality, LOED				[39]	L; lethal to 86% of fry in 28 days
			200 mg/kg (whole body)[4]	Mortality, LOED				[39]	L; 88% juvenile mortality in 28 days

Summary of Biological Effects Tissue Concentrations for Aroclor 1016

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
			1,100 mg/kg (whole body)[4]	Development, NOED				[39]	L; no effect on fertilization success, survival of embryos to hatching, and survival of fry two weeks after hatching
			4.2 mg/kg (whole body)[4]	Development, NOED				[39]	L; no effect on fertilization success, survival of embryos to hatching, and survival of fry two weeks after hatching
			17 mg/kg (whole body)[4]	Development, NOED				[39]	L; no effect on fertilization success, survival of embryos to hatching, and survival of fry two weeks after hatching
			66 mg/kg (whole body)[4]	Mortality, NOED				[39]	L; no effect on fry mortality in 28 days
			0.81 mg/kg (whole body)[4]	Mortality, NOED				[39]	L; no effect on fry mortality in 28 days
			4.9 mg/kg (whole body)[4]	Mortality, NOED				[39]	L; no effect on fry mortality in 28 days
			22 mg/kg (whole body)[4]	Mortality, NOED				[39]	L; no effect on fry mortality in 28 days
			38 mg/kg (whole body)[4]	Mortality, NOED				[39]	L; no effect on fry mortality in 28 days

Summary of Biological Effects Tissue Concentrations for Aroclor 1016

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
			5.9 mg/kg (whole body)[4]	Mortality, NOED				[39]	L; no effect on fry mortality in 28 days
			26 mg/kg (whole body)[4]	Mortality, NOED				[39]	L; no effect on fry mortality in 28 days
			57 mg/kg (whole body)[4]	Mortality, NOED				[39]	L; no effect on juvenile mortality in 28 days
			2.3 mg/kg (whole body)[4]	Mortality, NOED				[39]	L; no effect on juvenile mortality in 28 days
			8.9 mg/kg (whole body)[4]	Mortality, NOED				[39]	L; no effect on juvenile mortality in 28 days
			11 mg/kg (whole body)[4]	Mortality, NOED				[39]	L; no effect on juvenile mortality in 28 days
			79 mg/kg (whole body)[4]	Mortality, NOED				[39]	L; no effect on juvenile mortality in 28 days
			230 mg/kg (whole body)[4]	Mortality, NOED				[39]	L; no effect on juvenile mortality in 28 days
			10 mg/kg (whole body)[4]	Mortality, NOED				[39]	L; no effect on juvenile mortality in 28 days
			54 mg/kg (whole body)[4]	Mortality, NOED				[39]	L; no effect on juvenile mortality in 28 days
			220 mg/kg (whole body)[4]	Mortality, NOED				[39]	L; no effect on adult mortality in 28 days

Summary of Biological Effects Tissue Concentrations for Aroclor 1016

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
			0.84 mg/kg (whole body)[4]	Mortality, NOED				[39]	L; no effect on adult mortality in 28 days
			1.5 mg/kg (whole body)[4]	Mortality, NOED				[39]	L; no effect on adult mortality in 28 days
			12 mg/kg (whole body)[4]	Mortality, NOED				[39]	L; no effect on adult mortality in 28 days
			46 mg/kg (whole body)[4]	Mortality, NOED				[39]	L; no effect on adult mortality in 28 days
			100 mg/kg (whole body)[4]	Mortality, NOED				[39]	L; no effect on adult mortality in 28 days
			5.4 mg/kg (whole body)[4]	Mortality, NOED				[39]	L; no effect on adult mortality in 28 days
			22 mg/kg (whole body)[4]	Mortality, NOED				[39]	L; no effect on adult mortality in 28 days
			110 mg/kg (whole body)[4]					[39]	

[1] Concentration units based on wet weight unless otherwise noted.

[2] BCF = bioconcentration factor, BAF = bioaccumulation factor, BSAF = biota-sediment accumulation factor.

[3] L = laboratory study, spiked sediment, single chemical; F = field study, multiple chemical exposure; other unusual study conditions or observations noted.

[4] This entry was excerpted directly from the Environmental Residue-Effects Database (ERED, www.wes.army.mil/el/ered, U.S. Army Corps of Engineers and U.S. Environmental Protection Agency). The original publication was not reviewed, and the reader is strongly urged to consult the publication to confirm the information presented here.

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Chemical Category: POLYCHLORINATED BIPHENYLS

Chemical Name (Common Synonyms): Aroclor 1242 **CASRN:** 53469-21-9

Chemical Characteristics

Solubility in Water: 240 µg/L at 25°C [1] **Half-Life:** No data [2,3]

Log K_{ow}: 5.6 [4] **Log K_{oc}:** No data [4]

Human Health

Oral RfD: No data [5] **Confidence:** —

Critical Effect: PCBs have been shown to cause reproductive failure, birth defects, lesions, tumors, liver disorders, and death among sensitive species. Their toxicity is further enhanced by their ability to bioaccumulate and to biomagnify within the food chain due to extremely high lipophilicity [2].

Oral Slope Factor: No data [5] **Carcinogenic Classification:** A2 [5]

Wildlife

Partitioning Factors: No partitioning factors for Aroclor 1242 were identified for wildlife.

Food Chain Multipliers: For PCBs as a class the most toxic congeners have been shown to be selectively accumulated from organisms at one trophic level to the next [6]. At least three studies have concluded that PCBs have the potential to biomagnify in food webs based on aquatic organisms and predators that feed primarily on aquatic organisms [7,8,9]. The results from Biddinger and Gloss [7] and USACE [9] generally agreed that highly water-insoluble compounds (including PCBs) have the potential to biomagnify in these types of food webs. Thomann's [10] model also indicated that highly water-insoluble compounds (log K_{ow} values 5 to 7) showed the greatest potential to biomagnify. A biomagnification factor of 32 was determined for total PCBs from alewife to herring gull eggs in Lake Ontario [11]. No specific food chain multipliers were identified for Aroclor 1242.

Aquatic Organisms

Partitioning Factors: No partitioning factors for Aroclor 1242 were identified for aquatic organisms.

Food Chain Multipliers: Polychlorinated biphenyls as a class have been demonstrated to biomagnify through the food web. Oliver and Niimi [12], studying accumulation of PCBs in various organisms in the Lake Ontario food web, reported concentrations of total PCBs in phytoplankton, zooplankton, and several species of fish. Their data indicated a progressive increase in tissue PCB concentrations moving

from organisms lower in the food web to top aquatic predators. In a study of PCB accumulation in lake trout (*Salvelinus namaycush*) of Lake Ontario, Rasmussen et al. [13] reported that each trophic level contributed about a 3.5-fold biomagnification factor to the PCB concentrations in the trout. No specific food chain multipliers were identified for Aroclor 1242.

Toxicity/Bioaccumulation Assessment Profile

PCBs are a group (209 congeners/isomers) of organic chemicals, based on various substitutions of chlorine atoms on a basic biphenyl molecule. These manufactured chemicals have been widely used in various processes and products because of the extreme stability of many isomers, particularly those with five or more chlorines [14]. A common use of PCBs was as dielectric fluids in capacitors and transformers. In the United States, Aroclor is the most familiar registered trademark of commercial PCB formulations. Generally, the first two digits in the Aroclor designation indicate that the mixture contains biphenyls, and the last two digits give the weight percent of chlorine in the mixture (e.g., Aroclor 1242 contains biphenyls with approximately 42 percent chlorine).

As a result of their stability and their general hydrophobic nature, PCBs released to the environment have dispersed widely throughout the ecosystem [14]. PCBs are among the most stable organic compounds known, and chemical degradation rates in the environment are thought to be slow. As a result of their highly lipophilic nature and low water solubility, PCBs are generally found at low concentrations in water and at relatively high concentrations in sediment [15]. Individual PCB congeners have different physical and chemical properties based on the degree of chlorination and position of chlorine substitution, although differences with degree of chlorination are more significant [15]. Solubilities and octanol-water partition coefficients for PCB congeners range over several orders of magnitude [16]. Octanol-water partition coefficients, which are often used as estimators of the potential for bioconcentration, are highest for the most chlorinated PCB congeners.

Dispersion of PCBs in the aquatic environment is a function of their solubility [15] while PCB mobility within and sorption to sediment are a function of chlorine substitution pattern and degree of chlorination [17]. The concentration of PCBs in sediments is a function of the physical characteristics of the sediment, such as grain size [18,19] and total organic carbon content [18,19,20,21]. Fine sediments typically contain higher concentrations of PCBs than coarser sediments because of more surface area [15]. Mobility of PCBs in sediment is generally quite low for the higher chlorinated biphenyls [17]. Therefore, it is common for the lower chlorinated PCBs to have a greater dispersion from the original point source [15]. Limited mobility and high rates of sedimentation could prevent some PCB congeners in the sediment from reaching the overlying water via diffusion [17].

The persistence of PCBs in the environment is a result of their general resistance to degradation [16]. The rate of degradation of PCB congeners by bacteria decreases with increasing degree of chlorination [22]; other structural characteristics of the individual PCBs can affect susceptibility to microbial degradation to a lesser extent [16]. Photochemical degradation, via reductive dechlorination, is also known to occur in aquatic environments; the higher chlorinated PCBs appear to be most susceptible to this process [21].

Toxicity of PCB congeners is dependent on the degree of chlorination as well as the position of chlorine substitution. Lesser chlorinated congeners are more readily absorbed, but are metabolized more rapidly than higher chlorinated congeners [23]. PCB congeners with no chlorine substituted in the ortho (2 and

2′) positions but with four or more chlorine atoms at the meta (3 and 3′) and para (4 and 4′) positions can assume a planar conformation that can interact with the same receptor as the highly toxic 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) [24]. Examples of these more toxic, coplanar congeners are 3,3′,4,4′-tetrachlorobiphenyl (PCB 77), 3,3′,4,4′,5-pentachlorobiphenyl (PCB 126), and 3,3′,4,4′,5,5′-hexachlorobiphenyl (PCB 169). A method that has been proposed to estimate the relative toxicity of mixtures is to use toxic equivalency factors (TEFs) [25]. With this method, relative potencies for individual congeners are calculated by expressing their potency in relation to 2,3,7,8-TCDD. The following TEFs have been recommended [25,26]:

Congener Class	Recommended TEF
3,3′,4,4′,5-PentaCB	0.1
3,3′,4,4′,5,5′-HexaCB	0.05
3,3′4,4′-TetraCB	0.01
Monoortho coplanar PCBs	0.001
Diortho coplanar PCBs	0.00002

Due to the toxicity, high K_{ow} values, and highly persistent nature of many PCBs, they possess a high potential to bioaccumulate and exert reproductive effects in higher-trophic-level organisms. Aquatic organisms have a strong tendency to accumulate PCBs from water and food sources. The log bioconcentration factor for fish is approximately 4.70 [27]. This factor represents the ratio of concentration in tissue to the ambient water concentration. Aquatic organisms living in association with PCB-contaminated sediments generally have tissue concentrations equal to or greater than the concentration of PCB in the sediment [27]. Once taken up by an organism, PCBs partition primarily into lipid compartments [15]. Thus, differences in PCB concentration between species and between different tissues within the same species may reflect differences in lipid content [15]. PCB concentrations in polychaetes and fish have been strongly correlated to their lipid content [28]. Elimination of PCBs from organisms is related to the characteristics of the specific PCB congeners present. It has been shown that uptake and depuration rates in mussels are high for lower-chlorinated PCBs and much lower for higher-chlorinated congeners [29, 30]. In some species, tissue concentrations of PCBs in females can be reduced during gametogenesis because of PCB transfer to the more lipophilic eggs. Therefore, the transferred PCBs are eliminated from the female during spawning [31,32]. Fish and other aquatic organisms biotransform PCBs more slowly than other species, and they appear less able to metabolize, or excrete, the higher chlorinated PCB congeners [31]. Consequently, fish and other aquatic organisms may accumulate more of the higher chlorinated PCB congeners than is found in the environment [16].

The acute toxicity of PCBs appears to be relatively low, but results from chronic toxicity tests indicate that PCB toxicity is directly related to the duration of exposure [1]. Toxic responses have been noted to occur at concentrations of 0.03 and 0.014 µg/L in marine and freshwater environments, respectively [1]. The LC50 for grass shrimp exposed to PCBs in marine waters for 4 days was 6.1 to 7.8 µg/L [1]. Chronic toxicity of PCBs presents a serious environmental concern because of their resistance to degradation [33], although the acute toxicity of PCBs is relatively low compared to that of other chlorinated hydrocarbons. Sediment contaminated with PCBs has been shown to elicit toxic responses at relatively low concentrations. Sediment bioassays and benthic community studies suggest that chronic effects generally occur in sediment at total PCB concentrations exceeding 370 µg/kg [34].

A number of field and laboratory studies provide evidence of chronic sublethal effects on aquatic organisms at low tissue concentrations [16]. Field and Dexter [16] suggest that a number of marine and freshwater fish species have experienced chronic toxicity at PCB tissue concentrations of less than 1.0 mg/kg and as low as 0.1 mg/kg. Spies et al. [35] reported an inverse relationship between PCB concentrations in starry flounder eggs in San Francisco Bay and reproductive success, with an effective PCB concentration in the ovaries of less than 0.2 mg/kg. Monod [36] also reported a significant correlation between PCB concentrations in eggs and total egg mortality in Lake Geneva char. PCBs have also been shown to cause induction of the mixed function oxidase (MFO) system in aquatic animals, with MFO induction by PCBs at tissue concentrations within the range of environmental exposures [16].

Summary of Biological Effects Tissue Concentrations for Aroclor 1242

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
Invertebrates									
Hyalella azteca, Amphipod - freshwater			30 mg/kg (whole body)[4]	Mortality, NOED				[38]	L; radiolabeled compounds; Exp_conc = 3-100
Fishes									
Oncorhynchus mykiss; Rainbow trout			1.3 mg/kg (whole body)[4]	Mortality, LOED				[39]	L; 10% mortality
Salmo salar, Atlantic salmon			0.54 mg/kg (eggs)[4]	Mortality, ED75				[40]	L; estimated wet weight; eggs obtained from hatchery stock. 41 µg/g lipid
Ictalurus punctatus, Channel catfish			3.8 mg/kg (brain)[4]	Growth, LOED				[41]	L; 40% reduction in mean weight
			14.6 mg/kg (kidney)[4]	Growth, LOED				[41]	L; 40% reduction in mean weight
			11.9 mg/kg (muscle and skin)[4]	Growth, LOED				[41]	L; 40% reduction in mean weight
			14.3 mg/kg (whole body)[4]	Growth, LOED				[41]	L; 40% reduction in mean weight
			3.8 mg/kg (brain)[4]	Morphology; LOED				[41]	L; increased size of liver

Summary of Biological Effects Tissue Concentrations for Aroclor 1242

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
			14.6 mg/kg (kidney)[4]	Morphology; LOED				[41]	L; increased size of liver
			11.9 mg/kg (muscle and skin)[4]	Morphology; LOED				[41]	L; increased size of liver
			14.3 mg/kg (whole body)[4]	Morphology; LOED				[41]	L; increased size of liver
			1.16 mg/kg (blood)[4]	Cellular, NOED				[41]	L; no effect on histopathology of liver, brain, kidney
			3.8 mg/kg (brain)[4]	Cellular, NOED				[41]	L; no effect on histopathology of liver, brain, kidney
			14.6 mg/kg (kidney)[4]	Cellular, NOED				[41]	L; no effect on histopathology of liver, brain, kidney
			11.7 mg/kg (kidney)[4]	Cellular, NOED				[41]	L; no effect on histopathology of liver, brain, kidney
			11.9 mg/kg (muscle and skin)[4]	Cellular, NOED				[41]	L; no effect on histopathology of liver, brain, kidney
			11.4 mg/kg (muscle and skin)[4]	Cellular, NOED				[41]	L; no effect on histopathology of liver, brain, kidney

Summary of Biological Effects Tissue Concentrations for Aroclor 1242

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
			8.23 mg/kg (ovary)[4]	Cellular, NOED				[41]	L; no effect on histopathology of liver, brain, kidney
			5.76 mg/kg (testis)[4]	Cellular, NOED				[41]	L; no effect on histopathology of liver, brain, kidney
			14.3 mg/kg (whole body)[4]	Cellular, NOED				[41]	L; no effect on histopathology of liver, brain, kidney
			10.9 mg/kg (whole body)[4]	Cellular, NOED				[41]	L; no effect on histopathology of liver, brain, kidney
			1.16 mg/kg (blood)[4]	Mortality, NOED				[41]	L; no effect on mortality
			3.8 mg/kg (brain)[4]	Mortality, NOED				[41]	L; no effect on mortality
			14.6 mg/kg (kidney)[4]	Mortality, NOED				[41]	L; no effect on mortality
			11.7 mg/kg (kidney)[4]	Mortality, NOED				[41]	L; no effect on mortality
			11.9 mg/kg (muscle and skin)[4]	Mortality, NOED				[41]	L; no effect on mortality
			11.4 mg/kg (muscle and skin)[4]	Mortality, NOED				[41]	L; no effect on mortality

Summary of Biological Effects Tissue Concentrations for Aroclor 1242

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
			8.23 mg/kg (ovary)[4]	Mortality, NOED				[41]	L; no effect on mortality
			5.76 mg/kg (testis)[4]	Mortality, NOED				[41]	L; no effect on mortality
			14.3 mg/kg (whole body)[4]	Mortality, NOED				[41]	L; no effect on mortality
			10.9 mg/kg (whole body)[4]	Mortality, NOED				[41]	L; no effect on mortality

[1] Concentration units based on wet weight unless otherwise noted.

[2] BCF = bioconcentration factor, BAF = bioaccumulation factor, BSAF = biota-sediment accumulation factor.

[3] L = laboratory study, spiked sediment, single chemical; F = field study, multiple chemical exposure; other unusual study conditions or observations noted.

[4] This entry was excerpted directly from the Environmental Residue-Effects Database (ERED, www.wes.army.mil/el/ered, U.S. Army Corps of Engineers and U.S. Environmental Protection Agency). The original publication was not reviewed, and the reader is strongly urged to consult the publication to confirm the information presented here.

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Chemical Category: POLYCHLORINATED BIPHENYLS

Chemical Name (Common Synonyms): Aroclor 1248 **CASRN:** 12672-29-6

Chemical Characteristics

Solubility in Water: 54 µg/L at 25^0C [1] **Half-Life:** No data [2,3]

Log K_{ow}: 6.2 [4] **Log K_{oc}:** No data [4]

Human Health

Oral RfD: Inadequate data to calculate [5] **Confidence:** —

Critical Effect: PCBs have been shown to cause reproductive failure, birth defects, lesions, tumors, liver disorders, and death among sensitive species. Their toxicity is further enhanced by their ability to bioaccumulate and to biomagnify within the food chain due to extremely high lipophilicity [2]. —

Oral Slope Factor: No data [5] **Carcinogenic Classification:** A2 [5]

Wildlife

Partitioning Factors: No partitioning factors for Aroclor 1248 were identified for wildlife.

Food Chain Multipliers: For PCBs as a class the most toxic congeners have been shown to be selectively accumulated from organisms at one trophic level to the next [6]. At least three studies have concluded that PCBs have the potential to biomagnify in food webs based on aquatic organisms and predators that feed primarily on aquatic organisms [7,8,9]. The results from Biddinger and Gloss [7] and USACE [9] generally agreed that highly water-insoluble compounds (including PCBs) have the potential to biomagnify in these types of food webs. Thomann's [10] model also indicated that highly water-insoluble compounds (log K_{ow} values 5 to 7) showed the greatest potential to biomagnify. A biomagnification factor of 32 was determined for total PCBs from alewife to herring gull eggs in Lake Ontario [11]. No specific food chain multipliers were identified for Aroclor 1248.

Aquatic Organisms

Partitioning Factors: No partitioning factors for Aroclor 1248 were identified for aquatic organisms.

Food Chain Multipliers: Polychlorinated biphenyls as a class have been demonstrated to biomagnify through the food web. Oliver and Niimi [12], studying accumulation of PCBs in various organisms in the Lake Ontario food web, reported concentrations of total PCBs in phytoplankton, zooplankton, and several species of fish. Their data indicated a progressive increase in tissue PCB concentrations moving from organisms lower in the food web to top aquatic predators. In a study of PCB accumulation in lake

trout (*Salvelinus namaycush*) of Lake Ontario, Rasmussen et al. [13] reported that each trophic level contributed about a 3.5-fold biomagnification factor to the PCB concentrations in the trout. No specific food chain multipliers were identified for Aroclor 1248.

Toxicity/Bioaccumulation Assessment Profile

PCBs are a group (209 congeners/isomers) of organic chemicals, based on various substitutions of chlorine atoms on a basic biphenyl molecule. These manufactured chemicals have been widely used in various processes and products because of the extreme stability of many isomers, particularly those with five or more chlorines [14]. A common use of PCBs was as dielectric fluids in capacitors and transformers. In the United States, Aroclor is the most familiar registered trademark of commercial PCB formulations. Generally, the first two digits in the Aroclor designation indicate that the mixture contains biphenyls, and the last two digits give the weight percent of chlorine in the mixture (e.g., Aroclor 1260 contains biphenyls with approximately 60 percent chlorine).

As a result of their stability and their general hydrophobic nature, PCBs released to the environment have dispersed widely throughout the ecosystem [14]. PCBs are among the most stable organic compounds known, and chemical degradation rates in the environment are thought to be slow. As a result of their highly lipophilic nature and low water solubility, PCBs are generally found at low concentrations in water and at relatively high concentrations in sediment [15]. Individual PCB congeners have different physical and chemical properties based on the degree of chlorination and position of chlorine substitution, although differences with degree of chlorination are more significant [15]. Solubilities and octanol-water partition coefficients for PCB congeners range over several orders of magnitude [16]. Octanol-water partition coefficients, which are often used as estimators of the potential for bioconcentration, are highest for the most chlorinated PCB congeners.

Dispersion of PCBs in the aquatic environment is a function of their solubility [15] while PCB mobility within and sorption to sediment are a function of chlorine substitution pattern and degree of chlorination [17]. The concentration of PCBs in sediments is a function of the physical characteristics of the sediment, such as grain size [18,19] and total organic carbon content [18,19,20,21]. Fine sediments typically contain higher concentrations of PCBs than coarser sediments because of more surface area [15]. Mobility of PCBs in sediment is generally quite low for the higher chlorinated biphenyls [17]. Therefore, it is common for the lower chlorinated PCBs to have a greater dispersion from the original point source [15]. Limited mobility and high rates of sedimentation could prevent some PCB congeners in the sediment from reaching the overlying water via diffusion [17].

The persistence of PCBs in the environment is a result of their general resistance to degradation [16]. The rate of degradation of PCB congeners by bacteria decreases with increasing degree of chlorination [22]; other structural characteristics of the individual PCBs can affect susceptibility to microbial degradation to a lesser extent [16]. Photochemical degradation, via reductive dechlorination, is also known to occur in aquatic environments; the higher chlorinated PCBs appear to be most susceptible to this process [21].

Toxicity of PCB congeners is dependent on the degree of chlorination as well as the position of chlorine substitution. Lesser chlorinated congeners are more readily absorbed, but are metabolized more rapidly than higher chlorinated congeners [23]. PCB congeners with no chlorine substituted in the ortho (2 and 2′) positions but with four or more chlorine atoms at the meta (3 and 3′) and para (4 and 4′) positions can

assume a planar conformation that can interact with the same receptor as the highly toxic 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) [24]. Examples of these more toxic, coplanar congeners are 3,3′,4,4′-tetrachlorobiphenyl (PCB 77), 3,3′,4,4′,5-pentachlorobiphenyl (PCB 126), and 3,3′,4,4′,5,5′-hexachlorobiphenyl (PCB 169). A method that has been proposed to estimate the relative toxicity of mixtures is to use toxic equivalency factors (TEFs) [25]. With this method, relative potencies for individual congeners are calculated by expressing their potency in relation to 2,3,7,8-TCDD. The following TEFs have been recommended [25,26]:

Congener Class	Recommended TEF
3,3′,4,4′,5-PentaCB	0.1
3,3′,4,4′,5,5′-HexaCB	0.05
3,3′4,4′-TetraCB	0.01
Monoortho coplanar PCBs	0.001
Diortho coplanar PCBs	0.00002

Due to the toxicity, high K_{ow} values, and highly persistent nature of many PCBs, they possess a high potential to bioaccumulate and exert reproductive effects in higher-trophic-level organisms. Aquatic organisms have a strong tendency to accumulate PCBs from water and food sources. The bioconcentration factor for fish is approximately 50,000 [27]. This factor represents the ratio of concentration in tissue to the ambient water concentration. Aquatic organisms living in association with PCB-contaminated sediments generally have tissue concentrations equal to or greater than the concentration of PCB in the sediment [27]. Once taken up by an organism, PCBs partition primarily into lipid compartments [15]. Thus, differences in PCB concentration between species and between different tissues within the same species may reflect differences in lipid content [15]. PCB concentrations in polychaetes and fish have been strongly correlated to their lipid content [28]. Elimination of PCBs from organisms is related to the characteristics of the specific PCB congeners present. It has been shown that uptake and depuration rates in mussels are high for lower-chlorinated PCBs and much lower for higher-chlorinated congeners [29, 30]. In some species, tissue concentrations of PCBs in females can be reduced during gametogenesis because of PCB transfer to the more lipophilic eggs. Therefore, the transferred PCBs are eliminated from the female during spawning [31,32]. Fish and other aquatic organisms biotransform PCBs more slowly than other species, and they appear less able to metabolize, or excrete, the higher chlorinated PCB congeners [31]. Consequently, fish and other aquatic organisms may accumulate more of the higher chlorinated PCB congeners than is found in the environment [16].

The acute toxicity of PCBs appears to be relatively low, but results from chronic toxicity tests indicate that PCB toxicity is directly related to the duration of exposure [1]. Toxic responses have been noted to occur at concentrations of 0.03 and 0.014 µg/L in marine and freshwater environments, respectively [1]. The LC50 for grass shrimp exposed to PCBs in marine waters for 4 days was 6.1 to 7.8 µg/L [1]. Chronic toxicity of PCBs presents a serious environmental concern because of their resistance to degradation [33], although the acute toxicity of PCBs is relatively low compared to that of other chlorinated hydrocarbons. Sediment contaminated with PCBs has been shown to elicit toxic responses at relatively low concentrations. Sediment bioassays and benthic community studies suggest that chronic effects generally occur in sediment at total PCB concentrations exceeding 370 µg/kg [34].

A number of field and laboratory studies provide evidence of chronic sublethal effects on aquatic organisms at low tissue concentrations [16]. Field and Dexter [16] suggest that a number of marine and freshwater fish species have experienced chronic toxicity at PCB tissue concentrations of less than 1.0 mg/kg and as low as 0.1 mg/kg. Spies et al. [35] reported an inverse relationship between PCB concentrations in starry flounder eggs in San Francisco Bay and reproductive success, with an effective PCB concentration in the ovaries of less than 0.2 mg/kg. Monod [36] also reported a significant correlation between PCB concentrations in eggs and total egg mortality in Lake Geneva char. PCBs have also been shown to cause induction of the mixed function oxidase (MFO) system in aquatic animals, with MFO induction by PCBs at tissue concentrations within the range of environmental exposures [16].

Summary of Biological Effects Tissue Concentrations for Aroclor 1248

Species:	Concentration, Units in:			Toxicity:	Ability to Accumulate:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments
Invertebrates			**[NO DATA FOUND]**						
Fishes			**[NO DATA FOUND]**						
Wildlife			**[NO DATA FOUND]**						

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Chemical Category: POLYCHLORINATED BIPHENYLS

Chemical Name (Common Synonyms): Aroclor 1254 **CASRN:** 11097-69-1

Chemical Characteristics

Solubility in Water: 12 µg/L at 25°C[1] **Half-Life:** No data [2,3]

Log K_{ow}: — **Log K_{oc}:** —

Human Health

Oral RfD: 2 x 10^{-5} mg/kg-day [4] **Confidence:** Medium; uncertainty factor = 300

Critical Effect: Ocular exudate, inflamed and prominent Meibomian glands, distorted growth of fingernails and toenails; decreased antibody (IgG and IgM) response to sheep erythrocyte

Oral Slope Factor: No data [4] **Carcinogenic Classification:** A2 [4]

Wildlife

Partitioning Factors: No partitioning factors for Aroclor 1254 were identified for wildlife.

Food Chain Multipliers: For PCBs as a class, the most toxic congeners have been shown to be selectively accumulated from organisms at one trophic level to the next [5]. At least three studies have concluded that PCBs have the potential to biomagnify in food webs based on aquatic organisms and predators that feed primarily on aquatic organisms [6,7,8]. The results from Biddinger and Gloss [6] and USACE [8] generally agreed that highly water-insoluble compounds (including PCBs) have the potential to biomagnify in these types of food webs. Thomann's [9] model also indicated that highly water-insoluble compounds (log K_{ow} values 5 to 7) showed the greatest potential to biomagnify. A biomagnification factor of 28 was calculated by [10] for transfer of total PCBs from fish to bald eagle eggs. Similarly, a biomagnification factor of 32 was determined for total PCBs from alewife to herring gull eggs in Lake Ontario [11]. No specific foot chain multipliers were identified for Aroclor 1254.

Aquatic Organisms

Partitioning Factors: BSAFs for Dover sole were approximately 0.96 for muscle and 1.14 for liver. Invertebrates collected from New Bedford, MA, and Long Island Sound, NY, had BSAFs ranging from 3.2 to 4.8. These data are presented in the attached summary table.

Food Chain Multipliers: Polychlorinated biphenyls as a class have been demonstrated to biomagnify through the food web. Oliver and Niimi [12], studying accumulation of PCBs in various organisms in the Lake Ontario food web, reported concentrations of total PCBs in phytoplankton, zooplankton, and

several species of fish. Their data indicated a progressive increase in tissue PCB concentrations moving from organisms lower in the food web to top aquatic predators. In a study of PCB accumulation in lake trout (*Salvelinus namaycush*) of Lake Ontario, Rasmussen et al. [13] reported that each trophic level contributed about a 3.5-fold biomagnification factor to the PCB concentrations in the trout. No specific biomagnification data were identified for Aroclor 1254.

Toxicity/Bioaccumulation Assessment Profile

PCBs are among the most stable organic compounds known, and rates of chemical degradation in the environment are thought to be slow. Highly lipophilic, PCBs are generally found at low concentrations in water and at relatively high concentrations in sediment [14]. PCBs are a class of 209 discrete chemical compounds called congeners, in which one to ten chlorine atoms are attached to biphenyl. PCBs were commonly produced as complex mixtures of congeners for a variety of uses, including dielectric fluids in capacitors and transformers. In the United States, Aroclor is the most familiar requested trademark of commercial PCB formulations. The first two digits in the Aroclor designation (12) indicate that the mixture contains biphenyls, and the last two digits give the weight percent of chlorine in the mixture (e.g., Aroclor 1254 contains biphenyls with approximately 54 percent chlorine).

Individual PCB congeners have different physical and chemical properties based on the degree of chlorination and position of chlorine substitution, although differences in the degree of chlorination affect partitioning more significantly, but toxicity is more dependent on position [15]. Octanol-water partition coefficients, which are often used as estimators of the potential for bioconcentration, are highest for PCB congeners with the highest degree of chlorination. Solubilities and octanol-water partition coefficients range over several orders of magnitude. Due to their higher water solubility, lower-chlorinated PCBs might show greater dispersion from a point source, whereas the higher-chlorinated compounds might remain in the sediments closer to the source [15]. The mobility of PCBs in sediment is also a function of the chlorine substitution pattern and degree of chlorination and is generally quite low, particularly for the higher-chlorinated biphenyls [16]. Therefore, high rates of sedimentation could prevent PCBs in the sediment from reaching the overlying water via diffusion [16].

PCB concentrations in sediment are affected by physical characteristics of the sediment such as grain size and total organic carbon content [17,18]. Fine sediments typically contain higher concentrations of PCBs than coarser sediments [15]. Sorption to sediments is a function of total organic carbon content [19,20].

Toxicity of PCB congeners is dependent on the degree of chlorination as well as the isomer. Lesser chlorinated congeners are more readily absorbed, but are metabolized more rapidly than higher chlorinated congeners [21]. PCB congeners with no chlorine substituted in the ortho (2 and 2′) positions but with four or more chlorine atoms at the meta (3 and 3′) and para (4 and 4′) positions can assume a planar conformation that can interact with the same receptor as the highly toxic 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) [22]. Examples of these more toxic, coplanar congeners are 3,3′,4,4′-tetrachlorobiphenyl (PCB 77), 3,3′,4,4′,5-pentachlorobiphenyl (PCB 126), and 3,3′,4,4′,5,5′-hexachlorobiphenyl (PCB 169). A method that has been proposed to estimate the relative toxicity of mixtures is to use toxic equivalency factors (TEFs) [23]. With this method, relative potencies for individual congeners are calculated by expressing their potency in relation to 2,3,7,8-TCDD. The following TEFs have been recommended [23,24]:

Congener Class	Recommended TEF
3,3′,4,4′,5-TCB	0.1
3,3′,4,4′,5,5′-HCB	0.05
3,3′,4,4′-TeCB	0.01
Monoortho coplanar PCBs	0.001
Diortho coplanar PCBs	0.00002

Due to the toxicity, high K_{ow} values, and highly persistent nature of many PCBs, they possess a high potential to bioaccumulate and exert reproductive effects in higher-trophic-level organisms. Aquatic organisms have a strong tendency to accumulate PCBs from water and food sources. The bioconcentration factor for fish is approximately 50,000 [25]. This factor represents the ratio of concentration in tissue to the ambient water concentration. PCB concentrations in tissues of aquatic organisms will generally be greater than, or equal to, sediment concentrations [26]. PCB concentrations in fish have been strongly correlated to their lipid content. Elimination of PCBs from organisms is related to the characteristics of the specific PCB congeners present. It has been shown that uptake and depuration rates in mussels are high for lower-chlorinated PCBs and much lower for higher-chlorinated congeners [27,28]. Elimination of PCBs from the body can occur during egg production and spawning in females of some species [29,30]. There is a limited capacity for fish and other aquatic organisms to biotransform or metabolize PCBs.

Sediment contaminated with PCBs has been shown to elicit toxic responses at relatively low concentrations. Sediment bioassays and benthic community studies suggest that chronic effects generally occur in sediment at total PCB concentrations exceeding 370 µg/kg [31]. The LC50 for grass shrimp exposed to PCBs in marine waters for 4 days was 6.1 to 7.8 µg/L [1]. Chronic toxicity of PCBs presents a serious environmental concern because of their resistance to degradation [32], although the acute toxicity of PCBs is relatively low compared to that of other chlorinated hydrocarbons. Toxic responses have been noted to occur at concentrations of 0.03 and 0.014 µg/L in marine and freshwater environments, respectively [1].

Summary of Biological Effects Tissue Concentrations for Aroclor 1254

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
Nephtys incisa, Polychaete worm	New Bedford: 3,070-7,180 ng/g (TOC: 4.16-4.67%)						3.22 n = 3	[38]	F; New Bedford, MA; Long Island Sound, NY
									AF = $\frac{[\text{Organism}]_{(\text{ng/g lipid}}}{[\text{Sediment}]_{(\text{ng/g organ carb}}}$
	Long Island: 40.3-48.3 ng/g (TOC: 2.39-2.62%)						4.29 n = 3		
Crassostrea virginica, Oyster			425 mg/kg (whole body)[4]	Cellular, LOED				[49]	L; atrophy of digestive diverticulata
			425 mg/kg (whole body)[4]	Growth, LOED				[49]	L; reduced growth
			101 mg/kg (whole body)[4]	Cellular, NOED				[49]	L; no effect on histopathology of digestive diverticulata
			101 mg/kg (whole body)[4]	Growth, NOED				[49]	L; no effect on growth
			425 mg/kg (whole body)[4]	Mortality, NOED				[49]	L; no effect on mortality
			101 mg/kg (whole body)[4]	Mortality, NOED				[49]	L; no effect on mortality
Crassostrea virginica, Oyster			33 mg/kg (whole body)[4]	Growth, NA				[46]	L; 41% reduction in rate of shell growth
			8.1 mg/kg (whole body)[4]	Growth, NA				[46]	L; 19% reduction in rate of shell growth

Summary of Biological Effects Tissue Concentrations for Aroclor 1254

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
			33 mg/kg (whole body)[4]	Mortality, NOED				[46]	L; no effect on survival in 96 hours
			8.1 mg/kg (whole body)[4]	Mortality, NOED				[46]	L; no effect on survival in 96 hours
Yoldia limatula, Bivalve	New Bedford: 3,070-7,180 ng/g (TOC: 4.16-4.67%)						4.07 n = 3	[38]	F; New Bedford, MA; Long Island Sound, NY
	Long Island: 40.3-48.3 ng/g (TOC: 2.39-2.62%)						4.79 n = 3		
Macoma nasuta, Clam			Concentrations at Stations:					[39]	L; standard bioassay with field collected sediments with multiple contaminants.

Summary of Biological Effects Tissue Concentrations for Aroclor 1254

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
	<20 µg/kg dw		21.4 µg/kg, (variance = 9.8, n=5)	100% survival					Tissue burdens and toxicity were determined in separate aquaria after 20 and 10 days, respectively.
	<20 µg/kg dw		35.2* µg/kg, (variance = 27.2, n=5)	100% survival					
	<20 µg/kg dw		20 µg/kg, (variance = 0, n=5)	100% survival					
	<20 µg/kg dw		27.8* µg/kg, (variance = 20.7, n=5)	100% survival					
			**statistically significant increase*						
Daphnia magna, Cladoceran			10.4 mg/kg (whole body)[4]	Mortality, NOED				[52]	L; radiolabeled compound
Gammarus pseudolimnaeus, Amphipod			7.8 mg/kg (whole body)[4]	Mortality, NOED				[52]	L; radiolabeled compound
Gammarus tigrinus, Amphipod			4.64 mg/kg (whole body)[4]	Behavior, NOED				[51]	L; radiolabeled compound
Penaeus duorarum, Pink shrimp			3.9 mg/kg (whole body)[4]	Mortality, ED100				[46]	L; 100% mortality after 48 hours
Palaemonetes kadiakensis, Grass shrimp			3.2 mg/kg (whole body)[4]	Mortality, NOED				[52]	L; radiolabeled compound

Summary of Biological Effects Tissue Concentrations for Aroclor 1254

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
Orconectes nais, Crayfish			0.04 mg/kg (whole body)[4]	Mortality, NOED				[52]	L; radiolabeled compound
			16 mg/kg (whole body)[4]	Mortality, NA				[46]	L; lethal to 18 of 25 fish in 20 days
			33 mg/kg (whole body)[4]	Behavior, NOED				[46]	L; no effect on sense of equilibrium or behavior
			1.3 mg/kg (whole body)[4]	Mortality, NOED				[46]	L; no effect on survival in 48 hours
			33 mg/kg (whole body)[4]	Mortality, NOED				[46]	L; no effect on survival in 20 days
			0.14 mg/kg (whole body)[4]	Mortality, NOED				[46]	L; no effect on survival in 48 hours
Callinectes sapidus, Blue crab			23 mg/kg (whole body)[4]	Mortality, NOED				[46]	L; no effect on survival in 20 days
Culex tarsalis, Mosquito			5.4 mg/kg (whole body)[4]	Mortality, NOED				[52]	L; radiolabeled compound
Chaoborus punctipennis, Midge			1.2 mg/kg (whole body)[4]	Mortality, NOED				[52]	L; radiolabeled compound
Corydalus cornutus, Midge			1.02 mg/kg (whole body)[4]	Mortality, NOED				[52]	L; radiolabeled compound
Pteronarcys dorsata, Giant black stonefly			1.4 mg/kg (whole body)[4]	Mortality, NOED				[52]	L; radiolabeled compound

Summary of Biological Effects Tissue Concentrations for Aroclor 1254

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
Acheta domesticus, House cricket	Soil: 1,000 ppm 2,000 ppm		148.6 ppm 143.9 ppm	Signficant mortality (LC50 = 1,200 ppm)				[37]	L; 14-d soil bioassay; despite high mortality no significant differences were seen in growth rate or food consumption between surviving crickets and control crickets.
Fishes									
Oncorhynchus kisutch, Coho salmon			0.37 mg/kg (liver)	Mortality, ED10				[48]	L; 10% mortality of smolts
			0.15 mg/kg (whole body)[4]	Development, LOED				[48]	L; reduced ability of smolts to adapt to seawater
			0.5 mg/kg (liver)	Physiological, LOED				[48]	L; delayed increase in plasma thyroxine (T4) prior to smoltification by 30 days
Oncorhynchus mykiss, Rainbow trout			0.2 mg/kg (whole body)[4]	Physiological, LOED				[50]	L; increased ethoxyresorufin o-deethylase (EROD) activity

Summary of Biological Effects Tissue Concentrations for Aroclor 1254

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
Oncorhynchus mykiss, Rainbow trout			0.2 mg/kg (whole body)[4]	Physiological, LOED				[50]	L; increased ethoxyresorufin o-deethylase (EROD) activity
Oncorhynchus mykiss, Rainbow trout			Muscle or liver = 300 µg/kg	Elevated hepatic MFO (EROD) activity after 70 days				[40]	L
Pimephales promelas, Fathead minnow	0.82 µg/g dw 14-27 µg/g dw		5.25-11.6 µg/g 13.7-47.2 µg/g	No effect Reproduction inhibited. Frequency and fecundity 5-30% of control values.				[41]	L; organism survival and weight unaffected by PCB concentration. Increased lipid concentrations were seen with increased reproductive effects. Measurement endpoints for effects not well-defined.
Pleuronectes americanus, Winter flounder			Eggs = 7.1 µg/kg	Reduced growth in length and weight				[42]	F

Summary of Biological Effects Tissue Concentrations for Aroclor 1254

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
Microstomus pacificus, Dover sole	2.3* µg/kg, dw (median TOC - 7.6%)		Muscle = 1.1* µg/kg, dw (2.36 %lipids) Liver = 12.0* µg/kg, (24.8% lipids) **median concentration*				0.96 1.4	[43]	BSAFs are lipid and TOC normalized values reported in text.
Salvelinus fontinalis, Brook trout			39 mg/kg (fillet)	Physiological, LOED				[44]	L; 7 doses over 18-day period; effect at only exposure dose; hepatic enzyme induction
Cyprinus carpio, Common carp			0.1 mg/kg (whole body)[4]	Physiological, LOED				[50]	L; increased ethoxyresorufin o-deethylase (EROD) activity
Lagodon rhomboides, Pinfish			17 mg/kg (whole body)[4]	Mortality, NOED				[46]	L; no effect on survival in 48 hours
			3.8 mg/kg (whole body)[4]	Mortality, NOED				[46]	L; no effect on survival in 48 hours
			0.98 mg/kg (whole body)[4]	Mortality, NOED				[46]	L; no effect on survival in 48 hours
Ictalurus punctatus, Channel catfish			100 mg/kg (whole body)[4]	Physiological, NOED				[47]	L; no effect on neurotransmitters

Summary of Biological Effects Tissue Concentrations for Aroclor 1254

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
Platycephalus bassensis, Sand flathead			10 mg/kg (whole body)[4]	Physiological, ED50				[45]	L; 50% increase in activity of uridine diphosphoglucuro-nosyltransferase
			100 mg/kg (whole body)[4]	Physiological, LOED				[45]	L; induction (3x) of ethoxyresorufin o-deethylase (EROD) activity
			10 mg/kg (whole body)[4]	Physiological, NOED				[45]	L; no induction of ethoxyresorufin o-deethylase (EROD) activity

[1] Concentration units based on wet weight unless otherwise noted.

[2] BCF = bioconcentration factor, BAF = bioaccumulation factor, BSAF = biota-sediment accumulation factor.

[3] L = laboratory study, spiked sediment, single chemical; F = field study, multiple chemical exposure; other unusual study conditions or observations noted.

[4] This entry was excerpted directly from the Environmental Residue-Effects Database (ERED, www.wes.army.mil/el/ered, U.S. Army Corps of Engineers and U.S. Environmental Protection Agency). The original publication was not reviewed, and the reader is strongly urged to consult the publication to confirm the information presented here.

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Chemical Category: POLYCHLORINATED BIPHENYLS

Chemical Name (Common Synonyms): Aroclor 1260 **CASRN:** 11096-82-5

Chemical Characteristics

Solubility in Water: 0.027 mg/L at 25°C [1] **Half-Life:** No data [2,3]

Log K_{ow}: 6.8 [4] **Log K_{oc}:** No data [4]

Human Health

Oral RfD: No data [5] **Confidence:** —

Critical Effect: PCBs have been shown to cause reproductive failure, birth defects, lesions, tumors, liver disorders, and death among sensitive species. Their toxicity is further enhanced by their ability to bioaccumulate and to biomagnify within the food chain due to extremely high lipophilicity [2].

Oral Slope Factor: No data [5] **Carcinogenic Classification:** A2 [5]

Wildlife

Partitioning Factors: No partitioning factors for Aroclor 1260 were identified for wildlife.

Food Chain Multipliers: For PCBs as a class the most toxic congeners have been shown to be selectively accumulated from organisms at one trophic level to the next [6]. At least three studies have concluded that PCBs have the potential to biomagnify in food webs based on aquatic organisms and predators that feed primarily on aquatic organisms [7,8,9]. The results from Biddinger and Gloss [7] and USACE [9] generally agreed that highly water-insoluble compounds (including PCBs) have the potential to biomagnify in these types of food webs. Thomann's [10] model also indicated that highly water-insoluble compounds (log K_{ow} values 5 to 7) showed the greatest potential to biomagnify. A biomagnification factor of 32 was determined for total PCBs from alewife to herring gull eggs in Lake Ontario [11] No specific food chain multipliers were identified for Aroclor 1260.

Aquatic Organisms

Partitioning Factors: No partitioning factors for Aroclor 1260 were identified for aquatic organisms.

Food Chain Multipliers: Polychlorinated biphenyls as a class have been demonstrated to biomagnify through the food web. Oliver and Niimi [12], studying accumulation of PCBs in various organisms in the Lake Ontario food web, reported concentrations of total PCBs in phytoplankton, zooplankton, and several species of fish. Their data indicated a progressive increase in tissue PCB concentrations moving

from organisms lower in the food web to top aquatic predators. In a study of PCB accumulation in lake trout (*Salvelinus namaycush*) of Lake Ontario, Rasmussen et al. [13] reported that each trophic level contributed about a 3.5-fold biomagnification factor to the PCB concentrations in the trout. No specific food chain multipliers were identified for Aroclor 1260.

Toxicity/Bioaccumulation Assessment Profile

PCBs are a group (209 congeners/isomers) of organic chemicals, based on various substitutions of chlorine atoms on a basic biphenyl molecule. These manufactured chemicals have been widely used in various processes and products because of the extreme stability of many isomers, particularly those with five or more chlorines [14]. A common use of PCBs was as dielectric fluids in capacitors and transformers. In the United States, Aroclor is the most familiar registered trademark of commercial PCB formulations. Generally, the first two digits in the Aroclor designation indicate that the mixture contains biphenyls, and the last two digits give the weight percent of chlorine in the mixture (e.g., Aroclor 1260 contains biphenyls with approximately 60 percent chlorine).

As a result of their stability and their general hydrophobic nature, PCBs released to the environment have dispersed widely throughout the ecosystem [14]. PCBs are among the most stable organic compounds known, and chemical degradation rates in the environment are thought to be slow. As a result of their highly lipophilic nature and low water solubility, PCBs are generally found at low concentrations in water and at relatively high concentrations in sediment [15]. Individual PCB congeners have different physical and chemical properties based on the degree of chlorination and position of chlorine substitution, although differences with degree of chlorination are more significant [15]. Solubilities and octanol-water partition coefficients for PCB congeners range over several orders of magnitude [16]. Octanol-water partition coefficients, which are often used as estimators of the potential for bioconcentration, are highest for the most chlorinated PCB congeners.

Dispersion of PCBs in the aquatic environment is a function of their solubility [15] while PCB mobility within and sorption to sediment are a function of chlorine substitution pattern and degree of chlorination [17]. The concentration of PCBs in sediments is a function of the physical characteristics of the sediment, such as grain size [18,19] and total organic carbon content [18,19,20,21]. Fine sediments typically contain higher concentrations of PCBs than coarser sediments because of more surface area [15]. Mobility of PCBs in sediment is generally quite low for the higher chlorinated biphenyls [17]. Therefore, it is common for the lower chlorinated PCBs to have a greater dispersion from the original point source [15]. Limited mobility and high rates of sedimentation could prevent some PCB congeners in the sediment from reaching the overlying water via diffusion [17].

The persistence of PCBs in the environment is a result of their general resistance to degradation [16]. The rate of degradation of PCB congeners by bacteria decreases with increasing degree of chlorination [22]; other structural characteristics of the individual PCBs can affect susceptibility to microbial degradation to a lesser extent [16]. Photochemical degradation, via reductive dechlorination, is also known to occur in aquatic environments; the higher chlorinated PCBs appear to be most susceptible to this process [21].

Toxicity of PCB congeners is dependent on the degree of chlorination as well as the position of chlorine substitution. Lesser chlorinated congeners are more readily absorbed, but are metabolized more rapidly

than higher chlorinated congeners [23]. PCB congeners with no chlorine substituted in the ortho (2 and 2′) positions but with four or more chlorine atoms at the meta (3 and 3′) and para (4 and 4′) positions can assume a planar conformation that can interact with the same receptor as the highly toxic 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) [24]. Examples of these more toxic, coplanar congeners are 3,3′,4,4′-tetrachlorobiphenyl (PCB 77), 3,3′,4,4′,5-pentachlorobiphenyl (PCB 126), and 3,3′,4,4′,5,5′-hexachlorobiphenyl (PCB 169). A method that has been proposed to estimate the relative toxicity of mixtures is to use toxic equivalency factors (TEFs) [25]. With this method, relative potencies for individual congeners are calculated by expressing their potency in relation to 2,3,7,8-TCDD. The following TEFs have been recommended [25,26]:

Congener Class	Recommended TEF
3,3′,4,4′,5-PentaCB	0.1
3,3′,4,4′,5,5′-HexaCB	0.05
3,3′,4,4′-TetraCB	0.01
Monoortho coplanar PCBs	0.001
Diortho coplanar PCBs	0.00002

Due to the toxicity, high K_{ow} values, and highly persistent nature of many PCBs, they possess a high potential to bioaccumulate and exert reproductive effects in higher-trophic-level organisms. Aquatic organisms have a strong tendency to accumulate PCBs from water and food sources. The bioconcentration factor for fish is approximately 50,000 [27]. This factor represents the ratio of concentration in tissue to the ambient water concentration. Aquatic organisms living in association with PCB-contaminated sediments generally have tissue concentrations equal to or greater than the concentration of PCB in the sediment [27]. Once taken up by an organism, PCBs partition primarily into lipid compartments [15]. Thus, differences in PCB concentration between species and between different tissues within the same species may reflect differences in lipid content [15]. PCB concentrations in polychaetes and fish have been strongly correlated to their lipid content [28]. Elimination of PCBs from organisms is related to the characteristics of the specific PCB congeners present. It has been shown that uptake and depuration rates in mussels are high for lower-chlorinated PCBs and much lower for higher-chlorinated congeners [29, 30]. In some species, tissue concentrations of PCBs in females can be reduced during gametogenesis because of PCB transfer to the more lipophilic eggs. Therefore, the transferred PCBs are eliminated from the female during spawning [31,32]. Fish and other aquatic organisms biotransform PCBs more slowly than other species, and they appear less able to metabolize, or excrete, the higher chlorinated PCB congeners [31]. Consequently, fish and other aquatic organisms may accumulate more of the higher chlorinated PCB congeners than is found in the environment [16].

The acute toxicity of PCBs appears to be relatively low, but results from chronic toxicity tests indicate that PCB toxicity is directly related to the duration of exposure [1]. Toxic responses have been noted to occur at concentrations of 0.03 and 0.014 µg/L in marine and freshwater environments, respectively [1]. The LC50 for grass shrimp exposed to PCBs in marine waters for 4 days was 6.1 to 7.8 µg/L [1]. Chronic toxicity of PCBs presents a serious environmental concern because of their resistance to degradation [33], although the acute toxicity of PCBs is relatively low compared to that of other chlorinated hydrocarbons. Sediment contaminated with PCBs has been shown to elicit toxic responses at relatively low

concentrations. Sediment bioassays and benthic community studies suggest that chronic effects generally occur in sediment at total PCB concentrations exceeding 370 µg/kg [34].

A number of field and laboratory studies provide evidence of chronic sublethal effects on aquatic organisms at low tissue concentrations [16]. Field and Dexter [16] suggest that a number of marine and freshwater fish species have experienced chronic toxicity at PCB tissue concentrations of less than 1.0 mg/kg and as low as 0.1 mg/kg. Spies et al. [35] reported an inverse relationship between PCB concentrations in starry flounder eggs in San Francisco Bay and reproductive success, with an effective PCB concentration in the ovaries of less than 0.2 mg/kg. Monod [36] also reported a significant correlation between PCB concentrations in eggs and total egg mortality in Lake Geneva char. PCBs have also been shown to cause induction of the mixed function oxidase (MFO) system in aquatic animals, with MFO induction by PCBs at tissue concentrations within the range of environmental exposures [16].

Summary of Biological Effects Tissue Concentrations for Aroclor 1260

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
Invertebrates									
Clam, *Macoma nasuta*				Survival (out of 20):				[37]	L; standard bioassay with field collected sediments with multiple contaminants. Tissue burdens and toxicity were determined in separate aquaria after 20 and 10 days, respectively.
	1.2 mg/kg dw (reference station)		0.976 mg/kg (variance = 4.6×10^{6}, n=5)	19.8 (variance=0.2, n=5)					
	0.9 mg/kg dw		18.600 mg/kg (variance = na; n=5)	5.2 (variance=6.2, n=5)					
	3.8 mg/kg dw		9.170 mg/kg (variance = 3.96×10^{8}, n=5)	19.8 (variance=0.2, n=5)					

[1] Concentration units based on wet weight unless otherwise noted.

[2] BCF = bioconcentration factor, BAF = bioaccumulation factor, BSAF = biota-sediment accumulation factor.

[3] L = laboratory study, spiked sediment, single chemical; F = field study, multiple chemical exposure; other unusual study conditions or observations noted.

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Chemical Category: METAL

Chemical Name (Common Synonyms): ARSENIC **CASRN:** 7440-38-2

Chemical Characteristics

Solubility in Water: Insoluble [1] **Half-Life:** Not applicable, stable [1]

Log K_{ow}: – **Log K_{oc}:** –

Human Health

Oral RfD: 3 x 10^{-4} mg/kg/day [2] **Confidence:** Medium, uncertainty factor = 3

Critical Effect: Hyperpigmentation, keratosis, and possible vascular complications

Oral Slope Factor: 1.5 x 10^{+0} per (mg/kg)/day [2] **Carcinogenic Classification:** A [2]

Wildlife

Partitioning Factors: Partitioning factors for arsenic in wildlife were not found in the literature.

Food Chain Multipliers: Food chain multipliers for arsenic in wildlife were not found in the literature.

Aquatic Organisms

Partitioning Factors: Arsenic is a metal that occurs in aquatic systems in a number of chemical forms. The most prevalent form is arsenate, followed by arsenite, which usually is present at lower concentrations. The arsenate ions can be methylated and form alkylated compounds (methylarsenic acid and dimethylarsenic acid). In any aquatic environment only a small portion of the total arsenic (approximately 0.1 percent) exists as methylated species. The arsenic methylation rate is strongly correlated with sediment organic matter content in sediments and amount of sulfate-reducing bacteria.

Food Chain Multipliers: The simplified trophic transfer experiment conducted by Lindsay and Sanders [11] effectively ended speculation of food chain transfer to the second trophic level. Arsenic is taken up by aquatic organisms primarily through dietary exposure [3]

Toxicity/Bioaccumulation Assessment Profile

Arsenic (As) is accumulated by aquatic organisms primarily through dietary exposure [3]. The most toxic form of arsenic in aquatic systems is As III, follow by As V, and the least toxic forms are organic complexes. The bioavailability of arsenic is not dependent on the concentration of acid-volatile sulfides

(AVS). Pore water concentrations of arsenic are two to three orders of magnitude higher than surface water concentrations [4], a factor that can be of considerable toxicological importance to some benthic organisms. It has been demonstrated that sediments are the major source of arsenic to the infaunal organisms and the body burden is related to the concentration of extractable (1N HCL) arsenic normalized for iron [5].

Summary of Biological Effects Tissue Concentrations for Arsenic

Species:	Concentration, Units in[1]:					Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment		Water		Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
Invertebrates, field-collected	Total µg/g	SEM µg/g	Filt µg/L	Nonfit µg/L	µg/g					[8]	F
	404	202	57	1740	34						
	102	24	54	158	15						
	68	25	72	138	13						
	46	11	29	72	27						
	11	3	23	31	3						
	4	< 0.5	3	< 22	3						
Tubificidae	9.78 µg/g				6.96 mg/g					[7]	L
	1.15 µg/g				4.98 mg/g						
	26 µg/g				7.38 mg/g						
	18 µg/g				2.35 mg/g						
	17 µg/g				5.95 mg/g						
Helisoma campanulata, Snail					4.2 mg/kg (whole body)[4]	Mortality, ED16				[16]	L; mixture of 4 arsenic compounds, estimated body burden from graph
					16 mg/kg (whole body)[4]	Mortality, NOED				[16]	L; mixture of 4 arsenic compounds, estimated body burden from graph
					5.8 mg/kg (whole body)[4]	Mortality, NOED				[16]	L; mixture of 4 arsenic compounds, estimated body burden from graph
					4 mg/kg (whole body)[4]	Mortality, NOED				[16]	L; mixture of 4 arsenic compounds, estimated body burden from graph

Summary of Biological Effects Tissue Concentrations for Arsenic

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
Stagnicola emarginata, Snail			3.6 mg/kg (whole body)[4]	Mortality, NOED				[16]	L; mixture of 4 arsenic compounds, estimated body burden from graph
			3.6 mg/kg (whole body)[4]	Mortality, NOED				[16]	L; mixture of 4 arsenic compounds, estimated body burden from graph
			3.6 mg/kg (whole body)[4]	Mortality, NOED				[16]	L; mixture of 4 arsenic compounds, estimated body burden from graph
			3.6 mg/kg (whole body)[4]	Mortality, NOED				[16]	L; mixture of 4 arsenic compounds, estimated body burden from graph
Mytilus galloprovincialis, Mussel			0.44-0.51 mg/kg				0.047	[12]	F
Ceriodaphnia dubia, Cladoceran	1,120 µg/g	1295 µg/L		70% mortality				[4]	L
	2,720 µg/g	3580 µg/L		70% mortality					
	650 µg/g	901 µg/L		20% mortality/ no reproduction					
	569 µg/g	436 µg/L		0% mortality/ no reproduction					

Summary of Biological Effects Tissue Concentrations for Arsenic

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
Daphnia magna, Cladoceran			3.8 mg/kg (whole body)[4]	Mortality, NOED				[16]	L; mixture of 4 arsenic compounds, estimated body burden from graph, tissues exposed 21 d
			9.8 mg/kg (whole body)[4]	Mortality, NOED				[16]	L; mixture of 4 arsenic compounds, estimated body burden from graph, tissues exposed 21 d
			4.4 mg/kg (whole body)[4]	Mortality, NOED				[16]	L; mixture of 4 arsenic compounds, estimated body burden from graph, tissues exposed 21 d
			4 mg/kg (whole body)[4]	Mortality, NOED				[16]	L; mixture of 4 arsenic compounds, estimated body burden from graph, tissues exposed 21 d
			87 mg/kg (whole body)[4]	Mortality, ED50				[6]	L; lethal body burden after 21 d exposure
			33 mg/kg (whole body)[4]	Reproduction, ED10				[6]	L; 10% reduction in number of offspring
Hyallela azteca, Amphipod	3580 µg/g	1420 µg/L		Growth reduction				[4]	L

Summary of Biological Effects Tissue Concentrations for Arsenic

Species:	Concentration, Units in[1]:					Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment		Water		Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
Hyallela azteca, Amphipod	Total µg/g	SEM µg/g	Filt µg/L	Nonfilt µg/L	µg/g					[8]	F
	404	202	57	1740	7						
	102	24	54	158	12						
	68	25	72	138	4						
	46	11	29	72	2						
	11	3	23	31	1						
	4	<0.5	3	<22	0.4						
Palaemonetes pugio, Grass shrimp					1.15 mg/kg (whole body)[4]	Growth, NOED				[11]	L; no effect on growth
					1.03 mg/kg (whole body)[4]	Growth, NOED				[11]	L; no effect on growth
					1.28 mg/kg (whole body)[4]	Growth, NOED				[11]	L; no effect on growth
					1.14 mg/kg (whole body)[4]	Growth, NOED				[11]	L; no effect on growth
Pteronarcys dorsata, Giant black stonefly					8.4 mg/kg (whole body)[4]	Mortality, NOED				[16]	L; mixture of 4 arsenic compounds, estimated body burden from graph
					6 mg/kg (whole body)[4]	Mortality, NOED				[16]	L; mixture of 4 arsenic compounds, estimated body burden from graph
					7 mg/kg (whole body)[4]	Mortality, NOED				[16]	L; mixture of 4 arsenic compounds, estimated body burden from graph

Summary of Biological Effects Tissue Concentrations for Arsenic

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
			8.4 mg/kg (whole body)[4]	Mortality, NOED				[16]	L; mixture of 4 arsenic compounds, estimated body burden from graph
Fishes									
Oncorhynchus mykiss, Rainbow trout		8.4 mg/L 18.1 mg/L 240 mg/L	1.8 mg/kg 3.5 mg/kg (0.18 mmol/kg)					[10]	F
Oncorhynchus mykiss, Rainbow trout			3 mg/kg (whole body)[4]	Growth, NOED				[14]	L; exposure to arsenic for 21 d did not affect growth at the longest time interval tested
			4.7 mg/kg (whole body)[4]	Mortality, LOED				[14]	L; pre-exposure to arsenic for 7 d produced significant increase in LC50 (reduced sensitivity to exposure) at shortest time interval tested
			8.6 mg/kg (whole body)[4]	Behavior, ED50				[15]	L; loss of equilibrium, mortality
			13.5 mg/kg (whole body)[4]	Behavior, ED50				[15]	L; loss of equilibrium, mortality

Summary of Biological Effects Tissue Concentrations for Arsenic

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
			8.1 mg/kg (whole body)[4]	Behavior, ED50				[15]	L; loss of equilibrium, mortality
			8.6 mg/kg (whole body)[4]	Behavior, ED50				[15]	L; loss of equilibrium, mortality
Lepisosteus osseus, Longnose gar	673 µg/g	186 µg/L	0.051 mg/kg					[9]	F
Esox lucius, Northern pike	673 µg/g	186 µg/L	0.025 mg/kg					[9]	F
Notemigonus crysoleucas, Golden shiner	673 µg/g	186 µg/L	0.167 mg/kg					[9]	F
Notropis atherinoides, Emerald shiner	673 µg/g	186 µg/L	0.036 mg/kg					[9]	F
Notropis hudsonius, Spottail shiner	673 µg/g	186 µg/L	0.03 mg/kg					[9]	F
Pimephales notatus, Bluntnose minnow	673 µg/g	186 µg/L	0.0513 mg/kg					[9]	F

Summary of Biological Effects Tissue Concentrations for Arsenic

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
Pimephales promelas, Fathead minnow	9.10 µg/g 9.78 µg/g 1.25 µg/g 26 µg/g 15 µg/g 18 µg/g 17 µg/g 17 µg/g 11 µg/g		1.39 mg/g 1.14 mg/g 1.58 mg/g 2.40 mg/g 1.76 mg/g 0.66 mg/g 2.33 mg/g 2.22 mg/g 1.82 mg/g					[7]	L
Semotilus atromaculatus, Creek chub	673 µg/g	186 µg/L	2.36 mg/kg					[9]	F
Catostomus commersoni, White sucker	673 µg/g	186 µg/L	0.132 mg/kg					[9]	F
Fundulus diaphanus, Banded killifish	673 µg/g	186 µg/L	0.101 mg/kg					[9]	F
Amblolites repestris, Rock bass	673 µg/g	186 µg/L	0.128 mg/kg					[9]	F
Lepomis gibbosus, Pumpkinseed	673 µg/g	186 µg/L	0.342 mg/kg					[9]	F

Summary of Biological Effects Tissue Concentrations for Arsenic

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
Lepomis macrochirus, Bluegill			0.52 mg/kg (whole body)[4]	Mortality, NOED				[13]	L; no effect on mortality
Micropterus salmoides Largemouth bass	673 µg/g	186 µg/L	0.083 mg/kg					[9]	F
Perca flavescens Yellow perch	673 µg/g	186 µg/L	0.077 mg/kg					[9]	F
Stizostedion vitreum vitreum, Walleye	673 µg/g	186 µg/L	0.080 mg/kg					[9]	F

[1] Concentration units based on wet weight unless otherwise noted.

[2] BCF = bioconcentration factor, BAF = bioaccumulation factor, BSAF = biota-sediment accumulation factor.

[3] L = laboratory study, spiked sediment, single chemical; F = field study, multiple chemical exposure; other unusual study conditions or observations noted.

[4] This entry was excerpted directly from the Environmental Residue-Effects Database (ERED, www.wes.army.mil/el/ered, U.S. Army Corps of Engineers and U.S. Environmental Protection Agency). The original publication was not reviewed, and the reader is strongly urged to consult the publication to confirm the information presented here.

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Chemical Category: POLYNUCLEAR AROMATIC HYDROCARBON (high molecular weight)

Chemical Name (Common Synonyms): BENZO(A)ANTHRACENE **CASRN:** 56-55-3

Chemical Characteristics

Solubility in Water: 0.014 mg/L at 25°C [1] **Half-Life:** No data [2]

Log K_{ow}: 5.70 [3] **Log K_{oc}:** 5.60 L/kg organic carbon

Human Health

Oral RfD: No data [4] **Confidence:** —

Critical Effect: —

Oral Slope Factor (Reference): No data [4] **Carcinogenic Classification:** No data [4]

Wildlife

Partitioning Factors: Partitioning factors for benzo(a)anthracene in wildlife were not found in the literature.

Food Chain Multipliers: Food chain multipliers for benzo(a)anthracene in wildlife were not found in the literature.

Aquatic Organisms

Partitioning Factors: Partitioning factors for benzo(a)anthracene in aquatic organisms were not found in the literature.

Food Chain Multipliers: Food chain multipliers (FCMs) for trophic level 3 aquatic organisms were 12.8 (all benthic food web), 1.4 (all pelagic food web), and 8.0 (benthic and pelagic food web). FCMs for trophic level 4 aquatic organisms were 20.2 (all benthic food web), 2.3 (all pelagic food web), and 10.2 (benthic and pelagic food web) [16].

Toxicity/Bioaccumulation Assessment Profile

The acute toxicity of hydrocarbons, including benzo(a)anthracene, to both fresh and salt water crustaceans is largely nonselective, i.e., it is not primarily influenced by molecular structure, but is rather controlled by organism-water partitioning which, for nonpolar organic chemicals, is in turn a reflection

of aqueous solubility. The toxic effect is believed to occur at a relatively constant concentration within the organism [5]. Toxicity of benzo(a)anthracene, as well as chrysene and pyrene, to striped bass (*Morone saxatilis*) decreased as water salinity increased [6].

Bioavailability of sediment-associated polynuclear aromatic hydrocarbons (PAHs), e.g., benzo(a)anthracene, has been observed to decline with increased contact time [7]. The majority of investigations have shown that aquatic organisms are able to release PAHs from their tissues rapidly when they were returned to a clean environment. Mussels exposed to contaminated sediment rapidly accumulated benzo(a)anthracene reaching maximum concentrations at day 20 [8]. The concentration factors for mussels exposed to 675 ng/g of benzo(a)anthracene in sediment ranged from 2,470 to 35,700 [4]. Benzo(a)anthracene was rapidly taken up by the aquatic plant, *Fontinalis antipyretica* and the uptake kinetics plateaued between 48 and 168 h of exposure [9]. Roy et al. [9] suggested that slow elimination of benzo(a)anthracene from the plant tissue may be due to low aqueous solubility. Sediment-associated benzo(a)anthracene can be accumulated from two sources: interstital water and ingested particles. The accumulation kinetics of benzo(a)anthracene suggest that uptake occurs via the sediment interstitial water and ingested material and is controlled by desorption from sediment particles and dissolved organic matter [10]. Benzo(a)anthracene after 24 h exposure was accumulated by *Daphnia pulex* mostly from the water, while lower-molecular-weight PAHs like napththalene and phenanthrene were accumulated primarily through algal food [11].

Bioaccumulation of low-molecular-weight PAHs from sediments by *Rhepoxynius abronius* (amphipod) and *Armandia brevis* (polychaete) was similar, however, a large difference in tissue concentration between these two species was measured for high-molecular-weight PAHs including benzo(a)anthracene [12]. Meador et al. [12] concluded that the low-molecular-weight PAHs were available to both species from interstitial water, while sediment ingestion was a much more important uptake route for the high-molecular-weight PAHs. The authors also indicated that bioavailability of the high-molecular-weight PAHs to amphipods was significantly reduced due to their partitioning to dissolved organic carbon.

Summary of Biological Effects Tissue Concentrations for Benzo(a)anthracene

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
Invertebrates									
Corbicula fluminea, Asian clam	59 µg/kg OC		508 µg/kg lipid				8.643	[15]	F; %lipid = 0.61; %sed OC = 1.19
	3,613 µg/kg OC		1,049 µg/kg lipid				0.290	[15]	F; %lipid = 0.61; %sed OC = 1.19
Macoma nasuta, Clam	4.13 ng/g		16.5 ng/g			-0.21		[13]	F
	6.19 ng/g		6.1 ng/g			-0.82		[13]	F
	39.9 ng/g		14 ng/g			-0.62		[13]	F
	39.5 ng/g		11 ng/g			-0.68		[13]	F
	138 ng/g		66 ng/g			-0.36		[13]	F
	146 ng/g		53 ng/g			-0.32		[13]	F
Daphnia pulex, Cladoceran		5.27 µg/L	1.6 ng/g		3.04			[11]	L
Pontoporeia hoyi, Amphipod	28 ng/g		72 ng/g					[10]	L

Summary of Biological Effects Tissue Concentrations for Benzo(a)anthracene

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
Fishes									
Leuciscus idus, Golden ide			17.5 mg/kg (whole body)	Mortality, NOED				[14]	L; no effect on survivorship in 3 days

[1] Concentration units based on wet weight unless otherwise noted.

[2] BCF = bioconcentration factor, BAF = bioaccumulation factor, BSAF = biota-sediment accumulation factor.

[3] L = laboratory study, spiked sediment, single chemical; F = field study, multiple chemical exposure; other unusual study conditions or observations noted.

[4] This entry was excerpted directly from the Environmental Residue-Effects Database (ERED, www.wes.army.mil/el/ered, U.S. Army Corps of Engineers and U.S. Environmental Protection Agency). The original publication was not reviewed, and the reader is strongly urged to consult the publication to confirm the information presented here.

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Chemical Category: POLYNUCLEAR AROMATIC HYDROCARBON (high molecular weight)

Chemical Name (Common Synonyms): BENZO(A)PYRENE **CASRN:** 50-32-8

Chemical Characteristics

Solubility in Water: 0.0038 mg/L at 25°C [1]

Half-Life: 5.7 d - 1.45 yrs based on aerobic soil die-away test data at 10-30°C [2]

Log K_{ow}: 6.11 [3]

Log K_{oc}: 6.01 L/kg organic carbon

Human Health

Oral RfD: No Data [4]

Confidence: —

Critical Effect: Forestomach cancer in mice

Oral Slope Factor: $7.3 \times 10^{+0}$ per (mg/kg)/day [4]

Carcinogenic Classification: B2 [4]

Wildlife

Partitioning Factors: Partitioning factors for benzo(a)pyrene in wildlife were not found in the literature.

Food Chain Multipliers: Food chain multipliers for benzo(a)pyrene in wildlife were not found in the literature.

Aquatic Organisms

Partitioning Factors: Partitioning factors for benzo(a)pyrene in aquatic organisms were not found in the literature.

Food Chain Multipliers: Trophic tranfer of benzo(a)pyrene metabolites has been demonstrated between polychaetes and bottom-feeding fish [5]. The diatom *Thalassiosira pseudonana* cultured in 10 µg/L of benzo(a)pyrene and subsequently fed to larvae of the hard clam *Mercenaria mercenaria* accumulated 42.2 µg/g while clams accumulated only 18.6 µg/g [6]. The rate of direct uptake by the algae was thus approximately 20 times faster than the rate of trophic transfer. Dobroski and Epifanio [6] concluded that direct uptake and trophic transfer (2 µg/g/day) are equally important in accumulation of benzo(a)pyrene. Food chain multipliers (FCMs) for trophic level 3 aquatic organisms were 18.5 (all benthic food web), 1.6 (all pelagic food web), and 11.3 (benthic and pelagic food web). FCMs for trophic level 4 aquatic organisms were 37.4 (all benthic food web), 3.1 (all pelagic food web), and 17.8 (benthic and pelagic food web) [42].

Toxicity/Bioaccumulation Assessment Profile

Bioavailability of sediment-associated polynuclear aromatic hydrocarbon (PAHs), including benzo(a)pyrene has been observed to decline with increased contact time [7]. Oikari and Kukkonene [8] established a relationship between dissolved organic matter including the percentage of hydrophobic acids and accumulation of benz(a)pyrene. They observed that the bioavailability of benzo(a)pyrene decreases in waters with dissolved organic carbon having more high-molecular-weight hydrophobic acids. The reduced bioavailability has been observed for benzo(a)pyrene accumulation from field-collected sediments compared with laboratory spiked sediments [9]. Mean accumulation of benzo(a)pyrene declined by a factor of three in *Chironomus riparius* exposed to sediment stored one week versus the sediment stored for eight weeks [10]. The concentrations of benzo(a)pyrene in whole sediment and pore water were 0.27-80.9 ng/g and 0.004-0.913 mg/mL, respectively [10].

Short-term exposures (24-h) to 1 mg/L benzo(a)pyrene averaged 8.27 nmol in fish tissue. Of this total, 67 percent was accumulated in the gallbladder or gut, indicating rapid metabolism and excretion [11]. The bioaccumulation of benzo(a)pyrene can be influenced by the lipid reserves [12]. In an experiment conducted by Clements et al. [13], chironomidae larvae rapidly accumulated benzo(a)pyrene from spiked sediment and tissue concentrations were directly proportional to sediment concentrations. However, the level of benzo(a)pyrene in bluegill that were fed contaminated chironomids was generally low, indicating either low uptake or rapid metabolism. According to McCarthy [14], accumulation of hydrophobic chemicals like benzo(a)pyrene in aqueous systems appears to depend on the amount of chemical in solution and on the amount sorbed to particles entering the food chain. Uptake and accumulation of benzo(a)pyrene was reduced by 97 percent due to sorption to organic matter [14].

Studies that report body burdens of the parent compound may, depending on the species, grossly underestimate total bioaccumulation of benzo(a)pyrene and their metabolites [15]. Kane-Driscoll and McElroy [15] concluded that the body burden of the parent compound may represent less than 10 percent of the actual total body burden of parent plus metabolites. The accumulation kinetics of benzo(a)pyrene suggest that uptake occurs largely via the sediment interstitial water and is controlled by desorption from sediment particles and dissolved organic matter [16]. Accumulation of benzo(a)pyrene from water was not affected by the simultaneous presence of naphthalene or PCB [17].

Kolok et al. [18] showed that the concentration of benzo(a)pyrene equivalents in shad (*Dorosoma cepedianum*) increases when the fish ventilate water turbid with benzo(a)pyrene spiked sediments. Also the turbid water, not sediment ingestion, appears to be a significant source of benzo(a)pyrene for gizzard shad.

Bioaccumulation of low-molecular-weight PAHs from sediments by *Rhepoxynius abronius* (amphipod) and *Armandia brevis* (polychaete) was similar, however, a large difference in tissue concentration between these two species was measured for high-molecular-weight PAHs including benzo(a)pyrene [19]. Meador et al. [19] concluded that the low-molecular-weight PAHs were available to both species from interstitial water, while sediment ingestion was a much more important uptake route for the high-molecular-weight PAHs. The authors also indicated that bioavailability of the high-molecular-weight PAHs to amphipods was significantly reduced due to their partitioning to dissolved organic carbon.

Summary of Biological Effects Tissue Concentrations for Benzo(a)pyrene

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	**Sediment**	**Water**	**Tissue (Sample Type)**	**Effects**	**Log BCF**	**Log BAF**	**BSAF**	**Reference**	**Comments[3]**
Invertebrates									
Nereis diversicolor, Polychaeta worm	236.6 pmol/g		95.2 pmol/g					[15]	F
Scolecolepides viridis, Polychaeta worm	184.2 pmol/g		119 pmol/g					[15]	F
Leitoscoloplos fragilis, Polychaeta worm	475.8 pmol/g		3540 pmol/g					[15]	F
Thais haemostoma, Snail		BDL	1.45-3.89 µg/kg					[23]	F
Physa sp., Snail		3.39 µg/L	259.6 µg/kg					[20]	L
Dreissena polymorpha, Zebra mussel			3.1 - 4.7 x 10^6 mg/g					[12]	L; depending on the lipid content
Mytilus edulis, Mussel			3.2 mg/kg (whole body)[4]	Physiological, ED50				[30]	L; 50% reduction in feeding, clearance rate, and tolerance to aerial exposure

Summary of Biological Effects Tissue Concentrations for Benzo(a)pyrene

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
			0.161 mg/kg (whole body)[4]	Physiological, LOED				[30]	L; elevated activity of superoxide dismutase (SOD)
			3.2 mg/kg (whole body)[4]	Physiological, LOED				[30]	L; inhibition of superoxide dismutase (SOD) and catalase activity
			3.2 mg/kg (whole body)[4]	Reproduction, LOED				[30]	L, reduced gametogenesis, reproductive success rate
Macoma nasuta, Clam	9.2 ng/g		50 ng/g			-0.07		[12]	F
Macoma nasuta, Clam	4.7 ng/g		1.4 ng/g			-1.30		[21]	F
	70 ng/g		22 ng/g			-0.68		[21]	F
	99 ng/g		45 ng/g			-0.48		[21]	F
	228 ng/g		62 ng/g			-0.70		[21]	F
	440 ng/g		66 ng/g			-0.70		[21]	F
Macomona liliana, Mollusc	3,533 µg/kg OC		189.2 µg/kg lipid				0.0536	[40]	F, %lipid = 2.95; %sed OC = 0.30

Summary of Biological Effects Tissue Concentrations for Benzo(a)pyrene

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
	68,767 µg/kg OC		845.5 µg/kg lipid				0.0123	[40]	F, %lipid = 2.33; %sed OC = 0.73
	2,864 µg/kg OC		166.9 µg/kg lipid				0.0583	[40]	F, %lipid = 2.57; %sed OC = 0.22
	2,440 µg/kg OC		261.8 µg/kg lipid				0.1073	[40]	F, %lipid = 2.04; %sed OC = 0.25
	1,021 µg/kg OC		48.6µg/kg lipid				0.0476	[40]	F, %lipid = 3.13; %sed OC = 0.48
Austrovenus stutchburyi, Mollusc	3,533 µg/kg OC		19.2 µg/kg lipid				0.0054	[40]	F, %lipid = 5.62; %sed OC = 0.30
	68,767 µg/kg OC		24.6 µg/kg lipid				0.0004	[40]	F, %lipid = 5.21; %sed OC = 0.73
	2,864 µg/kg OC		18.8 µg/kg lipid				0.0066	[40]	F, %lipid = 4.85; %sed OC = 0.22
	2,440 µg/kg OC		14.5 µg/kg lipid				0.0059	[40]	F, %lipid = 3.87; %sed OC = 0.25
	1,021 µg/kg OC		11.0 µg/kg lipid				0.0108	[40]	F, %lipid = 4.27; %sed OC = 0.48
Sphaerium corneum, Fingernail Clam			1.25 mg/kg (whole body)[4]	Mortality, NOED				[28]	L; no effect on survivorship in 120 hours

Summary of Biological Effects Tissue Concentrations for Benzo(a)pyrene

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
Corbicula fluminea, Asian Clam	84 µg/kg OC		180.3 µg/kg lipid				2.146	[41]	F, %lipid =0.61; %sed OC = 1.19
	6,387 µg/kg OC		245.9 µg/kg lipid				0.039	[41]	F, %lipid =0.61; %sed OC = 1.19
Mercenaria mercenaria, Quahog Clam,			0.00221 mg/kg (whole body)[4]	Physiological, LOED				[27]	L;impaired ability to clear flavobacterium, exp_conc = < 0.001
			0.00221 mg/kg (whole body)[4]	Mortality, NOED				[27]	L; no effect on mortality, exp_conc = <0.001
Daphnia magna, Cladoceran					3.90 (without organic matter)			[14]	L
Daphnia magna, Cladoceran					3.00 (with organic matter)			[14]	L

Summary of Biological Effects Tissue Concentrations for Benzo(a)pyrene

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
Pontoporeia hoyi, Amphipod	15.5 ng/g		32 ng/g					[16]	L
	410 ng/g	3 ng/mL	600 ng/g			4.74		[22]	L
	40 ng/g	3.5 ng/mL	400 ng/g						
	30 ng/g	2.2 ng/mL	270 ng/g						
Chironomus riparius, Midge	3,920 µg/kg	2,160 ng/L	720 µg/kg					[13]	L
	4,290 µg/kg	1,680 ng/L	252 µg/kg					[13]	L
	4,035 µg/kg	2,640 ng/L	720 µg/kg					[13]	L
Chironomus riparius, Midge			0.23 mg/kg (whole body)[4]	Behavior, NOED				[38]	L; no effect on swimming behavior
			0.09 mg/kg (whole body)[4]	Behavior, NOED				[38]	L; no effect on swimming behavior
			0.04 mg/kg (whole body)[4]	Behavior, NOED				[38]	L; no effect on swimming behavior
Chironomus riparius, Midge			1.9 mg/kg (whole body)[4]	Mortality, NOED				[28]	L; no effect on survivorship in 120 hours
Culex pipiens quinquefasciatus, Mosquito larva		3.39 µg/L	73.1 µg/kg					[21]	L

Summary of Biological Effects Tissue Concentrations for Benzo(a)pyrene

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
Asterias rubens, Starfish			37.8 mg/kg (pyloric caeca)[4]	Physiological, ED100				[29]	L; 346% induction of benzo(a)pyrene hydroxylase activity
			40 mg/kg (whole body)[4]	Physiological, ED100				[29]	L; 346% induction of benzo(a)pyrene hydroxylase activity
			2.15 mg/kg (pyloric caeca)[4]	Physiological, LOED				[29]	L; 200% induction of benzo(a)pyrene hydroxylase activity
			13.2 mg/kg (pyloric caeca)[4]	Physiological, LOED				[29]	L; 200% induction of benzo(a)pyrene hydroxylase activity
			2.5 mg/kg (whole body)[4]	Physiological, LOED				[29]	L; 200% induction of benzo(a)pyrene hydroxylase activity
			10 mg/kg (whole body)[4]	Physiological, LOED				[29]	L; 200% induction of benzo(a)pyrene hydroxylase activity
			0.5 mg/kg (whole body)[4]	Mortality, NOED				[29]	L; no effect on mortality
			10 mg/kg (whole body)[4]	Mortality, NOED				[29]	L; no effect on mortality
			2.5 mg/kg (whole body)[4]	Mortality, NOED				[29]	L; no effect on mortality

Summary of Biological Effects Tissue Concentrations for Benzo(a)pyrene

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
			10 mg/kg (whole body)[4]	Mortality, NOED				[29]	L; no effect on mortality
			40 mg/kg (whole body)[4]	Mortality, NOED				[29]	L; no effect on mortality
			0.053 mg/kg (pyloric caeca)[4]	Physiological, NOED				[29]	L; no effect on benzo(a)pyrene hydroxylase activity
			0.5 mg/kg (whole body)[4]	Physiological, NOED				[29]	L; no effect on benzo(a)pyrene hydroxylase activity
Fishes									
Poeciliopsis monoacha, Viviparius	3.96 µmol/L		8.27 nmol	48-h LC50 3.75 mg/L				[11]	L
Oncorhynchus mykiss (Salmo gairdneri), Rainbow trout		5 µg/egg injection	32,090 cpm (egg) 25,448 cpm (fry)					[24]	L
			14-day 21,839 cpm fry) 35-day 8,922 cpm (fry)						

Summary of Biological Effects Tissue Concentrations for Benzo(a)pyrene

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
Oncorhynchus mykiss, Rainbow trout			0.35 mg/kg (whole body)[4]	Physiological, LOED				[34]	L; hepatic enzyme induction
			30 mg/kg (whole body)[4]	Physiological, LOED				[34]	L; induction of hepatic mixed function oxidases
			12.3 mg/kg (whole body)[4]	Development, NA				[35]	L; gross abnormalities in alevins noted at all test concentrations 0.08 mg/L and above, significant increase relative to the control
			1.93 mg/kg (whole body)[4]	Reproduction, NA				[35]	L; hatchability not significantly reduced
			7.18 mg/kg (whole body)[4]	Reproduction, NA				[35]	L; hatchability not significantly reduced
			10.2 mg/kg (whole body)[4]	Reproduction, NA				[35]	L; hatchability not significantly reduced
			12.3 mg/kg (whole body)[4]	Reproduction, NA				[35]	L; hatchability not significantly reduced

Summary of Biological Effects Tissue Concentrations for Benzo(a)pyrene

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
Cyprinus carpio, Common carp			155 mg/kg (liver)[4]	Physiological, NA				[39]	L; significant increase in EROD enzyme and P450 1A protein content
Gambusia affinis, Mosquito fish		3.39 µg/L	14.1 µg/kg					[20]	L
Lepomis macrochirus, Bluegill sunfish		1 µg/L	39,000 ng/g (gall bladder)		4.15			[25]	L
		1 µg/L	4,600 ng/g (liver)		3.20			[25]	L
		1 µg/L	2,200 ng/g (viscera)		2.89			[25]	L
		1 µg/L	250 ng/g (brain)		1.95			[25]	L
		1 µg/L	370 ng/g (carcass)		2.11			[25]	L
Dorosoma cepedianum, Gizzard shad					3.62			[18]	L

Summary of Biological Effects Tissue Concentrations for Benzo(a)pyrene

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
Dorosoma cepedianum, Gizzard shad			10 mg/kg (whole body)[4]	Physiological, LOED				[37]	L; statistically significant, maximum (11x) induction of ethoxyresorufin-o-deethylase (EROD)
			0.0289 mg/kg (whole body)[4]	Physiological, LOED				[37]	L; statistically significant induction of ethoxyresorufin-o-deethylase (EROD)
			0.0283 mg/kg (whole body)[4]	Physiological, LOED				[37]	L; statistically significant induction of ethoxyresorufin-o-deethylase (EROD)
			50 mg/kg (whole body)[4]	Physiological, NA				[37]	L; 10x induction of ethoxyresorufin-o-deethylase (EROD)
			0.0257 mg/kg (whole body)[4]	Physiological, NA				[37]	L; statistically significant induction of ethoxyresorufin-o-deethylase (EROD)

Summary of Biological Effects Tissue Concentrations for Benzo(a)pyrene

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
			0.0265 mg/kg (whole body)[4]	Physiological, NA				[37]	L; statistically significant induction of ethoxyresorufin-o-deethylaste (EROD)
			0.1 mg/kg (whole body)[4]	Physiological, NOED				[37]	L; no induction of ethoxyresorufin-o-deethylase (EROD)
			0.0337 mg/kg (whole body)[4]	Physiological, NOED				[37]	L; no induction of ethoxyresorufin-o-deethylase (EROD)
			0.0201 mg/kg (whole body)[4]	Physiological, NOED				[37]	L; no induction of ethoxyresorufin-o-deethylase (EROD)
			1 mg/kg (whole body)[4]	Physiological, NOED				[37]	L; no induction of ethoxyresorufin-o-deethylase (EROD)
			0.0239 mg/kg (whole body)[4]	Physiological, NOED				[37]	L; no induction of ethoxyresorufin-o-deethylase (EROD)
			0.0196 mg/kg (whole body)[4]	Physiological, NOED				[37]	L; no induction of ethoxyresorufin-o-deethylase (EROD)

Summary of Biological Effects Tissue Concentrations for Benzo(a)pyrene

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
Ictalurus punctatus, Channel catfish			100 mg/kg (whole body)[4]	Physiological, LOED				[31]	L; significant decrease in neurotransmitter levels
			0.1 mg/kg (whole body)[4]	Physiological, LOED				[32]	L; five to six-fold induction of cytochrome P450
Leuciscus idus, Golden ide			24 mg/kg (whole body)[4]	Mortality, NOED				[33]	L; no effect on survivorship in 3 days
Citharichthys stigmaeus, Sand dab		3 µg/L	130 ng/g (liver), 10 ng/g (gut), 400 ng/g (gill), 30 ng/g (flesh), 150 ng/g (heart)					[25]	L; accumulation within 1 h
Psettichthys melanostictus, Sand sole			2.1 mg/kg (whole body)[4]	Reproduction, ED50				[36]	L; reduced hatching success
			2.1 mg/kg (whole body)[4]	Development, LOED				[36]	L; larval abnormalities

Summary of Biological Effects Tissue Concentrations for Benzo(a)pyrene

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
Oligocottus maculosus, Tidepool sculpins		0.5 µg/L	120 ng/g (liver), 160 ng/g (gut), 200 ng/g (gill), 130 ng/g (flesh), 70 ng/g (heart)					[25]	L; accumulation within 1 h

[1] Concentration units based on wet weight unless otherwise noted.

[2] BCF = bioconcentration factor, BAF = bioaccumulation factor, BSAF = biota-sediment accumulation factor.

[3] L = laboratory study, spiked sediment, single chemical; F = field study, multiple chemical exposure; other unusual study conditions or observations noted.

[4] This entry was excerpted directly from the Environmental Residue-Effects Database (ERED, www.wes.army.mil/el/ered, U.S. Army Corps of Engineers and U.S. Environmental Protection Agency). The original publication was not reviewed, and the reader is strongly urged to consult the publication to confirm the information presented here.

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