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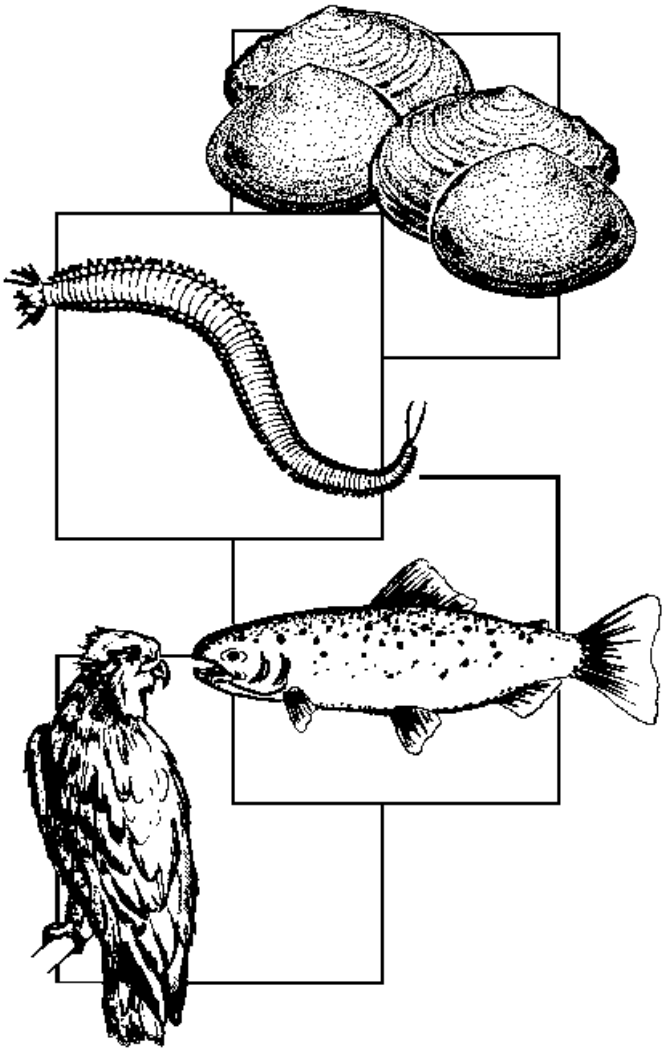
Bioaccumulation Testing And Interpretation For The Purpose Of Sediment Quality Assessment

Status and Needs



BIOACCUMULATION TESTING AND INTERPRETATION FOR THE PURPOSE OF SEDIMENT QUALITY ASSESSMENT

STATUS AND NEEDS

February 2000

U.S. Environmental Protection Agency
Bioaccumulation Analysis Workgroup
Washington, DC 20460

This document has been approved for publication by the U.S. Environmental Protection Agency. Mention of trade names or commercial products does not constitute endorsement or recommendation for use.

This document describes existing knowledge on the use of bioaccumulation data as part of sediment quality assessments. It is not intended to serve as guidance or regulation. This document cannot impose legally binding requirements on EPA, States, Indian tribes, or the regulated community.

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The Bioaccumulation Analysis Workgroup, co-chaired by OW/OST and OSWER/OSW, consists of 40 people from EPA headquarters, regional offices, and research laboratories with scientific and/or programmatic experience related to bioaccumulation testing and interpretation and sediment quality.

Co-Chairs:	**Mike Kravitz**	**OW/OST**
	Stephen Kroner	**OSWER/OSW**
Members:	**Robert Boethling**	**OPPTS/OPPT**
	Robert Jones	**OPPTS/OPPT**
	Ossi Meyn	**OPPTS/OPPT**
	Jerry Smrchek	**OPPTS/OPPT**
	Tom Bailey	**OPPTS/OPP**
	Bryan Wood-Thomas	**OIA**
	Brian Muehling	**OIA**
	Steve Ells	**OSWER/OERR**
	Mark Ralston	**OSWER/OSW**
	Thomas Armitage	**OW/OST**
	Jeffrey Bigler	**OW/OST**
	William Morrow	**OW/OST**
	Mary Reiley	**OW/OST**
	David Redford	**OW/OWOW**
	Sharon Lin	**OW/OWOW**
	Clay Miller	**OW/OWOW**
	Deborah Lebow	**OW/OWOW**
	Laura Phillips	**OW/OWM**
	Catherine Fox	**OECA (presently Region 4)**
	Philip Cook	**ORD/NHEERL (Duluth)**
	Michael Lewis	**ORD/NHEERL (Gulf Breeze)**
	Richard Pruell	**ORD/NHEERL (Narragansett)**
	Cynthia Nolt	**ORD/Headquarters**
	Bruce Boese	**ORD/NHEERL (Corvallis)**
	Marc Tuchman	**GLNPO**
	David Tomey	**Region 1**
	Alex Lechich	**Region 2**
	Douglas Pabst	**Region 2**
	Charles Lobue	**Region 2**
	William Muir	**Region 3**
	Doug Johnson	**Region 4**
	Bonnie Eleder	**Region 5**

John Dorkin	**Region 5**
Amy Mucha	**Region 5**
Philip Crocker	**Region 6**
Monica Young	**Region 6**
Gary Welker	**Region 7**
Brian Ross	**Region 9**
Erika Hoffman	**Region 10**
Justine Barton	**Region 10**
John Malek	**Region 10**

EXECUTIVE SUMMARY

Persistent bioaccumulative chemicals are distributed in sediments throughout the United States, with sediments serving as both a sink and a reservoir for these chemicals. As part of their sediment management decisions, the U.S. Environmental Protection Agency (EPA) and other regulatory agencies are frequently required to interpret the environmental significance from laboratory and field studies. Decisions that require the interpretation of bioaccumulation data are complicated by numerous factors, including variability in chemical bioavailability due to seasonal and physicochemical conditions. It is no longer sufficient to know only whether chemicals accumulate because bioaccumulation itself is not an effect but a process. Regulatory managers must know whether the accumulation of chemicals is associated with or responsible for adverse effects on the aquatic ecosystem and human health. Another complicating factor is that EPA programs have different mandates, often requiring different applications and uses of bioaccumulation data.

EPA prepared this document to serve as a status and needs summary of the use of bioaccumulation data. The document is the result of a collaborative effort among the members of the EPA Bioaccumulation Analysis Workgroup. This document was also prepared to respond to increased interest in the fate and effects of persistent, bioaccumulative, and toxic (PBT) pollutants, as evidenced by the development of EPA's multimedia PBT Strategy.

The purpose of this document is to describe existing knowledge on the use of bioaccumulation data as part of sediment quality assessments. This document:

- Provides a comprehensive summary of existing knowledge on bioaccumulation.
- Provides a compilation of exposure and effects data for persistent, bioaccumulative chemicals.
- Discusses factors that affect the bioavailability of sediment-associated contaminants.
- Identifies how various programs currently use bioaccumulation data for sediment management decisions.
- Identifies issues and research needs for interpreting bioaccumulation data for the purpose of assessing sediment quality.

Factors Affecting Bioavailability

A wide range of physical, chemical, and biological factors have the potential to influence the bioavailability of sediment contaminants. The bioavailability of contaminants in sediment is a function of the type of chemical and the chemical speciation, as well as the behavior and physiology of the organism. The two basic routes of exposure for organisms are transport of dissolved contaminants in pore water across biological membranes, and ingestion of contaminated food or sediment particles with subsequent transport across the gut. For upper-trophic-level species, ingestion of contaminated prey is the predominant route of exposure, especially to hydrophobic chemicals. Uptake through ingestion of or direct exposure to water or sediment can also be important depending on the trophic level of the organism and the physical-chemical characteristics of the contaminant.

Physical Factors

Sediments are dynamic environments with a wide range of interacting processes with variable rates. The rate of mixing in surficial sediment layers by physical processes such as turbulence and bioturbation competes with the rate of sedimentation to determine the depth to which contaminated sediment will be buried. Diffusion and resuspension can also have a large impact on the bioavailability of sediment-associated contaminants either by re-exposing epibenthic filter feeders to contaminated particulates or by increasing the aqueous concentration of a contaminant via desorption from the particulates within the water column.

Chemical Factors

The characteristics of a chemical, such as its molecular size and polarity, determine to a large extent the degree of association of the chemical with particles and thus have an effect on bioavailability. Large, nonpolar chemicals, such as highly chlorinated polychlorinated biphenyls (PCBs), have low aqueous solubilities and a strong tendency to be associated with dissolved and particulate organic matter; therefore, they are less bioavailable, at least to non-sediment-ingestors. Small, ionic species such as certain metals have high aqueous solubilities and tend to be more bioavailable. Even between these extremes, chemical characteristics of contaminants have a large influence on bioavailability.

The concentration of total metals in sediment is generally not predictive of the bioavailability of these elements. Metals concentrations in interstitial water (i.e., pore water) have been correlated with biological effects. For several divalent metals in sediments, acid-volatile sulfide (AVS) appears to have a strong influence on cationic metal activity and toxicity.

For nonionic organic chemicals, the most important factor determining bioavailability is sorption to dissolved and particulate organic matter. Sediment-pore water partitioning of nonionic organic compounds is influenced by the organic carbon content of the sediment. Hydrophobicity is the most important chemical characteristic determining the bioaccumulation behavior of organic chemicals in aquatic systems, although some have suggested that activity coefficients in water are a better estimator. Octanol-water partitioning has become a common method for evaluating the potential of a contaminant to bioaccumulate; however, a major disadvantage of octanol-water partitioning is that experimental determination can be subject to very large measurement errors. It has been demonstrated that bioaccumulation can be predicted from octanol-water partitioning when the partition coefficient (log K_{ow}) lies between 2 and 6. There is also a relationship between the K_{ow} of a chemical and its potential for biomagnification, with uptake efficiency increasing with increasing log K_{ow} for values between 3 and 6. For compounds with a log K_{ow} greater than 6, uptake efficiency begins to decrease. The predictive relationships between K_{ow} and bioaccumulation or biomagnification potentials assume that the compound is not metabolized. If metabolism occurs, these correlations are not applicable, making interpretation more difficult.

Biological Factors

Bioaccumulation is a function of the bioavailability of contaminants in combination with species-specific uptake and elimination processes. Toxicity is determined by the exposure of an animal to bioavailable contaminants in concert with the animal’s sensitivity to the contaminant. These processes have been shown to be a function of the organism's lipid content, size, growth rate, gender, diet, and ability to metabolize or transform a given contaminant, as well as the chemical conditions of the surrounding medium. Other biological factors that can affect contaminant bioavailability include the burrowing and

feeding behavior of the individual organism or species. The depth to which an organism burrows, the type of feeding mechanism it uses (e.g., filter feeding, particle ingestion), the size range of sediment particles it consumes, and its diet all have a large influence on the concentration of contaminant to which the organism will be exposed.

Methods for Assessing Bioaccumulation

Two basic approaches exist to assess bioaccumulation: the first consists of methods that directly measure bioaccumulation, and the second consists of methods that model bioaccumulation. The selection of the appropriate approach is dependent on what questions are being asked, the type of environment, the species, and the contaminants of concern.

The following questions are useful in determining the most appropriate approach for evaluating bioaccumulation:

- *Is the goal to monitor existing bioaccumulation or to predict bioaccumulation under future exposure conditions?*
- *Is the test or model being used as a screening tool or as an accurate predictor?*
- *Is the goal to qualitatively identify which sediment compounds will bioaccumulate or to develop quantitative estimates of tissue concentrations for these compounds?*
- *Will conditions required to reach steady state be met?*
- *Are uptake routes in addition to sediment exposure likely to be important?*
- *Is a goal to determine or predict the time course of uptake and/or elimination?*
- *Is it required that local species be used in the assessment?*
- *Is more than one species to be tested?*
- *Will field or experimentally spiked sediments be used?*

Direct measurement, the simplest approach to assessing bioaccumulation in aquatic organisms, can be conducted using either laboratory-exposed or field-collected organisms. This approach minimizes or eliminates many of the problems associated with modeling. Important issues associated with laboratory measurements of bioaccumulation of chemicals from sediment include selection of an appropriate test species, sediment sampling and handling methods, conditions during exposure to the sediment, exposure duration, and statistical analyses. Measuring bioaccumulation at a particular site requires consideration of which test species to use, whether to examine natural populations or use transplanted populations, and how to compare bioaccumulation occurring under conditions at a potentially contaminated site with that occurring at a reference site.

The two main approaches to bioaccumulation model development are (1) an empirical approach in which laboratory or field data are interpreted to calculate parameters such as bioaccumulation factors (BAFs) and biota-sediment accumulation factors (BSAFs) and (2) a deterministic modeling approach that employs kinetic or equilibrium models in which the mechanistic aspects of bioaccumulation are considered, usually referred to as food web models. Empirical models include bioconcentration factors, BAFs, BSAFs, food chain multiplier, and theoretical bioaccumulation potential. Mathematical models or food web models can be grouped into two categories - equilibrium-based and kinetic approaches. Equilibrium models are usually referred to as “dynamic” because absolute thermodynamic equilibrium

between contaminant concentrations in biota, water, and sediments is rarely reached in a natural setting. Thus, the equilibrium-based models assume steady-state conditions between organisms and the environment. In contrast to equilibrium-based models, kinetic models describe bioaccumulation as the net effect of rate processes (uptake and loss of contaminant). General assumptions of kinetic models include constant uptake rate(s), instantaneous mixing within the compartment(s), and a negative exponential depuration process for all compartments. A newer bioaccumulation assessment tool currently being evaluated for its utility is the critical body residue approach. The critical body residue approach links body burdens in an individual organism to toxicological effects in that organism. Considerable interest exists in using the above tools to identify sediment concentrations associated with threshold tissue concentrations that are protective of aquatic organisms or their predators, including humans.

Important Bioaccumulative Chemicals

The difficulty associated with interpreting bioaccumulation data is exacerbated by a need to address chemical mixtures in sediments as they normally occur. Although progress is being made toward a mixture approach, these types of analyses are in the early stages of development. Hence, the discussion in this document is focused on individual bioaccumulative chemicals. The bioaccumulative chemicals of potential concern listed in this document were selected based on input from the EPA Bioaccumulation Analysis Workgroup and a review of various documents. These chemicals are known to be found in sediment and in animal tissues at levels associated with toxic effects. The document contains information in tabular format for 11 metals, 1 chlorinated phenol, 10 polycyclic aromatic hydrocarbons (PAHs), 13 pesticides, selected dioxins and furans, selected Aroclors and congeners of the PCB group, and total PCBs. Criteria for selecting an initial set of chemicals to be researched and summarized in this document included the following: (1) information was readily available; (2) the chemical was of immediate concern and known to bioaccumulate; (3) the chemical was representative of a group or class of compounds; and (4) the chemical was considered to be important in one or more EPA programs.

The chemical tables (Appendix) summarize information on chemical characteristics, including water solubilities, half-lives, and partition coefficients (log K_{ow} and log K_{oc}); human health concerns; wildlife and aquatic organism partitioning factors; and food chain multipliers. A brief profile of the chemical's toxicity, mode of action, and potential for bioaccumulation is also included. Daily intake levels of concern for the protection of human health were compiled, including estimated values for carcinogenic endpoints (slope factors) and noncarcinogenic endpoints (reference doses) for the oral ingestion exposure pathway, and EPA’s carcinogenic classifications are provided. Factors affecting partitioning of the chemical in relation to wildlife and aquatic organisms, food chain multipliers (biomagnification factors), toxic effects and mode of action, and other information were compiled from various sources.

The data in the chemical summary tables will be useful in addressing the following issues pertaining to bioaccumulation:

- *What species are potentially available for testing?*
- *How should we account for differential partitioning of bioaccumulative contaminants among tissues?*
- *How can bioaccumulation methods be used to assess population-level effects?*
- *How can tissue-specific residue levels be coupled with chronic toxicity response data to develop dose-response relationships for bioaccumulative contaminants?*

Agency Information on Bioaccumulation Data Collection and Interpretation

Bioaccumulation of toxic persistent organic contaminants by aquatic organisms is an ongoing concern for EPA and other agencies. This review identifies a variety of EPA programs that interpret bioaccumulation data to help assess sediment quality. Specialized activities within each of these programs represent a broad spectrum of approaches that address specific statutory mandates as well as the goals of EPA's Contaminated Sediment Management Strategy. Table 1 summarizes the uses of bioaccumulation data by various EPA programs for the interpretation of sediment quality.

Several different activities are overseen by the various agencies, with each activity emphasizing a different need or issue associated with collecting and using bioaccumulation data. Research on bioaccumulation and the use of bioaccumulation to assess sediment contamination are ongoing activities in OPPT, NHEERL, NERL, OERR, OSW, OST, OWOW, the Chesapeake Bay Program, and Regions 5 and 10. OPPT, OSW, OST, and OERR have been working on identifying, based on various chemical properties, chemical structure, and structure-activity relationships, which chemicals might bioaccumulate and cause adverse environmental effects. Bioaccumulation of sediment contaminants in fish that might be consumed by humans is a major concern in the development of water quality criteria for the protection of human health, and research is under way on the most appropriate methods for predicting bioaccumulation in fish and developing chemical-specific BSAFs for setting tissue residue-linked sediment chemical levels for the protection of human health, particularly for mercury.

Assessment procedures that the EPA is focusing on are specific bioaccumulation issues related to assessing contaminated sediments, which include

- The appropriateness of bioaccumulation evaluation methods and interpretation of test results to determine the acceptability of proposed actions.
- Improving consistency in methods used to determine potential bioaccumulative chemicals, effects concentrations for these chemicals, and use of background data to assess bioaccumulation.
- Pesticide evaluation.
- Screening methods and modeling procedures.
- Evaluating human health risks.

OERR, OSW, GLNPO, and a number of international organizations are focusing on the use of bioaccumulation information to identify and evaluate sediments for remediation efforts. Clean-up levels for several Superfund sites have been based on the presence of bioaccumulative contaminants.

Table 1. Summary of Uses of Bioaccumulation Data by Various EPA Programs for the Interpretation of Sediment Quality

Agency Program	Components of Contaminated Sediment Management					
	Research	Assessment	Remediation	Dredged Material Management	Prevention	Outreach
Office of Prevention, Pesticides, and Toxic Substances (OPPTS)						
Office of Pesticide Programs (OPP)		O				
Office of Pollution Prevention and Toxics (OPPT)	O	O			O	O
Office of Research and Development (ORD)						
National Health and Environmental Effects Research Laboratory (NHEERL)	O	O				O
National Exposure Research Laboratory (NERL)	O	O				
Office of Solid Waste and Emergency Response (OSWER)						
Office of Emergency and Remedial Response (Superfund) (OERR)	O	O	O			
Office of Solid Waste (OSW)	O	O	O		O	
Office of Water (OW)						
Office of Science and Technology (OST)	O	O			O	O
Office of Wetlands, Oceans, and Watersheds (OWOW)	O	O	O	O	O	O
Office of Wastewater Management (OWM)					O	
Chesapeake Bay Program (CBP)	O	O	O	O	O	O
Great Lakes National Program Office (GLNPO)		O	O	O		
Office of Air and Radiation (OAR)		O				

The regions are using a number of approaches to target remediation of contaminated sediments; many are evaluating the fish ingestion pathway. Bioaccumulation tests with other organisms (small mammals, clams, amphibians) are also used during the evaluation of ecological risk.

Preventing the release of bioaccumulative contaminants is of concern to OPPTS, OSW, OST, OWM, the Chesapeake Bay Program, OWOW, and numerous countries around the world. OPPT is using its screening and prioritizing procedures to determine those chemicals which might accumulate in sediments and pose risks so that dangerous levels of loadings can be targeted. Under the Waste Minimization National Plan, OSW and OPPT are developing a tool that would prioritize chemicals in hazardous waste based on persistence, bioaccumulation potential, toxicity, and quantity. OWOW and OST use bioaccumulation data in the control of discharges of bioaccumulative chemicals under the CWA. Several international conferences have been held to examine methods for reducing or eliminating persistent organic and metal pollutants, some of which have resulted in agreements.

The dissemination of information pertaining to the bioaccumulation of chemicals has been important for OPPT, OST, OWOW, the Chesapeake Bay Program, NHEERL, and all EPA regions. OPPT recently released a draft multimedia strategy to reduce risks from exposures to priority PBT pollutants through increased coordination among EPA national and regional programs and various stakeholders. OST has published a series of documents on the analysis of fish tissue concentrations of contaminants and the use of such data to determine risks to humans from consumption of fish, as well as providing a database listing all fish and wildlife consumption advisories currently operating in the states. OWOW's outreach efforts include educating the public about the ocean- and coast-related programs and making program information readily available to the public. The toxics characterization of tidal tributaries of the Chesapeake Bay being performed by the Chesapeake Bay Program will be used as an outreach tool to put information in the hands of the public in order to target appropriate areas for additional monitoring and management activities.

Issues and Research Needs for Interpreting Bioaccumulation Data for the Purpose of Sediment Quality Assessment

The interpretation of bioaccumulation data will require an understanding of the relationship between environmental media concentrations, tissue concentrations, and effects on specific organisms or consumers of those organisms. Several issues and research needs have been identified by EPA's Bioaccumulation Analysis Workgroup and attendees of EPA's National Sediment Bioaccumulation Conference (September 11-13, 1996). These issues and needs have been grouped according to topic and include the following:

- Methods—*Laboratory and field methods for assessing bioaccumulation*
  Needs include standardization of methods and approaches, further identification of appropriate species for pathways of concern, and field validation of laboratory methods.

- Chemical Identification—*Identification of bioaccumulative chemicals*
  Needs include identifying a screening procedure for bioaccumulative contaminants, developing additional BAF and BSAF values to assign to known persistent bioaccumulators, or to identify potential ones, and better understanding the bioavailability processes of chemicals in sediments.

- Species Considerations—*Species selection for bioaccumulation testing*
  Needs include guidance on further identification or development of contaminant-specific receptors of choice, tissue residue values for terrestrial and avian wildlife, and additional information on amphibian and wetland species.

- Toxicology—*Dose-response relationships for bioaccumulative contaminants*
  Needs include determining the applicability of the critical body residue (CBR) approach to multiple classes of chemical mixtures, determining differential partitioning of bioaccumulative contaminants among tissues, and conducting further research on how to resolve or compensate for uncertainties related to the assessment of bioaccumulation of sediment-associated contaminants.

GLOSSARY

Note: Numbers in brackets refer to the source of the definition. The citations are provided in the list of references immediately following the glossary.

Acid-volatile sulfides (AVS) — The sulfides removed from sediment by cold acid extraction, consisting mainly of hydrogen sulfide (H_2S) and iron sulfide (FeS). AVS is a possible predictive tool for divalent metal sediment toxicity. [1]

Bioaccumulation — The accumulation of contaminants in the tissue of organisms through any route, including respiration, ingestion, or direct contact with contaminated water, sediment, pore water, or dredged material. [1]

Bioaccumulation factor (BAF) — The ratio of a substance's concentration in tissue of an aquatic organism to its concentration in the ambient water, in situations where both the organism and its food are exposed and the ratio does not change substantially over time. [2]

Bioaccumulative chemicals of concern (BCC) — Chemicals identified as a concern for sediment quality assessment because of their ability to accumulate in the tissue of organisms through any route, including respiration, ingestion, or direct contact with contaminated water, sediment, pore water, or dredged material. [1; modified]

Bioavailable — For chemicals, the state of being potentially available for biological uptake by an aquatic organism when that organism is processing or encountering a given environmental medium (e.g., the chemicals that can be extracted by the gills from the water as it passes through the respiratory cavity or the chemicals that are absorbed by internal membranes as the organism moves through or ingests sediment). In water, a chemical can exist in three different basic forms that affect availability to organisms: (1) dissolved, (2) sorbed to biotic or abiotic components and suspended in the water column or deposited on the bottom, and (3) incorporated (accumulated) into organisms. [3; paraphrased]

Bioconcentration — A process by which there is a net accumulation of a chemical directly from water into aquatic organisms resulting from simultaneous uptake (e.g., by gill or epithelial tissue) and elimination. [4]

Bioconcentration factor (BCF) — The ratio of a substance's concentration in tissue of an aquatic organism to its concentration in the ambient water, in situations where the organism is exposed through the water only and the ratio does not change substantially over time. [2]

Biomagnification — Result of the process of bioconcentration and bioaccumulation by which tissue concentrations of bioaccumulated chemicals increase as the chemical passes up through two or more trophic levels. The term implies an efficient transfer of chemical from food to consumer, so that residue concentrations increase systematically from one trophic level to the next. [4]

Biota-sediment accumulation factor (BSAF) — Relative concentration of a substance in the tissues of an organism compared to the concentration of the same substance in the sediment. [1]

Biotransformation — Enzyme-catalyzed conversion of one xenobiotic compound to another. [5]

Bioturbation — The act of resuspending contaminated fluid or sediment particles into the water column through turbulent activities of biota. [6]

Critical body residue (CBR) — The whole-body concentration of a chemical that is associated with a given adverse biological response. This assumes organisms consist of a single compartment rather than the multiple compartments of which they actually consist, but it has considerable utility as a first approximation of dose. [3]

Desorption — A process that involves the removal of a chemical from a solid to which it is attached or a liquid in which it is dissolved. [7]

Diffusion — (nonbiological) Nonadvective transport due to migration and mixing of dissolved suspended solutes (including particulates) in natural waters in response to concentration gradients. Diffusion can be at the molecular level, due to Brownian motion's producing random movements of the solute's molecules (molecular diffusion), or it can be movements of solutes (including particles) due to turbulent eddies, velocity shear, or bioturbation (turbulent diffusion). Both types of diffusion result in mixing and dispersal of dissolved and bound chemicals. [3]

Dissolved organic carbon (DOC) — The fraction of the organic carbon pool that is dissolved in water and that passes through a 0.45-µm glass fiber filter. DOC quantifies the chemically reactive organic fraction and is an accurate measure of the simple and complex organic molecules that make up the dissolved organic load. [3]

Eh — The measure of the electromotive force of a reaction. [8]

EROD — Ethoxyresorufin O-deethylase catalyzed by cytochrome P4501A (CYP1A proteins) and found in the liver. [9]

Food chain model — A mathematical model that estimates the quantitative transfer of chemicals through the different trophic levels of the food chain. These models vary in complexity, can contain many state variables and parameters, and consider the movement of a chemical through a food chain consisting of one or more trophic levels. These models are typically used with toxic, nonselective, and bioaccumulative chemicals that can affect the entire structure of an ecosystem. [10]

Food chain multiplier — A multiplier that is intended to be applied to the bioconcentration factor of a chemical to estimate the bioaccumulation factor, thereby taking into account accumulation of the chemical up the food chain due to predation. [11]

Food web transfer — Transfer of a chemical from food to consumer, so that residue concentrations increase systematically from one trophic level to the next. [11; paraphrased]

Half-life — Time required to reduce by one-half the concentration of a material in a medium (e.g., soil water) or organism (e.g., fish) by transport, degradation, transformation, or depuration. [4]

Hydrophobic (lipophilic) chemical — A chemical of low water solubility and correspondingly high solubility in lipids or nonpolar solvents. [12]

Hydrophobicity — Partitioning behavior between lipid and aqueous phases. [13]

In situ — In the natural or original position; the use of the field and natural conditions rather than the standardized conditions of laboratory experiments. [14]

Indicator species — A species that may be used as an indicator of changes in environmental quality or conditions. Measured endpoints include the presence, absence, or relative abundance; changes in growth; bioaccumulation of selected chemicals; and changes in reproductive status. [6; modified]

Interstitial water — Water occupying space between sediment or soil particles; also called pore water. [15]

Kinetic models — Models describing chemical reactions and physicochemical processes. [8; paraphrased]

Lethal concentration of 10 out of 100 organisms (LC10) — The concentration of a substance that kills 10 percent of the organisms tested in a laboratory toxicity test of specified duration. [1]

Lethal concentration of 50 out of 100 organisms (LC50) — The concentration of a substance that kills 50 percent of the organisms tested in a laboratory toxicity test of specified duration. [1]

Lipid-water equilibrium partition coefficient (K_l) — The ratio of the concentrations of a given chemical in lipid and in water. [16]

Molecular diffusion — A process of spontaneous intermixing of different substances attributable to molecular motion and tending to produce uniformity of concentration. [15; paraphrased]

Nonionic sorption — A process by which a solute becomes physically or chemically associated with a solid sorbent through nonionic mechanisms. [7; paraphrased]

Octanol-water partition coefficient (K_{ow}) — The ratio of the concentration of a substance in an n-octanol phase to its concentration in the aqueous phase in an equilibrated two-phase n-octanol-water system. The log K_{ow} of a substance represents its likelihood to complex or sorb to organic carbon. The log K_{ow} of a chemical is directly proportional to its n-octanol solubility. [2]

Particulate organic carbon coefficient (P_{oc}) — The fraction of the organic carbon pool that is not dissolved in water, but is retained on a 0.45-µm glass fiber filter. POC is identical to suspended organic carbon (SOC) and is composed of plant and animal organic carbon and organic coating on silt and clay. [3]

Partition coefficient — The ratio of chemical concentrations to two different compartments or phases under steady state conditions. [7]

Reference dose — The estimated dose at which a chemical would be expected to show no effects in a test organism. [11]

Pore water — Water occupying space between sediment or soil particles; also called interstitial water. [15]

Redox potential — The potential of a reversible oxidation-reduction electrode measured with respect to a reference electrode, corrected to the hydrogen electrode, in a given electrolyte. [15]

Resident species — Native, indigenous organisms found at a particular site. [17; modified]

Reversible sediment/pore water partition coefficient (K_p) — Ratio of the concentration of sediment to the concentration of pore water. [18]

Sediment organic carbon-water partition coefficient (K_{soc}) — The ratio of the concentration of chemical in the organic carbon phase to the concentration in water. [3]

Semipermeable membrane devices (SPMD) — A thin film or narrow column of lipid (usually triolein) enclosed in lay-flat or capillary polymeric tubing. Only dissolved or bioavailable organic pollutants diffuse through polymeric films used in SPMDs. [19]

Simultaneously extracted metals (SEM) — divalent metals—commonly cadmium, copper, lead, mercury, nickel, and zinc—that form less soluble sulfides than does iron or manganese and are solubilized during the acidification step (0.5 M HCl for 1 hour) used in the determination of acid-volatile sulfides in sediments. [20]

Slope factors — The slope of the dose-response curve in the low-dose region. When low-dose linearity cannot be assumed, the slope factor is the slope of the straight line from zero dose (and zero excess risk) to the dose at 1 percent excess risk. An upper bound on this slope is usually used instead of the slope itself. The units of the slope factor are usually expressed as 1/(mg/kg-day). [21]

Sorption — A process by which a solute becomes physically or chemically associated with a solid sorbent regardless of the mechanism (e.g., chemisorption, adsorption, absorption). [7]

Steady state — The state in which fluxes of material moving bidirectionally across a membrane or boundary between compartments or phases have reached a balance. An equilibrium between phases is not necessarily achieved. [3]

Superhydrophobic chemicals — Chemicals that are not water-soluble; chemicals that have a strong affinity for lipids (fats). These chemicals are characterized by a log K_{ow} greater than 6. [3; paraphrased]

Theoretical bioaccumulation potential (TBP) — An approximation of the equilibrium concentration in tissue if the material in question were the only source of contaminants to the organisms. [1]

Tortuosity — The quality of having repeated turns or bends; something twisted. [22]

Trophic level — The different feeding relationships in an ecosystem that determine the route of energy flow and the pattern of chemical cycling. [11]

Trophic level transfer — Efficient transfer of chemical from food to consumer, so that residual concentrations increase systematically. [11]

Trophic transfer coefficient — The ratio at which efficient transfer of chemical from food to consumer occurs. [11]

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BIOACCUMULATION TESTING AND INTERPRETATION FOR THE PURPOSE OF SEDIMENT QUALITY ASSESSMENT: STATUS AND NEEDS

1. INTRODUCTION

1.1 Problem Statement

Many federal agencies, including the U.S. Environmental Protection Agency (EPA), U.S. Army Corps of Engineers (USACE), National Oceanic and Atmospheric Administration (NOAA), U.S. Fish and Wildlife Service (FWS), and U.S. Geological Survey (USGS), are tasked with environmental monitoring and assessment issues and are routinely making sediment management decisions using bioaccumulation data. Toxic persistent organic contaminants are distributed in sediments throughout the United States (USEPA, 1996), with sediments serving as both a sink and a reservoir for these chemicals. Decision-making processes predicated on bioaccumulation are complicated by numerous factors, including site-specific issues and the variability in chemical bioavailability due to seasonal, physicochemical conditions, or anthropogenic changes to the environment. It is no longer sufficient to know only whether chemicals accumulate because bioaccumulation itself is not an effect but a process. Regulatory managers must know whether the accumulation of chemicals is associated with or responsible for adverse effects to aquatic organisms and organisms that prey on them, including humans. Another complicating factor is that EPA programs have different mandates, often requiring different applications and uses of bioaccumulation data. This document represents a summary of existing knowledge on the use of bioaccumulation data as part of sediment quality assessments.

1.2 Purpose

A number of sediment assessment methods have been developed to determine the bioaccumulation potential of contaminants in sediments. EPA's Office of Science and Technology and Office of Solid Waste formed a "Bioaccumulation Analysis Workgroup" consisting of 40 headquarters and regional participants. This workgroup has overseen the production of the present "status and needs document," the purpose of which is to provide background information and report on the status of bioaccumulation testing and interpretation in various EPA programs. This document:

- Provides a comprehensive summary of existing knowledge on bioaccumulation.
- Compiles exposure and effects data for persistent, bioaccumulative chemicals.
- Discusses factors that affect the bioavailability of sediment-associated contaminants.
- Identifies how various programs currently use bioaccumulation data for sediment management decisions.
- Identifies issues and research needs for interpreting bioaccumulation data for the purpose of assessing sediment quality.

This document is not intended to provide all that is known on bioaccumulation, but rather is a comprehensive summary of current knowledge and limitations, issues, and needs. It is an attempt to

identify, categorize, and enumerate effects resulting from the accumulation of chemicals. The document is intended to be a synopsis of tools in the regulator's toolbox.

1.3 Scope of the Document

This document is organized into two parts - the main text, which provides a summary of existing knowledge, discusses bioaccumulation processes, and reviews EPA's uses of bioaccumulation data and issues associated in that process, and an appendix containing the exposure and effects data. The following specific elements are contained within the respective chapters:

- Chapter 2 discusses factors that affect the bioavailability of sediment-associated contaminants.
- Chapter 3 describes methods and techniques that have been developed for measuring and modeling bioaccumulation.
- Chapter 4 identifies important bioaccumulative chemicals, presents an overview of the information contained in the chemical-specific summary tables (Appendix), and describes how the data were obtained.
- Chapter 5 presents brief synopses of current research on and uses of bioaccumulation data in several EPA programs.
- Chapter 6 summarizes further research needs for interpreting the bioaccumulation of persistent organic pollutants to assist in protecting aquatic and terrestrial biota and humans from toxic effects of bioaccumulative chemicals in sediments.
- The Appendix is a compilation of chemical-specific summary tables that represent bioaccumulation research conducted during the past 12 years. The summary tables contain information associating the presence and quantity of potentially bioaccumulative chemicals in sediment with uptake in the tissues of aquatic and terrestrial organisms and with the effects of those chemicals on the organisms.

1.4 Regulatory Uses

A brief synopsis of possible uses of bioaccumulation data in EPA programs under a variety of statutes is presented below. More detailed information on how bioaccumulation data are used by various programs is provided in Chapter 5. Typical applications of bioaccumulation guidance might be the characterization of sediment contamination at Superfund sites, the verification of contaminants of concern in sediment for purposes of NPDES permitting, and the selection of disposal options for dredged material.

The Office of Enforcement and Compliance Assurance (OECA) is responsible for developing and implementing enforcement and compliance assurance strategies for the National Environmental Policy Act (NEPA) and other federal regulations. Consequently, it may use bioaccumulation data under a broad range of statutes to determine the environmental acceptability of proposed federal actions.

The Office of Prevention, Pesticides, and Toxic Substances (OPPTS) uses the results of bioaccumulation tests to support review of new and existing chemicals under the Toxic Substances Control Act (TSCA) and the registration/re-registration of chemicals under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA). In addition bioaccumulation information may be used to provide guidance on the design of new chemicals to reduce bioavailability and partitioning of toxic chemicals to sediment.

The Office of Solid Waste and Emergency Response (OSWER) is responsible for controlling hazardous wastes and remediating hazardous waste sites under the Resource Conservation and Recovery Act (RCRA) and the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA). Under CERCLA, the Office of Emergency and Remedial Response (OERR)—the Superfund Program—uses sediment assessment methods, including bioaccumulation data, as a standard part of initial sampling during the preliminary site assessment and the more in-depth Remedial Investigation/Feasibility Study for Superfund sites where sediment contamination might be present. Under RCRA, the Office of Solid Waste (OSW) has proposed the Hazardous Waste Identification Rule (HWIR; USEPA, 1999). The proposed rule addresses listed hazardous wastes, as well as mixtures of and residues derived from managing the hazardous wastes, that pose low risks to human health and the environment. The rule provides the framework for establishing chemical-specific concentrations in wastes that may not be exceeded to be eligible for a self-implementing exemption from the hazardous waste management system requirements under Subtitle C of RCRA. The framework includes a risk-based methodology that is being developed for use as the basis for the exit concentrations. The methodology considers the bioaccumulative potential of relevant chemicals in the evaluation of potential exposures from multiple pathways, in multiple media, and from a variety of waste management units.

In response to the Hazardous and Solid Waste Amendments of 1984 (HSWA), which amended RCRA, and the Pollution Prevention Act of 1990 (PPA), EPA released the Waste Minimization National Plan (WMNP) in November 1994. The WMNP focuses on reducing the generation and subsequent release to the environment of the most persistent, bioaccumulative, and toxic chemicals in hazardous wastes. One of the objectives of the WMNP was to develop a flexible risk-based screening tool that would assist stakeholders in identifying source reduction and recycling priorities. EPA committed to fulfill this objective by developing a tool that would prioritize chemicals based on their persistence, bioaccumulation potential, toxicity, and quantity. This screening tool, the Waste Minimization Prioritization Tool (WMPT), has been developed by OSW and the Office of Pollution Prevention and Toxics (within OPPTS). EPA used WMPT scores for thousands of chemicals to develop a RCRA PBT Chemical List, which includes chemicals of greatest concern to the RCRA program on a national basis (USEPA, 1998). EPA will use the RCRA PBT List to focus attention on actions that reduce the generation of these chemicals in RCRA hazardous waste by fifty percent by 2005.

The Office of Water (OW) is responsible for EPA's water quality activities, which represent a coordinated effort to restore the nation's waters. The functions of this program include developing national programs, technical policies, and regulations relating to drinking water, water and sediment (including dredged material) quality, and ground water; establishing environmental and pollution source standards; and providing for the protection of wetlands. In addition, this office furnishes technical direction, support, and evaluation of regional water activities; enforces standards; and develops programs for technical assistance and technology transfer. OW oversees the provision of training in the fields of water quality, economic and long-term environmental analysis, and marine and estuarine protection.

OW and the U.S. Army Corps of Engineers (USACE) developed joint technical guidance for evaluating the potential for contaminant-related impacts associated with the discharge of dredged material in the ocean under the Marine Protection, Research, and Sanctuaries Act (MPRSA) (USEPA and USACE, 1991). Similar updated guidance has been published for evaluating dredged material discharges in fresh, estuarine, and saline (near-coastal) waters under section 404 of the Clean Water Act (CWA) (USEPA and USACE, 1998). These documents employ a tiered testing protocol in which bioaccumulation data figure prominently.

Under sections 301, 304, 306, and 307 of the CWA, the Office of Science and Technology (OST) within OW promulgates technology-based national effluent limitations guidelines that control the discharge of toxic chemicals and other pollutants by categories of industrial dischargers. Bioaccumulation data and modeling are used in support of this effort.

In response to the Water Resources Development Act (WRDA) of 1992 requirement that EPA conduct a national survey of data regarding sediment quality in the United States, OST prepared *The National Sediment Quality Survey* (NSQS) (USEPA, 1997). For calculations related to bioaccumulation, the survey makes use of fish tissue residue data and models bioaccumulation from sediment using the theoretical bioaccumulation potential approach. A national database containing information in the NSQS, the National Sediment Inventory, will be maintained and updated on a regular basis so that it can be used to assess trends in both sediment quality and the effectiveness of existing regulatory programs at the federal, state, and local levels.

Section 403 of the CWA requires determination of the quantities of and potential for bioaccumulation of released chemicals, the potential for pollutant transport, potential harm to biological communities, and direct and indirect effects on humans. *CWA Section 403: Procedural and Monitoring Guidance* (USEPA, 1994), developed by the Office of Wetlands, Oceans and Watersheds (OWOW) within OW discusses the qualities of target species and methods for assessing bioaccumulation; monitoring program design, including sampling of caged or indigenous indicator species; the type of tissue to be analyzed in invertebrates and fishes; and techniques for extracting and analyzing chemical contaminants. USEPA (1995a) provides additional information on some of these topics.

EPA's National Estuary Program (NEP), authorized under CWA section 320, is a national demonstration program that uses a comprehensive watershed management approach to address water quality and habitat problems in designated estuaries on the Atlantic, Gulf, and Pacific coasts and in the Caribbean. OWOW developed guidance for this program (USEPA, 1992b), which is similar to that for section 403 (above) and which includes the design and conduct of bioaccumulation monitoring studies to link exposure and effects and to examine risks to target species and humans.

Section 402 of the CWA authorizes the National Pollutant Discharge Elimination System (NPDES) permitting program, administered by the Office of Wastewater Management (OWM) within OW, to regulate the discharge of pollutants from point sources into navigable waters. Bioaccumulation screening methods can be used to identify chemicals of potential concern in the sediments, followed by chemical-specific analysis for confirmatory purposes. Until the states adopt numeric criteria into their standards for sediment contaminants based on bioaccumulation, the NPDES program does not require permitting authorities to include, in their NPDES permits, sediment bioaccumulation-based numeric limits. However, states have the discretion to include such limits in permits based on an interpretation of their narrative standards for toxics. To establish such permit limits, it will be necessary for permitting authorities to develop Waste Load Allocations (WLAs) for the relevant sediment contaminants.

Section 118(c)(2) of the CWA (Public Law 92-500 as amended by the Great Lakes Critical Programs Act of 1990 [CPA], Public Law 101-596, November 16, 1990) required EPA to publish proposed and final water quality guidance on minimum water quality standards, antidegradation policies, and implementation procedures for the Great Lakes System. In response to these requirements, EPA developed the *Final Water Quality Guidance for the Great Lakes System* (USEPA, 1995b). The guidance incorporates bioaccumulation factors (BAFs) into the derivation of criteria and values to protect human health and wildlife.

Section 118(c)(3) established the Assessment and Remediation of Contaminated Sediments (ARCS) Program to assess the extent of sediment contamination in the Great Lakes and to demonstrate bench- and pilot-scale treatment technologies for contaminated sediment. Under the ARCS Program, the Great Lakes National Program Office (GLNPO) used bioaccumulation data and models to estimate comparative human health risks associated with direct and indirect exposures to contaminated sediments in the lower Buffalo River under selected remedial alternatives. It was shown that risks could be reduced under the different remedial alternatives compared to no action, particularly if dredging was the selected option.

Sediment Management Standards (SMS) for the state of Washington were promulgated by the Washington State Department of Ecology under Chapter 173-204 WAC in March 1991. The purpose of these standards is to "reduce and ultimately eliminate adverse effects on biological resources and significant human health threats" resulting from contaminated sediments. The state of Washington is developing human health sediment quality criteria for bioaccumulative compounds in Puget Sound sediments, which will be incorporated into the state's existing SMS. These criteria are based on standard risk assessment methodologies in conjunction with empirically derived biota-sediment accumulation factors (BSAFs).

1.5 Definitions

The terms *bioconcentration*, *bioaccumulation*, and *biomagnification* are sometimes used interchangeably in the literature, but they have distinct meanings in this document. The following definitions represent the most accepted usage of these terms. These definitions apply throughout this document.

- Bioconcentration is defined as the process by which there is a net accumulation of a chemical directly from water into aquatic organisms resulting from simultaneous uptake (e.g., by gill or epithelial tissue) and elimination.
- Bioaccumulation is defined as the accumulation of chemicals in the tissue of organisms through any route, including respiration, ingestion, or direct contact with contaminated water, sediment, and pore water in the sediment.
- Biomagnification is the result of the process of bioconcentration and bioaccumulation by which tissue concentrations of bioaccumulated chemicals increase as the chemical passes up through two or more trophic levels. The term implies an efficient transfer of chemical from food to consumer, so that residue concentrations increase systematically from one trophic level to the next.

Another important concept is *trophic transfer*. Trophic transfer is simply the movement of contaminants from one trophic level, i.e., prey, to another trophic level, i.e., predators (Schwarz and Lee, 1980). Trophic transfer can result in either bioaccumulation or biomagnification. A chemical's capacity for trophic transfer can be measured in terms of the trophic transfer coefficient (TTC), which is determined by dividing the concentration of a contaminant in a predator's tissue by the concentration of that contaminant in its prey (Suedal et al., 1994). Trophic transfer has been noted as a significant source of chemicals to predator species such as fish, and fish tissue levels are dependent primarily on the ability of the organism to excrete or store the contaminant (Bryan, 1979). Biomagnification is said to occur when a TTC is greater than one. TTCs below one indicate contaminants that do not biomagnify, although bioaccumulation is still possible for these substances.

Biomagnification is of concern to those tasked with assessing sediment quality because of the potential for apparently low, no-effects concentrations of chemicals in abiotic media to elicit adverse effects in higher-trophic-level species. The fact that some higher-trophic-level species can biomagnify certain chemicals makes it crucial that the pathways and mechanisms of transfer be understood. A great deal of information and numerous measurements are involved in determining biomagnification, including information regarding an organism's position in a complex food web, predator-prey relationships, feeding habits, age and sex of the sampled organisms, tissue chemistry data, lipid composition, and gut content analyses. In many cases, studies attempting to assess biomagnification have been plagued by poor experimental design, resulting in a high degree of variability (Suedel et al., 1994). These results have limited the ability to determine whether trophic transfer and biomagnification were occurring.

Bioconcentration, biomagnification (Connell, 1989), and bioaccumulation operate with most aquatic organisms, with bioaccumulation and biomagnification the most likely processes by which air-breathing aquatic animals (e.g., seals, whales, and dolphins) and semiaquatic species (e.g., aquatic birds) acquire chemicals. These higher-trophic-level species lack an organism-to-water exchange interface, making it difficult for the bioconcentration mechanism to operate (Connell, 1989). Bioconcentration may be the sole process for organisms that draw their food and oxygen requirements from dissolved components in the water mass (i.e., phytoplankton and some bacteria) (Connell, 1989). For many bioaccumulative compounds, the principal route of movement into and through aquatic food webs appears to be dietary ingestion rather than bioconcentration from water because these compounds generally exhibit low water solubility and tend to concentrate in the lipid fractions of biological tissues (Suedel et al., 1994). Therefore, the principal pool of these compounds available to upper-trophic-level consumers is from dietary items rather than from abiotic media (Suedel et al., 1994). Biomagnification is most likely to occur with persistent compounds having log K_{ow}s greater than 5, and with organisms that have long lives and probably are among the top predators (Connell, 1989; Suedel et al., 1994). Organisms that cannot excrete or otherwise regulate contaminants can readily biomagnify these materials with age (Bryan, 1979).

Many metals show a potential for trophic transfer via uptake from food, but not in sufficient quantities to result in biomagnification. Those metals which show a propensity to biomagnify include arsenic, methylmercury, and perhaps inorganic mercury (Suedel et al., 1994). Biomagnification of methylmercury, relative to inorganic mercury, has been attributed to higher lipid solubility and an ability to transfer across membranes, long biological half-life, and long life span of top predators, such as aquatic birds (Bryan and Langston, 1992; Huckabee et al., 1979). For organic compounds, PCBs, DDT, DDE, and toxaphene have shown the greatest potential to biomagnify in aquatic ecosystems (Suedel et al., 1994).

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2. FACTORS AFFECTING BIOAVAILABILITY

2.1 Overview

A wide range of physical, chemical, and biological factors have the potential to influence the bioavailability of sediment contaminants. This chapter presents a brief summary of some of the more important of those factors. The distinction between these categories is somewhat artificial. In reality, complex interactions between physical, chemical, and biological factors result in changes in contaminant bioavailability. A summary of important factors is given in Table 2.1. This discussion is not intended to be a definitive review of all factors that potentially affect contaminant bioavailability. Several reviews have outlined the major factors affecting the bioavailability of sediment contaminants, including Landrum and Robbins (1990), Hamelink et al. (1992), and others.

2.2 Routes of Exposure

The observed bioavailability of sediment contaminants is the result of the chemical and environmental speciation of the contaminant as well as the behavior and physiology of the organism. The two basic routes of exposure for organisms are transport across biological membranes exposed to dissolved contaminants in sediment pore water and the ingestion of contaminated sediment particles. Exposure to dissolved contaminant concentrations in sediment pore water appears to be the predominant route of exposure for most benthic organisms (Muir et al., 1985; Oliver, 1987; Shaw and Connell, 1987). However, exposure due to sediment ingestion might be a significant route of exposure for some species (Landrum, 1989; Harkey et al., 1994a; Meador et al., 1995; and others) and appears to be the predominant pathway for some benthic species, such as the oligochaete *Lumbriculus variegatus* (Leppanen and Kukkonen, 1998).

For upper-trophic-level species, ingestion is clearly the predominant route of exposure, especially to hydrophobic chemicals, although uptake through water and sediment ingestion or exposure can also be important depending on the trophic level of the organism and the physical-chemical characteristics of the contaminant.

2.3 Physical Factors

Sediments are dynamic environments characterized by a wide range of competing processes with variable rates. The rate of mixing in surficial sediment layers by physical processes such as turbulence and bioturbation competes with the rate of sedimentation to determine the depth to which contaminated sediment will be buried. The net effect of these processes can be modeled in a variety of fate and transport models, such as those developed by the USEPA-Athens laboratory, (e.g., WASP5). The concentration profile within the sediment is very important to bioavailability and subsequent potential for bioaccumulation because it determines the chemical environment of the contaminated sediment and the physical availability of the contaminant to biological organisms (i.e., whether organisms will be in contact with the contaminant).

Diffusion processes also affect the mobility and bioavailability of sediment-associated contaminants. Diffusion processes are driven by concentration gradients that can be established within sediment pore waters as well as between sediment pore waters and the overlying water column. Diffusion in sediments is limited by the twisted path among sediment particles (tortuosity), as well as molecular diffusion limits.

Table 2-1. Summary of Factors Influencing Bioavailability of Sediment-associated Chemicals

Physical Factors	Chemical Factors	Biological Factors
• Rate of mixing • Rate of sedimentation • Diffusion • Resuspension	• AVS concentrations for Cu, Cd, Pb, Ni, Zn • Redox conditions • pH • Interstitial water hardness • Sediment organic carbon content • Dissolved organic carbon content • Organic-water equilibration constants for organic compounds • Organic matter characteristics • Equilibration time with sediment	• Biotransformation • Bioturbation • Organism size/age • Lipid content • Gender • Organism behavior • Diet, including sediment ingestion, feeding mechanism • Organism response to physicochemical conditions

Resuspension can also have a large impact on the bioavailability of sediment-associated contaminants by either reexposing epibenthic filter feeders to contaminated particulates or increasing the aqueous concentration of a contaminant through desorption from the particulates within the water column (Landrum et al.,1994).

2.4 Chemical Factors

Chemical conditions determine to a large extent the degree of association with particles (e.g., sorption, desorption, precipitation, dissolution). This association is important because the potential bioavailability of sediment-associated chemicals such as divalent metals and nonionic organic compounds is primarily related to the amount of chemical freely available in the interstitial (pore) water of the sediment.

In general, chemical characteristics, such as molecular size and polarity, have a large influence on bioavailability. Large, nonpolar chemicals such as highly chlorinated PCBs have low aqueous solubilities and a strong tendency to be associated with dissolved and particulate organic matter and thus are less bioavailable (at least to non-sediment ingestors). In contrast, small, ionic species such as certain metals, especially alkali or alkaline earth metals like sodium (Na^+) and calcium (Ca^{2+}), have high aqueous solubilities and therefore tend to be more bioavailable. Even between these extremes, chemical characteristics of contaminants have a large influence on bioavailability. In the following subsections, the specific chemical factors important in controlling the pore water concentrations of divalent metals and nonionic organic compounds are discussed.

2.4.1 Metals

Key factors that affect the partitioning and speciation, and thus the bioavailability, of sediment metals include the redox conditions (i.e., whether the conditions are oxic or anoxic), pH, interstitial water hardness, organic carbon content of the sediment, and pore water concentration of dissolved organic carbon. The redox conditions and pH influence the oxidation state and the dissolved speciation of the metal. Metals exhibit a range of reactivities with respect to complexation with both organic and inorganic phases present in the sediment, resulting in varying concentrations of dissolved versus particulate metals.

Total sediment metals concentrations are usually not predictive of the bioavailability of these trace elements. However, metals concentrations in interstitial water (pore water) have been correlated with biological effects (Swartz et al, 1985; Kemp and Swartz, 1988; and others). For several divalent metals, a key partitioning phase controlling cationic metal activity and toxicity in sediments appears to be acid-volatile sulfide (AVS), which is an operationally defined fraction of sulfide minerals of which iron sulfide (FeS) predominates (Di Toro et al. 1990; Carlson et al. 1991; Di Toro et al. 1992; and others). Simultaneously extracted metals (SEM) and AVS measurements can be made to assess the potential bioavailability of SEM metals (Cd, Cu, Pb, Ni, and Zn). The SEM/AVS theory assumes that the concentration of metals dissolved in pore water within the sediment (to which benthic species would be exposed) is related to the abundance of a "controlling phase" in the sediment (i.e., AVS). SEM are theoretically defined as metals that form more stable minerals with sulfide than does Fe (e.g., Cd, Cu, Ni, Zn, Pb).

The SEM/AVS model states that if the AVS concentration is less than the concentration of SEM, toxicity will be observed (Di Toro et al., 1990). In other words, if the SEM/AVS ratio is greater than 1, sufficient AVS is not available to bind all the SEM and benthic organisms *might* be exposed to toxic concentrations of metals. In contrast, if the ratio is less than 1, sufficient AVS exists to bind all SEM and adverse effects in benthic species are not expected. Another way of expressing this relationship is through a difference (i.e., SEM – AVS). This approach gives a quantitative measure of the metal available, in excess of AVS, instead of just a ratio that can be high even in lightly contaminated sediment if the AVS levels are low (Hansen et al. 1996; Ankley et al. 1996).

While SEM/AVS theory has successfully predicted the toxicity of sediment contaminated with Cd and Ni (Ankley et al., 1991; Carlson et al., 1991) and zinc and lead (Casas and Crecelius, 1994), success predicting the toxicity of Cu-contaminated sediments has been mixed (Ankley et al., 1993).

There are at least two possible explanations for the mixed results of the AVS theory: (1) other solid phases (e.g., iron and manganese oxides) and complexing ligands (e.g., natural organic matter) might successfully compete for dissolved metals in sediments or (2) organisms might alter the condition of their immediate environment, thereby exposing themselves to conditions different from those measured in the bulk sediment (e.g., different AVS concentrations or pH).

The bioavailability of mercury, although theoretically mercury is a SEM metal, appears to be controlled more by methylation than by AVS concentrations. Mercury commonly occurs in three chemical forms in the environment—elemental mercury (Hg^0), inorganic mercury (Hg^{+1}, Hg^{+2}), and organic mercury (e.g., methylmercury, phenylmercury). Methylmercury compounds are extremely toxic and are efficiently bioaccumulated through aquatic food chains. Methylmercury is formed in aquatic sediments due to microbial methylation of inorganic mercury. Sulfate-reducing bacteria appear to be particularly efficient methylators in sediment systems. The relationship between total mercury and methylmercury concentrations in water appears to be quite variable. In a recent study of aqueous samples collected

from several streams, an experimentally flooded wetland, and peat pore water, no obvious relationships between total mercury and methylmercury concentrations were observed. Methylmercury as a percent of the total mercury concentration ranged from less than 1 percent to over 90 percent (Kelly et al., 1995). Therefore, direct measurement of methylmercury concentrations might be necessary to determine the bioavailability of mercury.

2.4.2 Nonionic organic compounds

The most important factor determining the bioavailability of nonionic organic chemicals is sorption to dissolved and particulate organic matter. Sediment-pore water partitioning of nonionic organic compounds is influenced by the organic carbon content of the sediment; thus, $K_p = K_{oc} \times f_{oc}$, where K_p represents the reversible sediment/pore water partition coefficient, K_{oc} is the particle organic carbon coefficient, and f_{oc} is the fraction of organic carbon in the sediment (kg organic carbon/kg dry weight of the sediment).

Octanol-water partitioning has become a common method for evaluating the potential of a contaminant to bioaccumulate (Donkin, 1994). Hydrophobicity is the most important chemical characteristic determining the bioaccumulation behavior of organic chemicals in aquatic systems, although some have suggested that activity coefficients in water are a better estimator (Nagel, 1991). Octanol is not a perfect model for biological lipids. A major disadvantage of octanol-water partitioning is that experimental determination can be subject to very large measurement errors. The octanol-water partition coefficient (K_{ow}) is the ratio of a chemical's solubility in n-octanol and water at equilibrium. A key parameter in determining K_{ow} values, water solubility, is subject to large experimental errors, particularly for the very hydrophobic molecules of greatest environmental interest (Donkin, 1994). An EPA workgroup is developing standardized log K_{ow} values for selected chemicals based on a review of available laboratory measurement and modeling data.

Connell (1991) has shown that bioaccumulation can be predicted from octanol-water partitioning when the log K_{ow} lies between 2 and 6. Chemicals with a log K_{ow} less than 2 usually bioconcentrate more than would be expected from their K_{ow} values; chemicals with a log K_{ow} greater than 6 tend to bioconcentrate less than expected (Connell, 1991).

Thomann (1989) extensively evaluated the relationship between the K_{ow} of a chemical and its potential for biomagnification. Uptake efficiency increases with increasing log K_{ow}, reaching a maximum when log K_{ow} is between 3 and 6 (depending on an organism's size). For compounds with a log K_{ow} greater than 6, uptake efficiency begins to decrease. Thomann also concluded that biomagnification through the food chain is unlikely to occur for chemicals with a log K_{ow} less than 5, but is likely for chemicals with log K_{ow} between 5 and 6.5. Biomagnification remains important for chemicals with log K_{ow} values up to 8, although other factors, such as top predator growth rates and bioconcentration by phytoplankton, take on greater significance (Thomann, 1989). It is important to note that the predictive relationships between K_{ow} and bioaccumulation or biomagnification potentials assume that the compound is not metabolized. If metabolism occurs, these correlations are not applicable.

The nature of the sediment organic matter also appears to affect the observed sediment-pore water partitioning of nonpolar organic contaminants. Enhanced partitioning has been observed in heavily contaminated sediments relative to uncontaminated sediments (Boyd and Sun, 1990; Chin and Gschwend, 1992). The high anthropogenic organic content of these sediments appeared to provide a more effective sorbent for nonpolar organic compounds than did natural sediment organic matter. Studies of differences in partition coefficients between soil and sediment organic matter suggest that

higher partition coefficients measured for sediment organic matter relative to soil organic matter were due to the relative polarity of soil organic matter relative to the sediment organic matter (Grathwohl, 1990; Kile et al. 1995).

Finally, the results of several laboratory studies suggest that increased equilibration time between contaminants and sediments results in decreased bioavailability of organic contaminants to benthic organisms (McElroy and Means, 1988; Landrum, 1989). Several mechanisms have been proposed to explain the observed decreases in bioavailability including contaminant diffusion into less bioavailable sediment compartments and removal of ingestible particles through packaging into fecal pellets (Landrum, 1989).

2.5 Biological Factors

The degree of bioavailability of the contaminant under various exposure scenarios determines the *potential* for bioaccumulation and toxicity. Toxicity ultimately is determined by the exposure of an animal to bioavailable contaminants in concert with the animal's sensitivity to the contaminant. Bioaccumulation is a function of the bioavailability of contaminants in combination with species-specific uptake and elimination processes. These processes have been shown to be a function of the organism's lipid content (Landrum, 1988), size (Landrum et al., 1992), growth rate, gender, diet, and ability to metabolize or transform a given contaminant, as well as the chemical conditions of the surrounding medium. For example, conditions such as temperature and dissolved oxygen concentrations can affect the observed contaminant uptake rate by influencing an organism's metabolic rate and thus the respiration and ventilation rate. Spigarelli et al. (1983) found that ambient temperature affected PCB bioaccumulation in brown trout by affecting food consumption, growth, and lipid content. Connell and Miller (1984) reported that low dissolved oxygen concentrations can increase the ventilation rate of aquatic organisms, which may result in increased uptake rates.

Bioturbation of sediments by benthic organisms can alter the physical and chemical characteristics of the sediment. A recent study of the effects of bioturbation on the bioavailability of cationic metals showed that bioturbation can enhance the bioavailability of some cationic metals in surficial sediments. Burrowing activity of the oligochaete *Lumbriculus variegatus* significantly reduced AVS concentrations in surficial sediments and resulted in elevated interstitial water concentrations of cadmium (Peterson et al., 1996).

The physical reworking of sediment in laboratory microcosms by oligochaetes has been shown to result in enhanced contaminant concentrations (i.e., hexachlorobenzene, pentachlorobenzene, and trifluralin) in the surface sediments (Karickhoff and Morris, 1985). This reverse "conveyor belt" type of bioturbation occurs when fine sediments are ingested at depth and egested as fecal material in the sediment surface. The rate of contaminant transport as a result of bioturbation is independent of the chemical characteristics of the sorbed contaminants and is much more rapid than the chemical transport of these compounds through diffusion and sorption processes. However, the release of the reworked contaminants to the overlying water was less than that predicted due to slow contaminant desorption from fecal pellets (Karickhoff and Morris, 1985).

The degree of biotransformation or biodegradation of a contaminant may be strongly influenced by the degree to which the compound is associated with sediment particles. Shimp et al. (1990) stated that modeling chemical residence time in sediments is more difficult than modeling biodegradation in other media because of the influence of partitioning (which determines the proportion of a discharged mass that reaches the sediments), the rate at which sediment particles settle to the bottom, the rate at which particles can be buried, and the extent to which particles are resuspended and transported downstream.

They also noted that biodegradation of a chemical mass adsorbed to sediment particles can change when the particle is buried and the environment becomes anoxic or anaerobic. Boethling et al. (1995) found that contaminant adsorption to sediment particles resulted in enhanced biodegradability.

Other biological factors that can affect contaminant bioavailability include the burrowing and feeding behavior of the individual organism or species. The depth to which an organism burrows, the type of feeding mechanism it uses (e.g., filter feeding, particle ingestion), the size range of the sediment particles it consumes (Harkey et al., 1994b), and its diet all have a large influence on the concentration of contaminant to which the organism will be exposed.

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3. METHODS FOR ASSESSING BIOACCUMULATION

3.1 Introduction

The factors controlling the partitioning of a contaminant in the environment and its movement through the food chain are as complex as the factors controlling its toxicity once it has been taken up by an organism. This chapter describes a number of approaches that are potentially useful tools for assessing bioaccumulation. It has been divided into two main sections, with the first describing methods to measure bioaccumulation directly and the second describing methods to model bioaccumulation. The selection of the appropriate approach is dependent on what questions are being asked, the type of environment, the species, and the contaminants of concern.

Because of the uncertainties associated with the use of predictive bioaccumulation and biomagnification models, EPA prefers that the bioaccumulation potential of a chemical be determined by direct field measurements, although the Agency recognizes that in many cases direct measurements are not possible (USEPA, 1996a). As part of the Great Lakes Water Quality Initiative (USEPA, 1993a; 1995a), EPA listed three possible approaches for determining bioaccumulation factors (BAFs), in order of the Agency's preference:

1. A field-measured BAF, using animals living at or near the top of the food chain.

2. A laboratory-measured BCF, multiplied by an appropriate factor (food chain multiplier, or FCM), reflecting the difference between the laboratory organisms and the field organisms.

3. A BCF modeled from a chemical's octanol-water partitioning coefficient, multiplied by an appropriate FCM.

The following questions, as presented in Boese and Lee (1992), are useful in determining the most appropriate approach for evaluating bioaccumulation. Although this list of questions was developed for bedded sediment tests, the questions are generally applicable to any type of direct measurement of bioaccumulation.

- *Is the goal to monitor existing bioaccumulation or to predict bioaccumulation under future exposure conditions?*

Direct measurement of bioaccumulation will give a snapshot of the tissue residues at the time and location of sampling. Direct measurement can be accomplished either by measuring tissue concentrations in field-collected organisms or by conducting laboratory bioaccumulation tests using field-collected sediments. On the other hand, predictive modeling can also be used to estimate the extent and/or pattern of bioaccumulation of specific substances under specified exposure conditions. Predictive models can be used when it is not practical to directly measure tissue concentrations (i.e., to determine how tissue concentrations will change over time following a change in exposure conditions). Predictive models can also be used as a screening tool to determine whether it is appropriate to make direct measures of bioaccumulation.

- *Is the test or model being used as a screening tool or as an accurate predictor?*

It is important to clearly identify the data quality objectives before a bioaccumulation study is conducted because the types of questions answered will be dependent on the data collected or generated. Predictive models can cost less and can be run without site-specific data, but the more generic the model, the more the predictions will be subject to high uncertainty. On the other hand, site-specific measurements of contaminant concentrations in sediment and tissue can be highly accurate and precise, but they generally have more limited predictive capacity; i.e., they are not useful for prediction of the impacts of changing environmental conditions or for cross-species extrapolation of results.

- *Is the goal to qualitatively identify which sediment compounds will bioaccumulate or to develop quantitative estimates of tissue concentrations for these compounds?*

If the ultimate goal of an investigation is to determine whether chemicals that bioaccumulate are present in a sediment sample, relatively simple and inexpensive short-term tests can be used. However, the duration of such tests is generally insufficient to allow tissue concentrations in test organisms to reach steady state with sediment or water concentrations. If quantitative estimates of tissue concentrations in exposed organisms are required, longer-term tests, (i.e., a minimum of 28 days) are required. Boese and Lee (1992) list a number of studies using different organisms and exposures, along with estimates of the percentages of steady-state tissue concentrations reached after 10- and 28-day exposures.

- *Will conditions required to reach steady state be met?*

Bioaccumulation is typically measured at steady state (Spacie and Hamelink, 1984). Four factors might prevent steady-state conditions from being achieved in an exposure study: (1) sediment contaminant concentration or bioavailability changes during the course of the study, (2) the exposure period is insufficient to reach steady state, (3) an animal's ability to bioaccumulate the chemical is altered during the course of the exposure period, and (4) additional uptake routes that have not been accounted for exist.

- *Are uptake routes in addition to sediment exposure likely to be important?*

A number of potential exposure routes might be present, depending on the test species and chemical of concern. It is important to identify each of these routes prior to test initiation to prevent substantial underestimations of bioaccumulation.

- *Is a goal to determine or predict the time course of uptake and/or elimination?*

A kinetic model will be more useful than a partitioning model to predict how tissue concentrations will change over time. Kinetic models are very useful for determining how long it will take for tissue concentrations to reach a desired level, for example, how long it will take to decrease to a no-effect level after cleanup has occurred.

- *Is it required that local species be used in the assessment?*

Either standard testing species or indigenous organisms can be used in a bioaccumulation study. The use of indigenous species is desirable since the results of a study using such species would have greater ecological significance as well as relevance to the site under investigation. Standard testing species have the obvious advantage of being easier to maintain in the laboratory because the requirements for their

survival in the laboratory have been well studied. Results of studies using standard test species are also directly applicable to other studies using the same species. On the other hand, a number of problems are associated with the use of standard, nonindigenous species, particularly the potential inability to extrapolate results to indigenous species.

- *Is more than one species to be tested?*

A single species might be adequate for survey work and most research studies. For regulatory testing, however, two species from different taxa are often required. Bivalve species have been identified as good candidates for testing the bioaccumulation potential of PAHs (USEPA and USACE, 1991).

- *Will field or experimentally spiked sediments be used?*

Field sediments are used when a particular site is being evaluated or when dredged material is being tested. Spiked sediments are typically used when a new compound is being evaluated. There are inherent problems in both approaches. With field sediments, collection, handling, and transportation can result in chemical alterations, and the presence of multiple contaminants can confound results. Although spiked test sediments, using artificially prepared sediments or sediments taken from a "clean" reference area, can avoid some of the problems associated with field-collected sediments, they may not be fully representative of natural conditions found at the site of concern.

3.2 Laboratory and Field Methods for Assessing Bioaccumulation

Direct or empirical determination is the simplest approach to measure bioaccumulation in aquatic organisms. Direct determination can be conducted using either laboratory-exposed or field-collected organisms, and generally this approach minimizes or eliminates many of the problems associated with modeling (see below). However, the costs associated with empirical determinations of bioaccumulation can be high, and the results of these determinations typically have only site-specific or study-specific applicability. It must also be recognized that bioaccumulation testing may only deal with the first step in a bioaccumulation chain in which higher levels are typically of interest.

For compounds with a log K_{ow} greater than 6.5, there is a loss of linearity between K_{ow} and bioaccumulation, resulting in considerable uncertainty in modeled predictions of biological uptake. Thus, for these compounds, direct determination is generally the most reliable method for assessing bioaccumulation.

3.2.1 Laboratory Determination of Bioaccumulation

Important issues in laboratory determinations of bioaccumulation of chemicals from sediments include selection of an appropriate test species, sediment sampling and handling methods, conditions during exposure to the sediment, exposure duration, and statistical analyses.

3.2.1.1 Test Species Selection

Ankley et al. (1992) list a number of key factors to consider when selecting appropriate test organisms for standardized sediment bioaccumulation tests, including the following:

- The ability to reflect the ecological position of the species of concern in the field to ensure a realistic exposure scenario.

- A healthy condition and capability to remain healthy during the course of the laboratory exposure.
- Availability throughout the year.
- A relatively well defined exposure history.
- The ability to be reared in a laboratory.
- Adequate biomass for chemical analyses.
- The ability to withstand the long-term exposure periods necessary to reach equilibrium.

For bioaccumulation tests with whole sediment, Boese and Lee (1992) have identified a number of additional factors that are important to consider when selecting an invertebrate test species. Species that provide the most meaningful results for sediment bioaccumulation tests are (1) those in which ingested sediment is the major uptake route for higher-K_{ow} compounds (Landrum, 1989), (2) those which are sufficiently pollution-tolerant and can survive relatively long exposure times, (3) infaunal species tolerant to a wide range of sediment and water quality characteristics, and (4) those which have a low potential for metabolizing contaminants of concern.

Based on the factors listed above, Ankley et al. (1992) believe that the oligochaete *Lumbriculus variegatus* is an appropriate freshwater test species for most situations. They prefer this species because it is relatively easy to culture and handle; it is tolerant to a relatively wide range of sediment characteristics, including particle size and organic carbon content; and it is adaptable to long-term test exposures. Other freshwater oligochaete species that appear to satisfy the criteria are *Stylodrilus heringianus, Limnodrilus hoffmeisteri, Tubifex tubifex,* and *Pristina leidyi*. Using essentially the same criteria, Ingersoll et al. (1995) recommended the use of the amphipod *Hyalella azteca* and the midge *Chironomus tentans* for testing freshwater sediments, although Ingersoll et al. (1998) noted that amphipods may not be suitable test species because of their small tissue mass and sensitivity, while the midge's life cycle may be too short.

Lee et al. (1993) recommended five bioaccumulation test species for marine evaluations based on their feeding type, biomass, salinity tolerance, pollution tolerance, culture potential, bioaccumulation toxicity information, commercial availability, and historical use in a substantial number of experimental bioaccumulation studies and regulatory monitoring programs. The five species are the polychaetes *Nereis diversicolor* and *Neanthes (Nereis) virens* and the bivalves *Macoma nasuta, Macoma balthica*, and *Yoldia limatula*. Lee et al. (1993) recommended that at least one of these species be used in all testing situations, and USEPA and USACE (1991) recommend that bioaccumulation tests include a deposit-feeding bivalve mollusk and a burrowing polychaete. Eight secondary taxa were also recommended by Lee et al. (1993) if certain site-specific requirements are important such as large organism size (arenicolid worms), additional phylogenetic groups (i.e., crustaceans), adaptability to culturing (e.g., *Neanthes arenaceodentata*), and high pollution tolerance (*Capitella* spp.).

Mac and Schmitt (1992) argue that bioaccumulation testing with fish has a number of advantages over testing with invertebrates. Certain fish species resuspend sediments, increasing contaminant bioavailability. Fish can provide adequate tissue mass for chemical analysis, and because they have higher lipid mass than invertebrates, tissue mass requirements for organic contaminant analyses are further reduced. Fish can also receive sediment-associated contaminant exposure from several routes, including direct ingestion and accumulation through gills and skin, and their gills may act as a substrate for the dissociation of hydrophobic chemicals from sediment particles. Finally, fish can be used in

laboratory exposure experiments, as well as in *in situ* studies with caged or free-ranging animals, and they are often of more direct concern regarding biomagnification and human health.

Although fish have the potential to be good laboratory models for bioaccumulation studies, to date only one standardized testing protocol has been published (Mac et al., 1990). This test, using the fathead minnow (*Pimephales promelas*), measures the bioavailability of contaminants in sediments from freshwater systems. No similar test for marine species has achieved wide acceptance, although several species have been used successfully in laboratory studies on bioaccumulation. These species include spot *(Leiostomus xanthurus*), sheepshead minnow (*Cyprinodon variegatus*), and Atlantic silverside (*Menidia menidia*) (Rubinstein et al., 1984a,b).

3.2.1.2 Sediment Sampling and Exposure Conditions

Laboratory testing can eliminate a number of the problems associated with field testing, particularly the spatial and temporal variability associated with natural exposure. However, laboratory testing can create a number of additional problems that are important to consider when designing a study. For example, proper handling of sediment samples and test animals is critical, as is maintaining optimal conditions for animal survival throughout the test period.

Either field-collected or experimentally manipulated (spiked) sediments can be used in laboratory tests. In both cases, however, handling, transport, and manipulation of test sediments can result in the loss of fine sediments, interstitial water, and water-soluble compounds. Cross contamination with metals and organic compounds can occur, as can changes in grain size distribution, chemical concentrations, and sorption equilibria. Details on methods for sediment collection, laboratory setup, and design and conduct of tests can be found in Lee et al. (1993), ASTM (1988, 1992, 1996), and USEPA (In prep.).

3.2.1.3 Exposure Duration

Exposure duration is a critical aspect of experimental design in bioaccumulation testing. If the goal of a test is simply to determine whether contaminants are bioavailable or the potential for bioaccumulation exists, short-term (10- to 28-day) tests can be used, although USEPA and USACE (1998) recommend 28-day tests. However, even data from 28-day exposures should not be assumed to represent steady-state conditions (Mac and Schmitt, 1992). Boese et al. (1997) conducted 28-day bioaccumulation tests with *Macoma nasuta* and both PCB-spiked sediment and DDT-contaminated sediment. Compared to steady-state BAFs measured under similar laboratory conditions, BAFs (C_t/C_s) measured after 28 days ranged from 10 percent to 155 percent of steady-state BAFs, with only 5 of 22 PCB congeners or DDT family compounds within 80 percent of steady state. Four of these five congeners were the most hydrophobic congeners measured (i.e., PCB 170, 180, 194, and 209). This is particularly true of chemicals with log K_{ow} greater than 4, for which bioaccumulation through the food chain becomes more important.

Ankley et al. (1992) compared bioaccumulation of PCBs by laboratory-exposed *L. variegatus* and field-collected oligochaetes. They found good agreement between both groups in terms of uptake of PCB homologues with six or fewer chlorines, but field-collected oligochaetes tended to have higher concentrations of the heptachloro through decachloro congeners. The authors concluded that the field-collected oligochaetes were at equilibrium, suggesting that laboratory exposures of greater than 30 days would be required to achieve steady state for compounds having log K_{ow} values greater than 7.0 to 8.0. Similar results were obtained by Rathbun et al. (1987, cited in Mac and Schmitt, 1992) when comparing PCB uptake by caged and resident fish in a confined disposal facility.

As a general goal, ASTM (1996) recommends that bioaccumulation tests be of sufficient duration to reach 80 percent of steady state. For a variety of reasons, this is not always possible. However, methods have been developed to extrapolate results of 28-day bioaccumulation tests to derive steady-state tissue concentration. For example, the log K_{ow} of a neutral organic compound of concern can be compared with the log K_{ow} in Figure 3-1 to indicate the proportion of steady-state concentration (Css) expected in 28 days. This will allow estimation of the steady-state value from the 28-day laboratory exposure data through the use of a steady-state correction factor. The correction factor is the reciprocal of the decimal fraction indicating the proportion of Css expected in 28 days (USEPA and USACE 1998). For example, if the 28-day uptake is one-third of steady state, the correction factor is 1/0.33 or 3. Correction factors can be obtained from previous laboratory studies (e.g., Boese and Lee, 1992, cited in Lee et al., 1993).

3.2.2 Field Determination of Bioaccumulation

Measuring bioaccumulation at a particular site requires consideration of which test species to use, whether to examine natural populations or use transplanted populations, and how to compare bioaccumulation occurring under conditions at a potentially contaminated site with that occurring at a reference site.

3.2.2.1 Caged vs. Natural Populations

For field determinations of bioaccumulation, test animals can be taken from resident populations at the site of concern or can be transplanted from other locations and maintained at the site of concern in cages or other holding systems. The advantages and disadvantages of each approach are discussed in this subsection.

The use of transplanted test animals confined to cages can simplify field assessment of bioaccumulation in a number of ways. Test animals can often be obtained from commercial vendors in large quantities or from uncontaminated reference locations. Because of this, variability associated with exposure conditions and times for individual test animals can be eliminated. All test organisms can begin the exposure period with the same, or similar, background contaminant concentrations in their tissues, and they will receive essentially the same exposure during the test period. Test animals can be selected based on size and age, as well as sex. Studies can also be designed so that water column and sediment exposures can be differentiated and uptake kinetics can be studied *in situ*. Finally, and perhaps most importantly, exposure concentrations or conditions can be accurately determined because the animals will be confined to a small, well-defined area. The exposure period is controlled by the investigator, permitting time to steady state and other issues to be considered when designing a transplant study with caged animals. Transplant studies provide a balance between experimental control and environmental realism not usually attained with either standard laboratory bioassays or assessment of resident populations.

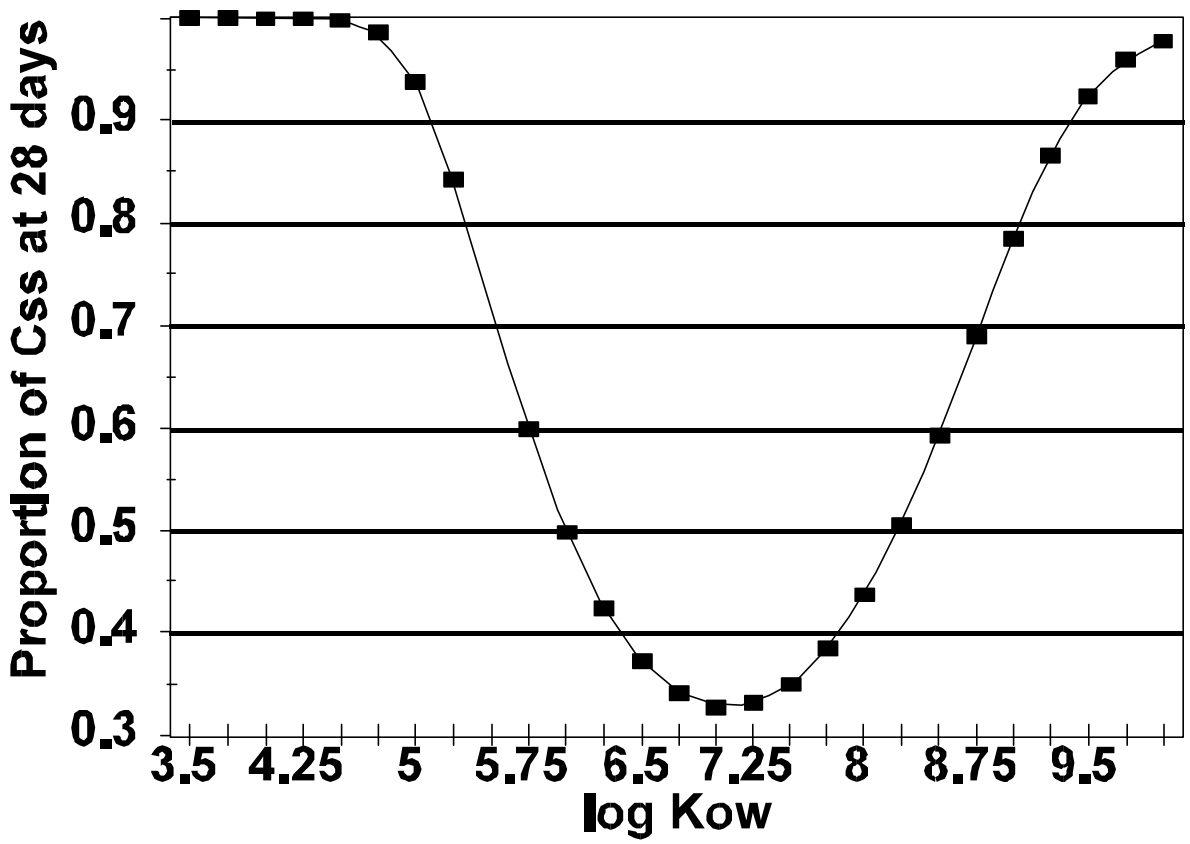

Figure 3-1. Expected Proportion of Steady-state Concentration (Css) of Neutral Organic Compounds Reached in 28-day Laboratory Exposures. The proportion is a function of the log K_{ow} of the compound of interest. Figure adapted from McFarland (1994).)

Transplant studies with caged animals are usually conducted with sedentary species (i.e., bivalves) or mobile species with very limited home ranges, so that accurate exposures can be determined. For the most part, this requirement limits test species to lower-trophic-level (i.e., prey) organisms. If larger, more mobile species such as fish are used, exposure might not be realistic because these animals might have difficulty feeding properly and food chain transfer might not be adequately assessed, thus introducing large uncertainty into estimates of bioaccumulation. Furthermore, stresses associated with handling and crowding could have important effects on the physiology of larger, mobile species.

The greatest advantage to assessing native, free-ranging populations is the potential to evaluate bioaccumulation from all routes of exposure *in situ*. Unfortunately, it is difficult to determine exposure concentrations for all but the most sedentary wild animals. Free-ranging animals might experience a wide range of exposures throughout their home ranges, and for predatory species these home ranges can be quite large. Exposure concentrations can be averaged throughout an animal's home range, although this approach might underestimate the contribution of contaminant hot spots to the animal's total loading, resulting in an overestimation of bioaccumulation factors. Another common approach is to use an upper confidence limit on the range of exposures. This latter approach provides a more conservative estimate of the actual exposure concentration an animal receives, although it might result in an underestimation of

bioaccumulation factors.

In addition to problems associated with estimating exposure concentrations, it is also difficult to estimate exposure duration in native, free-ranging populations. Animals might have been in the sampling area for a considerable length of time or only briefly. Without some means of tracking a free-ranging animal's movements prior to capture, the duration of its exposure becomes an unknown variable. Also, collection of adequate sample sizes can be difficult, particularly when data quality objectives require the use of single species, sexes, and age classes.

3.2.2.2 Species Selection

Selection of test species for field determinations of bioaccumulation is somewhat simplified compared to species selection for laboratory testing. If resident species are used, the choice of species will obviously be limited to whatever species are available at the site of concern. If one is attempting to accurately determine the relationship between tissue concentrations in a species and sediment exposure point concentrations, it is critical that test animals be closely matched according to age, sex, and size. Otherwise, variability in a population of animals will be too great. Animals that are experiencing major physiological changes, e.g., female fish producing vitellogenin prior to spawning, might not be at steady state and, therefore would be inappropriate for bioaccumulation studies. Although it might be desirable to evaluate bioaccumulation in higher-trophic-level organisms, such as predatory birds and fish, these species typically have large home ranges, introducing considerable uncertainty into estimates of exposure concentrations. Furthermore, these species integrate exposures over relatively long time frames, and their tissue concentrations are generally not reflective of short-term changes in exposure concentrations. Thus, if the goal of a study is to evaluate changes in bioaccumulation over short time frames (e.g., during the course of a dredging or remediation project), sedentary or lower-trophic-level species can be used.

A variety of species can be used in transplant studies, with the preferred species for a particular site a function of natural occurrence. In general, it is preferred to use species that already inhabit the area of concern, but surrogate species can be used with permission from state regulatory officials. The use of surrogate or closely related species is often a desired approach because it provides flexibility in the number of species under consideration and available sources. The primary concerns with transplanted species are introduction of unwanted or exotic species and introduction of disease or parasites.

Another approach that might have some utility in field investigations involves the deployment of semipermeable membrane devices (SPMDs), which use thin-walled, lay-flat polyethylene tubing filled with a thin film of a large-molecular-mass, neutral lipid, such as triolein. Huckins et al. (1990, 1993, 1994) have described the theory and use of SPMDs in detail. Although compounds associated with particles, DOC, or colloids cannot pass through the membranes of SPMDs, and therefore are not accumulated by them, SPMDs can be used to estimate the concentration of superhydrophobic compounds in the freely dissolved state. Since bioaccumulation of compounds that are both freely dissolved and associated with dissolved or particulate matter can occur, concentrations measured by SPMDs may likely underestimate uptake by organisms.

3.2.2.3 Reference Sediment Selection

When assessing bioaccumulation at a given site, it is often desirable to use an appropriate reference site to obtain sediment for comparison. A reference sediment is collected near an area of concern and is used to assess sediment conditions exclusive of material(s) of interest (ASTM, 1994). Because reference sediment is not "control" sediment, the reference sediment may have some level of contamination. A control sediment is a well-characterized sediment that provides a measure of test conditions and acceptability by providing information on the health and relative quality of the test organisms. Control sediment is not necessarily collected near the site of concern. Testing a reference sediment, therefore, provides a site-specific basis for evaluating toxicity.

Ideally, reference sites should reflect ambient conditions. Selection of a reference site should avoid areas in the immediate vicinity of, including depositional zones of, spills, outfalls, or other significant sources of contaminants, in addition to areas that are subject to sediment migration of previous dredge material discharges (Federal Register, 1995). In addition to reference site selection based on contaminant concentrations, it is also critical to select reference locations based on their similarity to the test site in terms of habitat type, flow characteristics, physical characteristics of the sediment, and aquatic communities present.

3.2.3 Statistical Design Considerations

Although uncertainty and bias are to a certain degree unavoidable in field- or laboratory-based bioaccumulation studies, it is critical that they be minimized as much as possible. It would be beyond the scope of this document to provide detailed information on how to design statistically sound sampling or testing programs; however, some key statistical design considerations are discussed below. More detailed information on this subject has been summarized elsewhere (EVS, 1995; Baudo, 1990).

According to EVS (1995), bias and uncertainty can be reduced by carefully defining the population(s) of interest. This can be accomplished in a number of ways, including the following:

- Dividing the population to be sampled into logical strata, e.g., substrate type for sediments; sex, age, and weight for biota samples.
- Using adequate replication and sampling over spatial and temporal scales relevant to the sample types; i.e., the size of an area to be characterized is based on the size of the test species' home range.
- Conducting synoptic sampling; i.e., sampling all media types over the same spatial and temporal scales
- Ensuring that samples being collected are representative of the population being evaluated; i.e., if the study is being conducted to evaluate bioaccumulation in an entire population of organisms, sampling should not be biased to include only one segment of that population.

Compositing of samples can also be used to reduce costs and increase statistical power. The main drawback of compositing sediment samples is that, unless taken into full consideration in the sampling design, information regarding the concentrations of contaminants at hot spots can be lost by mixing these sediments with other less contaminated sediment. On the other hand, if one is attempting to establish the relationship between contaminant concentrations in sediment and contaminant concentrations in the tissues of a fish species with a large home range, compositing sediment samples might be appropriate since the composite concentration might provide a more accurate representation of

the animal's exposure. When dealing with sedentary organisms or organisms with limited home ranges, it is more appropriate to relate tissue concentrations to single exposure point concentrations.

By using the widest range of exposure point concentrations, site-specific relationships can be developed between contaminant exposures and biological uptake, particularly if existing information on contaminant gradients at the site is available. A wide range of exposure point concentrations make it possible to perform regression analyses. The collection of organisms from hot spots and the chemical analysis of their tissues might not be representative of their population as a whole and might bias the site-specific relationship. Similarly, it might be inappropriate to chemically analyze tissues of test animals from laboratory bioassays characterized by significant mortality among test species (i.e., sediments from hot spots) because of the strong potential for bias due to unrepresentative accumulation by the test organisms.

While investigators frequently attempt to make predictions at one site based on relationships observed at another, such predictions are valid only within the ranges of the variables measured. Attempts to extrapolate outside these ranges will introduce unquantifiable uncertainty into the predictions.

Detailed information on appropriate methods for stratified sampling can be found in Snedecor and Cochran (1980) and Gilbert (1987). USEPA (1994a) provides a discussion on spatial averaging, and Gilbert (1987) discusses factors to consider when deciding whether to composite samples.

3.3 Approaches for Modeling Bioaccumulation

USEPA currently believes that both mechanistic and empirical modeling approaches are needed to improve bioaccumulation predictions (USEPA, 1996a). Empirically derived bioaccumulation indicators appear to be more accurate, but properly validated models can be used to predict biomagnification and bioaccumulation in some cases (USEPA, 1996a).

3.3.1 Introduction

There are two main approaches to model development: (1) an empirical approach in which laboratory or field data are interpreted to calculate parameters such as bioaccumulation factors (BAFs) and biota-sediment accumulation factors (BSAFs) and (2) a mathematical approach that employs kinetic or equilibrium models, generally referred to as food web models, in which the mechanistic aspects of bioaccumulation are considered. These two approaches are not mutually exclusive since quality field data are normally used to calibrate and confirm mathematical-approach models, and in turn these models provide useful insight regarding field study design and key parameters to measure. The validity and utility of any model is largely dependent on the quality of input parameters used and the level of uncertainty acceptable in model outputs. The type of model chosen is dependent on the goals of the study, exposure scenario, required accuracy and precision, availability of data, and available resources (Boese and Lee, 1992), as discussed in Section 3.1.

The different approaches for modeling are described in this section to provide a basis for understanding the link between concentrations of contaminants in sediment and bioaccumulation by aquatic and terrestrial biota, including humans.

3.3.2 Empirical Models

Direct or empirical determination is the simplest approach to monitoring or assessing bioaccumulation in aquatic organisms and is the recommended approach when the highest degree of accuracy is required (e.g., in support of regulatory actions) (USEPA, 1996a). Methods for bioaccumulation testing are discussed in Section 3.2. For field-measured bioaccumulation estimates to be reliable and useful, the field study design and data interpretation components must be carefully considered and standardized. Several studies have shown that field-measured BAFs and BSAFs can vary as a function of location and time and among species, even at similar trophic levels (Connolly and Glaser, 1998; Hydroqual, Inc., 1995). This section discusses the different quantitative measures of bioaccumulation processes currently in use.

3.3.2.1 Bioconcentration Factors

Bioconcentration is the uptake of a contaminant from water by aquatic organisms where water is the only pathway. Bioconcentration factors (BCFs, expressed as L/kg of tissue), as defined in Equation 1, are based on laboratory studies of organisms exposed to water containing a chemical of concern, where C_t is the concentration of a contaminant in the tissue of the organism (mg/kg, preferably dry weight, but moisture content should also be reported) and C_w is the concentration in water (mg/L) defined in terms of whether it is the total, dissolved, or truly dissolved (noncomplexed) concentration. (The last is preferred.)

$$BCF = C_t/C_w \qquad (1)$$

BCFs are most useful for small, low-trophic-level organisms such as phytoplankton, which are in rapid equilibrium with substances in water, for chemicals with log K_{ow}s up to 5 (Spacie et al., 1995). BCFs do not account for biomagnification of chemicals in the food web (USEPA, 1996a). Hence, for larger, higher-trophic-level organisms, BCFs have been shown to underestimate bioaccumulation potential for hydrophobic compounds that have log K_{ow} values greater than or equal to 4.5 and are resistant to metabolism and degradation.

3.3.2.2 Bioaccumulation Factors

In USEPA (1995b), the bioaccumulation factor (BAF) is defined as “the ratio of a substance's concentration in tissue of an aquatic organism to its concentration in the ambient water, in situations where both the organism and its food are exposed and the ratio does not change substantially over time.” BAFs have been used successfully to predict tissue residues of polar organic compounds, neutral organic compounds, and metals.

Data from laboratory or field bioaccumulation studies can be used to generate BAFs, which are the ratios between contaminant concentrations in organisms and environmental media. BAFs can be calculated as the product of BCFs and food chain multipliers (FCMs) or they can be measured empirically. EPA prefers the latter (USEPA, 1995b, 1996a). Empirical BAFs are most useful since they integrate all environmental routes of exposure and take into account bioavailability considerations for the system under study. Field-measured BAFs are especially important for compounds with log K_{ow} values greater than 6 since prediction of bioaccumulation of these compounds is overestimated if based on their hydrophobicity alone (Spacie et al., 1995). Note that for compounds with very low solubility, which makes their analytical detection in water challenging (e.g., certain PCB congeners, dibenzo-*p*-dioxins,

dibenzofurans), BSAF-corrected BAFs are also recommended (USEPA, 1995b). See Cook et al. (1993) and USEPA (1995b) for methods.

Two empirical BAFs have been reported in the literature, where they are defined as the concentration of contaminant in tissue divided by either the concentration of contaminant measured in water (USEPA, 1995b) or the concentration of contaminant measured in sediment (Lee, 1992; Boese and Lee, 1992) (Equations 2 and 3, respectively). Note that BAFs can be converted to less variable BSAFs if lipid and total organic carbon (TOC) contents are known, as discussed in the next section. As with BCFs, the type of water concentration (whether it be freely dissolved [uncomplexed], dissolved [filtered], and total [unfiltered]) in the BAF expression must be clearly defined. Guidance is available to calculate the BAF in terms of freely dissolved concentration in the water when data regarding total concentrations in the water, particulate organic carbon (POC), and dissolved organic carbon (DOC) measurements are available (USEPA, 1995b).

$$BAF_w = C_t/C_w \qquad (2)$$

$$BAF_S = C_t/C_s \qquad (3)$$

Where BAF is the BAF calculated using empirical data (L/kg of tissue, or kg of sediment/kg of tissue); C_t is the concentration of a contaminant in the tissues of an organism (mg/kg, preferably dry weight, but moisture content should also be reported); C_s is the concentration of contaminant in the sediment (mg/kg, preferably dry weight); and C_w is the concentration of the contaminant in the water (mg/L).

Empirical BAFs are general and do not discriminate among exposure routes, rates of uptake or elimination, or species' physiology. However, the BAFs can be calculated correctly only if the field design accounts for the exposure scenarios for the species of interest, including migration, spatial and temporal patterns in contaminant concentrations, and life history considerations of the organism (age, gender) (USEPA, 1994a; EVS, 1995). These factors are typically derived on a site- or study-specific basis, and they can vary considerably with water body, sediment type, and species (Rubinstein et al., 1983).

BAFs can also vary with time if the concentrations are changing as a function of time in water or organisms. As environmental conditions change, the predictive capacity of BAF diminishes considerably since the BAF is operationally defined for only those conditions during which the measurements were made. For situations with varying contaminant exposure, kinetic models, described in Section 3.3.3.1, are more appropriate.

3.3.2.3 Biota-Sediment Accumulation Factors

In USEPA (1995b), BSAFs are defined as

> the ratio of a substance's lipid-normalized concentration in tissue of an aquatic organism to its organic carbon-normalized concentration in surface sediment, in situations where the ratio does not change substantially over time, both the organism and its food are exposed, and the surface sediment is representative of average surface sediment in the vicinity of the organism.

Site-specific BSAFs (kg of organic carbon/kg of lipid) are calculated for nonpolar organic compounds using the formula

$$BSAF = (C_t/f_1) / (C_s/f_{oc}) \qquad (4)$$

where C_t is the contaminant concentration in the organism (both wet and dry weight are commonly used, so moisture content should be provided whichever is used, as well as a clear delineation of which is selected), f_1 is the lipid fraction in tissue, C_s is the contaminant concentration in sediment (generally dry weight), and f_{oc} is the organic carbon fraction in sediment. This lipid-normalized relationship was developed for neutral (nonionic) organic compounds and is not appropriate for inorganic substances (e.g., metals), although it has been applied to tributyltin (Eisler, 1989). This relationship is not applicable to methylmercury because methylmercury binds tightly to tissue macromolecules (Spacie et al., 1995; Bridges et al. 1996).

One of the basic premises of equilibrium-based modeling as related to sediments is the equilibrium partitioning theory (Di Toro et al., 1991). This theory is being used to propose sediment quality guidelines for two nonionic organic compounds (e.g., USEPA, 1994b), as well as for PAH mixtures and metals mixtures. The essence of the theory is that concentrations of hydrophobic chemicals in sediments are more predictive of biological effects when they are normalized to sedimentary organic carbon. Through this normalization, the concentration of these compounds in the pore water can be predicted based on Equation 5. Evidence to date indicates that chemicals that are freely dissolved in the pore water are more bioavailable than chemicals sorbed to sediments. Thus the pore water concentration, as measured or as predicted through equilibrium partitioning, is a better predictor of bioaccumulation than concentrations of chemicals on a dry weight basis in the sediment (Di Toro et al., 1991).

$$C_w = C_s/f_{oc}K_{oc} \qquad (5)$$

where C_w is the freely dissolved concentration of nonionic chemical compound in pore water, C_s is the concentration of the chemical in the sediment, f_{oc} is the fraction of sedimentary organic carbon, and K_{oc} is the organic carbon-water partition coefficient (which can be related to K_{ow}).

As with BAFs, BSAFs are typically derived on a site- and species-specific basis, using empirical data (USEPA, 1992a). Therefore, they incorporate the effects of metabolism, biomagnification, growth, and bioavailability. BSAFs can also be used to estimate BAF_{fd}, as described in Cook et al. (1993) and USEPA (1995b), where BAF_{fd} is defined as follows, where C_{fd} is the freely dissolved concentration of a contaminant in water:

$$BAF_{fd} = C_t/C_{fd} \qquad (6)$$

Accurate information on organism lipid content and sediment TOC content is required to calculate a BSAF. Lipid content can vary considerably within a single species, based on life stage, sex, and season, so caution is necessary when attempting to use site- or species-specific BSAFs as predictors of tissue burdens in different systems. As with BAFs, proper calculation requires a reasoned approach regarding species exposure, including movement and life history as well as spatial and temporal trends.

BSAFs are most directly applied to infaunal organisms with known home range. For example, Lake et al. (1990) found that analysis of PCBs in mollusks and polychaetes at field sites representing a range of TOC and contaminant concentrations showed that BSAF calculations (i.e., lipid- and TOC-normalized concentrations) significantly reduced the variability in the raw tissue-sediment data relative to non-

normalized data. Work by Hydroqual, Inc. (1995), however, has shown that lipid normalization does not always decrease the variability in BAFs (or BSAFs) and that the decision to lipid normalize and the method by which lipid normalization is achieved depend on species-specific factors as well as lipid contents.

Since ecosystems are rarely in equilibrium, BSAFs include an inherent measure of disequilibrium of the system, which can be quantified as described in USEPA (1995b). Disequilibrium is caused by kinetic limitations for chemical transfer from sediment to water, sediment to biota, or water to the food chain, as well as biological processes such as growth or biotransformation (USEPA, 1995b). Theoretically, at equilibrium BSAFs range from 1 to 4 since the ratio of K_l to (K_l/K_{soc}) is thought to range from 1 to 4, where K_l is defined as the lipid-water equilibrium partition coefficient and K_{soc} is defined as the sediment organic carbon-water equilibrium partition coefficient (USEPA, 1995b). However, since most systems are not at equilibrium, a wider range of BSAFs is reported. This wider range of BSAFs measured in the field does not invalidate the concept. On the contrary, it underlines the need for a field-measured BSAF that is able to incorporate disequilibrium processes (as well as exposure conditions). Several compilations of BSAFs are available, including Lee (1992), Boese and Lee (1992), and Parkerton et al. (1993), as well as a USACE Contaminants Database accessible via the Internet (McFarland and Fergusen, 1994a).

The use of site-specific BSAFs using techniques described in USEPA (1994a) is preferred. However, if literature values are used, available options include selecting a given percentile of the BSAF distribution (as in the TBP method, which uses the 94th percentile) (McFarland and Ferguson, 1994a) or using a regression equation as in the proposed Washington State guidance for sediment quality criteria for human health (PTI, 1995).

BSAFs are most useful for systems that are in steady state, which is technically defined as concentrations in sediment, water, and organisms that do not change as a function of time even though they may not reflect a thermodynamic equilibrium distribution between sediment, water, and organisms. In a practical sense, systems are often considered steady state if the concentrations do not change within the period of study. Therefore, the use of BSAFs to predict tissue concentrations might not be reliable in situations in which the chemical of interest is rapidly degraded or inputs of the chemical to the system vary. In these instances, kinetic models might be more appropriate (see Section 3.3.3.1).

Hydroqual, Inc. (1995) has developed a database of field-measured bioaccumulation factors for a variety of superhydrophobic compounds. Part of this effort involved development of a procedure whereby BAFs or BSAFs could be predicted for previously unstudied chemicals, species, or water bodies. Hydroqual concluded that within a homogeneous group of compounds (e.g., PCB congeners) BAFs and BSAFs can be predicted only within a factor of 10. The uncertainty arises from site- and species-specific differences in food web structure, partitioning at the base of the food web, and the physiology of the organisms, as well as measurement error (Hydroqual Inc., 1995). Predicting BAFs and BSAFs for chemicals outside the “homogeneous group” results in even greater uncertainty. However, results of chemical class-specific analyses in Tracey and Hansen (1996) revealed a similarity of BSAF values among species and habitat types.

The biota-suspended solids accumulation factor (BSSAF) has also been proposed for some studies. It is identical to the BSAF approach, with the exception that contaminant uptake by fish is from suspended solids, rather than in-place sediments (USEPA, 1994a). Its use has been limited.

3.3.2.4 Food Chain Multiplier

As discussed in Section 3.3.2.2, a BAF can be estimated from a BCF if the BCF is multiplied by a factor to account for food web transfer. This factor is referred to as a food chain multiplier (FCM) (USEPA, 1993a, 1995b).

$$BAF = (BCF)(FCM) \qquad (7)$$

The FCM is defined as the ratio of a BAF to an appropriate BCF (USEPA, 1995b). It has been calculated in a variety of different ways, two of which are discussed briefly below. In both approaches, FCMs are calculated assuming metabolism is negligible.

USEPA (1993a) calculates FCMs using a model of the stepwise increase in the concentration of an organic chemical from phytoplankton (trophic level 1) through the top predatory fish level of a food chain (trophic level 4) (Thomann, 1989). Thomann's model was used to generate BCFs and BAFs for trophic level 2 species (e.g., zooplankton) and BAFs for trophic level 3 and 4 species (small fish and top predator fish, respectively) over a range of chemicals with log K_{ow} values from 3.5 to 6.5. At each log K_{ow} value, FCMs were calculated as follows:

$$FCM2 = BAF2/BCF2 \qquad (8)$$

$$FCM3 = BAF3/BCF2 \qquad (9)$$

$$FCM4 = BAF4/BCF2 \qquad (10)$$

where FCM2, FCM3, and FCM4 are the food chain multipliers for trophic level 2, 3, and 4 species, respectively; BCF2 is the BCF for trophic level 2 organisms; and BAF2, BAF3, and BAF4 are the BAFs for trophic level 2, 3, and 4 species, respectively. Field-measured BAFs from the Great Lakes for trophic level 4 were found to be within an order of magnitude of those predicted using this approach (Thomann, 1989; USEPA, 1993a). At log K_{ow} values of 6.5 and greater, the relationship was less certain.

The FCM is defined below as given in USEPA (1995b), where BAF_{fd} is predicted using the Gobas (1993) bioaccumulation model. In the Gobas (1993) model disequilibrium, as discussed relative to BSAFs in the last section, is included in BAF predictions to some extent by inputting the measured concentrations of the chemical in the sediment and in the water column into the model (USEPA, 1995b). This disequilibrium is then propagated through the food web model.

$$FCM = BAF_{fd}/K_{ow} \qquad (11)$$

The trophic level of an organism is needed when applying FCMs to determine BAFs. Trophic levels have traditionally been described in discrete terms as primary producers, primary consumers, secondary consumers, and top predators. Using this approach, trophic levels are symbolized by whole numbers. However, organisms have clearly defined or uniform food sources only in very rare circumstances. Typically, any organism higher in the food chain than primary consumers is likely at an intermediate trophic level, feeding on multiple trophic levels. As a result, attempting to model trophic transfer using linear food chain models introduces considerable variability into predictions of top predator tissue burdens.

Some methodologies have been developed to address trophic level issues. For example, Broman et al. (1992) have described a method to quantitatively estimate *in situ* biomagnification of organic contaminants that uses ratios of stable isotopes of nitrogen to classify trophic levels of organisms. Carbon and nitrogen isotopes are useful in characterizing an organism's trophic level because animals' metabolic processes tend to enrich the heavy isotopes of these elements, ^{13}C and ^{15}N (Peterson and Fry, 1987). Using this approach, significant enrichment of ^{15}N in tissue relative to ^{15}N in unmetabolized reference samples (i.e., in air) is indicative of increasing trophic levels.

Broman et al. (1992) have used the stable isotope approach to classify trophic levels in a littoral and a pelagic food web in the Baltic, as part of an attempt to study trophic transfer of dioxins and furans in that ecosystem. Based on their results, the authors have concluded that the isotopic method is a powerful tool for quantitatively estimating trophic biomagnification of a contaminant from field data at steady state. However, to evaluate non-steady-state conditions and the relative contributions of various exposure pathways, a more mechanistic approach, such as that described by Thomann (1989), is required. Stable isotope ratios can then be used in conjunction with a more mechanistic approach to provide more refined information on trophic pathways and consumption patterns.

3.3.2.5 Theoretical Bioaccumulation Potential

The theoretical bioaccumulation potential (TBP) has been used to evaluate the environmental impacts of bioaccumulative nonpolar organic compounds associated with dredged material (USEPA and USACE, 1991; 1998). It is defined in USEPA and USACE (1998) as "an approximation of the equilibrium concentration in tissues if the dredged material in question were the only source of contaminant to the organism." It is a "coarse screen" and is applicable only to nonpolar organic compounds at present.

The TBP can be calculated relative to the BSAF as follows (USEPA and USACE, 1998):

$$TBP = BSAF\ (C_s/f_{oc})\ fl \quad (12)$$

where TBP is expressed on a whole-body wet-weight basis in the same concentration units as C_s, C_s is the concentration of the chemical in the dredged material or reference sediment, BSAF is set equal to 4, f_{oc} is the total organic carbon content of the dredged material expressed as a decimal fraction, and fl is the organism lipid content expressed as a decimal fraction of whole-body wet weight. Since the use of the default value of 4 can lead to inaccurate estimates (McFarland and Ferguson, 1994a, 1994b), the USACE Contaminants Database, as documented in Lutz and McFarland (1995), has been compiled to provide empirically measured BSAFs for use in TBP calculations.

Although the TBP approach has been commonly applied as a screening tool in the evaluation of dredged material, it has not been extensively used in risk assessments or in the development of sediment quality thresholds. Using the TBP method with the default BSAF value of 4 (as well as the recommended use of geometric means for all parameters) tended to overestimate the body burden of PCBs in carp and walleye at the Manistique, Michigan, site by factors of 2.9 and 18, respectively (Pelka, 1998). This result is consistent with the intended use of 4 as a default value, which is to be protective but not necessarily predictive, since a BSAF of 4 is at the 94th percentile of the USACE Contaminants Database and is a factor of 12 greater than the median BSAF in the database (McFarland, 1998).

3.3.3 Mechanistic Models

In general, mechanistic models or food web models can be grouped into two categories—equilibrium-based and kinetic approaches (Lee, 1992). Equilibrium and kinetic models have been successfully used to describe bioaccumulation of nonpolar organic compounds (i.e., dioxins, furans, and PCBs). The models have not been so successfully applied in describing bioaccumulation of metals or organic compounds subject to metabolism or degradation, such as PAHs.

3.3.3.1 Modeling Within Aquatic Food Chains

Equilibrium Models. Equilibrium models can incorporate uptake of contaminants from the water column, from contaminated sediments, and via the food chain (Lee, 1992). These models are usually referred to as "dynamic" because absolute thermodynamic equilibrium between contaminant concentrations in biota, water, and sediments is rarely reached in a natural setting. Thus, the equilibrium-based models assume steady-state conditions between organisms and the environment. Even steady-state conditions, where the concentrations do not change significantly as a function of time, are not always satisfied in natural settings, which might indicate the need for kinetic modeling as discussed in the Kinetic Models section below.

Equilibrium models also assume a closed system, reversibility of reactions, and sufficient time for the substance to distribute throughout the system. This time requirement to reach full distribution is highly variable. For example, the time required for equilibration of a substance between small organic particles and water might be seconds, days, or years depending on the particle size and the K_{ow} of the substance (Spacie et al., 1995). Equilibrium through a food chain is likely to have an even higher time requirement to reach steady state, depending on the complexity of the food chain and myriad other factors (Gobas and Z'Graggen, 1994).

In general, equilibrium models are applicable to compounds with log K_{ow} values of 3.5 to 6.0 that do not degrade or transform at rapid rates. Field validation of these models has been limited primarily to large lake systems. EPA is interested in including terms for degradation and transformation in the models, expanding the validation efforts, and characterizing the uncertainty in food web models (USEPA, 1996a).

More complex models have been developed to predict the steady-state concentration of contaminants throughout the food web. The Thomann et al. (1992) model (referred to as the Thomann model) and the Gobas (1993) model have been used in recent regulatory efforts, so they are discussed in more detail below as examples of complex equilibrium-based food web models that incorporate interactions with sediment.

The Thomann model, an extension of a previous model (Thomann [1989] model), is a five-compartment steady state model that includes four exposure routes in the description of accumulation by benthic species. This model predicts the accumulation of hydrophobic chemicals in pelagic aquatic systems. In the Thomann model, ingestion of particulate contaminants is considered associated with (1) sediment organic carbon, (2) overlying phytoplankton and ventilation of free dissolved contaminant, (3) interstitial water, and (4) overlying water. The five compartments in the model are phytoplankton/detritus, zooplankton, forage fish, piscivorus fish, and benthic invertebrates. In Thomann et al. (1992), the benthic model is applied to an amphipod-sculpin food web in Lake Ontario to assess the relative importance of different exposure routes for each species.

The equations in the Thomann model include rates for uptake, growth, and excretion, but since it is a steady-state model, the change in concentration in tissues as a function of time is assumed to be zero. Two broad classes of parameters are identified: (1) chemical-specific parameters, including uptake and excretion rates and assimilation efficiency, and (2) organism physiology-specific parameters, including growth and respiration rates and feeding preferences.

Thomann et al. (1992) conclude that with respect to the Lake Ontario food web, (1) the amphipod accumulates most of its body burden from the sediment and not from phytoplankton that make up approximately 80 percent of its diet, and (2) the sculpin accumulates virtually all of its body burden through uptake via the food chain for chemicals with log K_{ow} greater than 5.0.

The Gobas (1993) model is also a steady-state model that has been applied to a Lake Ontario food web. This model provides estimates of site-specific concentrations of hydrophobic organic chemicals in aquatic macrophytes, phytoplankton, zooplankton, benthic invertebrates, and fish from few measured input data including chemical concentrations in water and sediments. Similar to the Thomann model, this model combines the toxicokinetics of individual organisms (e.g., uptake, elimination) as well as the trophodynamics of food webs.

No significant differences were observed between predicted or observed concentrations in the four fish species and two benthic invertebrates modeled. However, the observed concentrations in phytoplankton and zooplankton were higher than predicted, possibly due to small sample sizes and sampling difficulties (Gobas, 1993). One of the key conclusions was that Lake Ontario fish are more sensitive to changes in sediment concentration than to changes in water concentrations. This result and the conclusions of Thomann et al. (1992) emphasize the importance of sediment interactions when modeling aquatic food chains in equilibrium-based steady-state models.

Kinetic Models. In contrast to equilibrium-based models, kinetic models describe bioaccumulation as the net effect of rate processes (uptake and loss of contaminant). Kinetic models have primarily focused on bioconcentration of contaminants from the water column, although more recently they have been applied to uptake from sediment as well (e.g, Boese et al. 1997). Incorporation of bioaccumulation through the food chain in kinetic models requires substantial amounts of data on uptake and loss kinetics in fish. Kinetic models have the benefit that they can be used to predict tissue concentrations with time under non-steady-state conditions (Lee, 1992). General assumptions of kinetic models include constant uptake rate(s), instantaneous mixing within the compartment(s), and a negative exponential depuration process for all compartments (Spacie et al., 1995). Under field conditions, violations of these assumptions are likely to occur. For example, the uptake rate of a substance can vary due to changes in behavior, food type, food availability, and other environmental factors (Spacie et al., 1995).

In general, two types of kinetic models are used—simple first-order kinetic models and bioenergetics-based bioaccumulation models. In the first-order model, all uptake routes are aggregated into a single uptake and a single elimination rate constant as shown below (Spacie and Hamelink, 1982; Davies and Dobbs, 1984). In this model, the organism is considered to be a single, homogeneous, membrane-bound compartment placed in water containing an infinite supply of the substance at a given concentration (Spacie et al., 1995). Slow desorption kinetics from sediment can violate this assumption in natural systems.

$$dCt/dt = k1 * C_w - k2 * Ct \qquad (13)$$

where dCt/dt is the rate of change of the chemical concentration in tissue over time (mg/kg/t), C_w is the chemical concentration in the water (µg/L), k1 is the uptake rate constant (L water/kg tissue * t), k2 is the elimination rate constant (1/t), and t is time. This equation can be modified for bioaccumulation from sediments by substituting k_s (sediment uptake rate coefficient) for k1, and C_s for C_w. Good estimates of k_s and k2 are required for this model to successfully predict the time to steady state and the steady state body burden (Boese and Lee, 1992). In this model, the concentration in the tissue increases over time at a rate that is highest initially. This rate then gradually decreases over time until some asymptotic concentration is reached (Spacie et al., 1995; Boese et al., 1997). These models are most useful to model nonequilibrium processes in open systems that approach or achieve a steady state.

Complex kinetic models, sometimes called bioenergetics-based toxicokinetic bioaccumulation models, allow uptake to occur from multiple routes including overlying water, interstitial water, and ingested sediment, as well as elimination via multiple routes. Uptake from each route is assumed to be independent and additive. The uptake from each route is based on the flux from that medium (e.g., ingestion rate of sediment), its contaminant concentration, and the appropriate assimilation efficiency associated with that route (gill or gut). Loss from the organism is due to elimination or metabolism. Metabolism can be difficult to predict since it is poorly correlated with chemical hydrophobicity and appears to vary widely across species (Hydroqual, 1995).

These complex kinetic models consider the chemical and biological processes that occur in the water column and biota, and each step is modeled using first-order kinetics, second-order kinetics, or steady-state kinetics. These models can be used for non-steady-state exposures, in separating the importance of different uptake routes, and with organisms undergoing substantial growth (Boese and Lee, 1992). However, extensive data are required to correctly apply the models, which makes them in general better suited as a research tool.

As an example of this approach, Landrum and Robbins (1990) and Landrum et al. (1992) extensively modeled the kinetics of PAH uptake and elimination by the freshwater amphipod *Diporea* sp. to show the relative importance of sediment, pore water, and overlying water sources. They found that ingestion or desorption kinetics from sediments appear to control the bioavailability of PAHs to the organisms, and that the rate of desorption was a function of the season as well as the aging of the sediments. Landrum and Robbins (1990) concluded that instantaneous equilibrium (as is assumed in the equilibrium models) is not appropriate for estimating the amount of contaminant that is bioavailable or for predicting the amount of bioaccumulation directly from sediment.

As with equilibrium-based models, application of kinetic models to extremely hydrophobic chemicals (log K_{ow} greater than 6) can be problematic due to the very long uptake times required to reach steady state with the water (one or more years) (Spacie et al., 1995). Since many organisms do not live this long, steady state is never reached. In addition, uptake of these chemicals might be difficult to predict due to the breakdown in relationships between various rates of uptake and depuration for these chemicals (Hawker and Connell, 1988; Gobas et al., 1989, cited in Spacie et al., 1995) or due to growth dilution effects (Niimi and Cho, 1981; Sijm et al., 1992, cited in Spacie et al., 1995).

3.3.3.2 Modeling from Aquatic to Terrestrial Systems

Aquatic organisms provide a direct link for exposure of contaminants to terrestrial species that have an aquatic diet, such as kingfishers, herons, and muskrats, or have a large aquatic component in their diet, such as bald eagles and mink. As discussed in the previous sections, numerous models exist to predict the transfer of contaminants from sediment and surface water through the aquatic food chain. In the

terrestrial environment, a food web modeling approach is used to predict the transfer of contaminants from the aquatic to the terrestrial food chain. Food web modeling is used to calculate a total daily dose of a chemical consumed by a terrestrial species. Therefore, all food web models include terrestrial species, with the exposure parameters and assumptions compiled or derived, and bioavailability of the contaminants determined.

Terrestrial species that represent important components of the ecosystem and are exposed to chemical contamination are used in the food web model. The receptors can be important or rare species or surrogate species that represent major functional groups at the area of concern. The following factors are considered when selecting ecological receptor species:

- Importance to the ecological community, including such factors as high abundance and biomass and importance as a prey species.
- Sensitivity to the contaminants.
- Potential for exposure.
- Relevance to human beneficial uses (i.e., bird watching, hunting, fishing).
- Availability of information.

Once terrestrial species have been selected, exposure parameters and assumptions related to the chemicals of concern are compiled for model development. Exposure parameters include total daily ingestion of food and water, important aquatic dietary components (e.g, fish, crayfish, benthic macroinvertebrates), and feeding territory. Depending on the terrestrial species, exposure parameters can be compiled from the primary literature and USEPA (1993b, 1993c). However, in many cases ingestion rates are not available and are thus calculated using allometric equations (Opresko et al., 1993).

Chemicals are rarely 100 percent bioavailable from the diet, especially when consumed from the field diet. In many cases the chemicals administered to the test species are in a more bioavailable form because they are generally dissolved in a carrier (e.g., acetone, corn oil) before being mixed with the test diet. Therefore, it is important to determine what the relative bioavailability of the chemical is in the diet and to select an appropriate bioavailability factor for each receptor group (e.g., bird or mammal) and contaminant. Information on the bioavailability of chemicals from the diets of birds and mammals is available from a number of sources such as texts by Hrudley et al. (1996) and Ammerman et al. (1995).

The structure of a generic food web model is

$$IR_T = SUM\ [(C * I * BF)_i * EF]/BW \qquad (14)$$

where IR_T is the total rate of contaminant ingestion (mg/kg bw-day wet weight); C_i is the concentration of the chemical in medium i (mg/kg wet weight); I_i is the rate of ingestion of medium i (mg/day wet weight); BF_i is the relative bioavailability factor of the chemical from media i (unitless); EF is the proportion of study area relative to entire home range of terrestrial species (exposure fraction, unitless); and BW is the body weight of terrestrial species (kg).

3.3.3.3 Modeling from Aquatic and Terrestrial Systems to Humans

Exposure to humans is modeled using an approach similar to that described above for terrestrial species. The link between sediment contamination and human health is primarily assessed through the direct

consumption of sediment-dwelling invertebrates (such as clams and crabs) or less frequently through indirect consumption of fish that are contaminated through trophic transfer from a sediment source (Boese and Lee, 1992). In theory, humans could also be exposed through consumption of terrestrial species that ingest aquatic species, although this pathway is limited since the eating preferences of humans do not typically include any terrestrial wildlife species with large aquatic diets.

Important considerations in modeling exposure to humans include the correct determination of fish or shellfish consumption rates (which differ widely across the United States) and a proper definition of the portion of the fish or shellfish actually consumed by humans in terms of both its chemical concentration and its lipid content. As discussed in the previous sections, numerous models exist to link the concentration in fish or shellfish to the concentration in sediment through the aquatic food chain.

As an example, exposure to humans through fish consumption was recently modeled as part of the Great Lakes Water Quality Initiative (GLWQI) (USEPA, 1993a; 1995a). Humans typically eat fish fillets, which usually have lower lipid content than the whole fish generally consumed by wildlife. Therefore, lower standard lipid values have been developed for humans (5 percent) than for wildlife (7.9 percent) (USEPA, 1993a). As part of the GLWQI process, percent lipid data for edible tissues (mostly skin-on fillets) were gathered from the fish contaminant monitoring programs in Michigan, Wisconsin, Ohio, Indiana, New York, and Minnesota. In the proposed guidance, when BAFs for human health are derived from BCFs through the application of an FCM, the appropriate FCM based on the chemical's log K_{ow} is selected from the trophic level 4 (top carnivore) fish species (USEPA, 1993a). These BAFs are based on the Gobas (1993) model, as described in Section 3.3.2.4. Additional guidance is available in USEPA (1997) regarding human health risk assessment and seafood consumption limits.

3.4 Use of Critical Body Residue Approach

The previous sections have focused on the ability to predict or measure the body burden of a chemical in biota. Although the bioaccumulation of a contaminant is an important consideration, bioaccumulation in and of itself is not a hazard. The critical question becomes at what point does bioaccumulation result in body burdens that result in adverse effects on individual organisms (prey and predator species) and ultimately on populations or whole ecosystems, although extensions beyond the individual organism are not well developed at this time.

One increasingly popular approach that links body burdens in an individual organism to toxicological effects in that organism is the critical body residue (CBR) approach. This approach effectively shifts the focus from measuring concentrations in water or sediment to predict toxicity to measuring concentrations in tissues. The CBR approach has several advantages over more commonplace approaches that measure concentrations in water and sediment, as discussed in McCarty and MacKay (1993):

- Bioavailability and exposure from all routes is integrated by the organism.
- Assumptions regarding steady state, equilibrium or uptake kinetics are not required.
- Effects of metabolism are explicitly considered (though the approach is not readily amenable to compounds that are rapidly metabolized).
- Mixture toxicity can be assessed.

The basis of CBR is the assumption that whole body residues are a useful surrogate measurement of the amount of chemical at the site(s) of toxic action and that toxic responses can be predicted from these whole body concentrations. Several researchers, including Ferguson (1939), McGowan (1952), and

McCarty et al. (1992), have shown that for neutral organic compounds, the body residue level which causes narcotic toxicity is relatively constant across a wide variety of organisms.

There are several approaches for estimating screening level CBRs. Many CBRs can be estimated from acute and chronic studies from any exposure-based endpoint, whether it be an LC10 or an LC50, by establishing relationships between exposure-based and residue-based dose estimates (McCarty and MacKay, 1993). In particular, a great deal of work has been done with the fathead minnow (McCarty et al., 1985, 1992; McCarty, 1986, 1990; Mayer et al., 1986, 1992), and this work has been used to form the basis for guidance on the interpretation of bioaccumulation test results (Dillon et al., 1992). Ambient water quality criteria and BCFs have been proposed to calculate body burdens associated with toxicity (Cook et al., 1992; Shephard 1997). Bridges et al. (1996) note that potential body residues could be calculated using chronic water concentrations associated with effects (e.g., Final Chronic Values for Water Quality Criteria or Great Lakes water quality criteria from the GLWQI) and BCFs, for comparison to bioaccumulation test data. Additionally, the range of sediment quality assessment values, including proposed EPA equilibrium partitioning sediment guidelines, has been suggested as a possible basis to extrapolate body residues from BAFs determined in the bioaccumulation test (Bridges et al., 1996), although this approach has yet to be tested. Additivity of toxicants would have to be addressed.

Although the CBR approach has great potential, it is not readily amenable to many carcinogens or mutagens with short half-lives, to chemicals that are rapidly metabolized, or to chemicals such as metals that when present in aqueous media elicit organism responses such as excess mucus excretion that results in suffocation of the organism (McCarty and MacKay, 1993). Therefore, knowledge of the mode of toxic action is an important prerequisite to applying the CBR approach.

Several groups have developed databases to assess the link between body burden and toxic effects. Among these is the group at EPA-Duluth (Jarvinen and Ankley, 1999). The U.S. Army Corps of Engineers and EPA have jointly developed the Environmental Residue-Effects Database (ERED), a compilation of literature data where tissue contaminant concentrations and biological effects were simultaneously measured in the same organism (USACE, 1997).

3.5 Application of Approaches in Deriving Tissue Residue-linked Sediment Chemical Levels

Considerable interest exists today in using the approaches or tools discussed above to identify sediment concentrations associated with threshold tissue concentrations that are protective of aquatic organisms or their predators, including humans. Acute sediment toxicity is relatively easy to evaluate through the use of standardized laboratory bioassays, but the effects of chronic, low-level exposure can be much more difficult to assess or predict, particularly when these effects are the result of exposure to bioaccumulative compounds. As noted in the next chapter, a key aspect of developing tissue residue-linked sediment chemical levels for evaluating the potential for chronic toxicity is to understand the relationships between concentrations of chemicals in sediment and corresponding concentrations of these chemicals in the tissues of exposed organisms along with their associated effects.

The development of tissue residue-linked sediment chemical levels may focus on the protection of benthic species or higher-trophic-level organisms. Several approaches to develop tissue residue-linked sediment chemical levels for species other than benthic organisms were discussed in Cook et al. (1992). These approaches were based on linking sediment concentrations to tissue residues associated with toxicity to those organisms analyzed or projected toxicity to organisms up the food chain.

The comparison of exposure and effects forms the basis of ecological and human health risk assessment, for which guidance is available (USEPA, 1989, 1992b, 1996b). The development of tissue residue-linked sediment chemical levels for the protection of human health requires consideration of several important parameters. These include identification of the target human population to be protected, the species and portions of the fish that are consumed, and the rate of fish consumption. Guidance on the collection and evaluation of this information is provided in USEPA (1997). Many risk assessments have been conducted to establish tissue residue-linked sediment chemical levels on a site-specific basis. A few key examples include concentrations of tetrachlorodibenzo-*p*-dioxin (TCDD) in sediment associated with impacts on fish and birds as well as ongoing development of sediment quality criteria for human health by Washington State.

For TCDD, an interim risk assessment was conducted to relate concentrations of TCDD in water, sediment, and fish tissue to a low or high likelihood of population failure in aquatic life and wildlife (USEPA, 1993d). The report discussed the TCDD exposure, bioaccumulation, and toxic effects data used to estimate sediment concentrations that may be of concern. BSAFs were among the techniques used. Although the approach was qualified as a simple demonstration and not a definitive characterization of TCDD risk, sediment concentrations were presented, on a pg/g dry weight basis, that are believed to be associated with low and high risk to sensitive fish, mammalian wildlife, and avian wildlife species.

In another example, the state of Washington is developing human health criteria for bioaccumulative compounds in Puget Sound sediments. The criteria will be incorporated into the state's existing Sediment Management Standards (Chapter 173-204 WAC). Human health sediment quality criteria (HHSQC) are based on standard risk assessment methodologies in conjunction with empirically derived biota-sediment accumulation factors (BSAFs). The formulas used to calculate the criteria are outlined in more detail below.

For carcinogenic compounds:

$$\text{HHSQC} = \frac{R * BW * AT * UCF}{CPF * ED * IR * BSAF * FL}$$

For noncarcinogens:

$$\text{HHSQC} = \frac{RfD * BW * UCF}{IR * BSAF * FL}$$

where

R = risk level (unitless);
BW = average human adult body weight (kg);
AT = time period over which exposure is averaged (years);
UCF = unit conversion factor (mg/g);
CPF = chemical-specific cancer potency factor as defined by EPA (IRIS);
RfD = reference dose (mg chemical/kg body weight/day);
ED = exposure duration (years);
IR = fish ingestion rate (grams/day);
BSAF = chemical-specific biota-sediment accumulation factor (mg chemical/kg lipid per mg chemical/kg organic carbon); and
FL = percent fish lipid (g lipid/g tissue).

Washington State Department of Ecology's (Ecology) proposed construct for human health criteria relies on a tiered approach, with "Tier I" representing an initial evaluation to determine if sediment chemical concentrations pose a significant human health risk. Additional, site-specific analysis would then be available ("Tier II") to verify the results of the Tier I analysis.

Ecology has focused its efforts on developing HHSQC for a short list of nonpolar organic compounds. The focus is on chemicals for which confidence in the toxicity and bioaccumulation is the highest:

DDD	benzo(a)pyrene
DDE	benzo(a)anthracene
DDT	benzo(b)fluoranthene
hexachlorobenzene	benzo(k)fluoranthene
hexachlorobutadiene	chrysene
PCBs	dibenzo(a,h)anthracene
dioxins/furans	indeno(1,2,3-cd)pyrene

A database of more than 1,200 chemical-specific BSAF values for finfish and shellfish was compiled by Washington State Department of Health (WDOH) and used to develop recommended BSAFs for HHSQC (WDOH, 1995). Analyses of these data showed highly significant regressions of BSAF on K_{ow} for PCBs and dioxins in finfish and for PAH and PCBs in shellfish (PTI, 1995).

For several chemicals (PAHs, PCBs, and dioxins/furans), sediment criteria are calculated based on a summing of chemicals within each group. For PAHs and dioxins/furans, Ecology is proposing the use of toxicity equivalence factors (TEFs), which allows scientific evidence about the cancer potency of individual compounds to be taken into account in the calculation of human health risk. For PCBs, a criterion is proposed for total PCBs (although congener-specific analysis might be considered for Tier II analysis). Ecology is currently investigating the cost and liability implications of the HHSQC through "case studies" (Weiss, 1998).

Other approaches that have been used include whole river sediment strategies, in which concentrations of contaminants in fish, for example, are predicted as a function of different sediment remediation scenarios. One example of this approach was applied in the Fox River (Paulson, 1998). In this example, PCB transport models were used to predict PCB residues in fish in different stretches of the Fox River over an extended period of time. Again, BSAFs were used in the modeling effort, so although the results were presented as PCB concentrations in fish (as fish consumption advisories), PCB concentrations in sediments associated with these tissue residues could also be calculated for different portions of the river.

In summary, developing tissue residue-linked sediment chemical levels involves an explicit assessment of both exposure and effects. The link between these two considerations is most clear for species in direct contact with sediment, such as benthic invertebrates, but even that link can be obscured through multi contaminant exposures. For higher trophic level organisms (e.g., fish, humans and avian species), models have been developed to establish the link.

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4. IMPORTANT BIOACCUMULATIVE CHEMICALS

4.1 Overview

Knowledge of the relationship between quantities of chemicals in sediments and the toxicological effects of those chemicals in the tissues of organisms has gradually evolved, and the relationship is better understood for some chemicals than for others. As noted by Adams et al. (1992), current efforts to develop a tissue residue approach for evaluating sediment quality require linking toxic effects to organism residues, as well as linking the chemical residues in those organisms to concentrations of chemicals in the sediment. This approach could lead to the identification of sediment concentrations associated with threshold tissue concentrations that are protective of aquatic organisms or their predators, including humans. However, as was discussed in Chapter 2, a number of physical, chemical, and biological variables can affect bioaccumulation and must be considered when establishing such "tissue residue-linked sediment chemical levels".

To assist in understanding both the importance of bioaccumulation in the interpretation of sediment quality and the status of our knowledge of the bioaccumulation of potentially toxic chemical contaminants, information associating the presence and quantity of potentially bioaccumulative chemicals in sediment with uptake in the tissues of aquatic and terrestrial organisms and with the effects of those chemicals on the organisms was collected and reviewed. An attempt was made to include only data from literature published since 1986. In limited cases, however, it was necessary to use literature from 1985 or earlier. Initially, only studies reporting exposure (including sediment or water/pore water concentrations), tissue concentrations, *and* toxic effects were examined; later, data from studies linking exposure concentrations with tissue concentrations *or* tissue concentrations with toxic effects were also included.

Toxicity and chemistry data for the chemicals reviewed in this document were obtained from various sources, including local and EPA libraries. Keyword searches were conducted in WAT-TOX (University of Waterloo) and other on-line databases such as DIALOG and the Chemical Information System (CIS), retrieving chemical-specific citations from the Environmental Protection Agency's AQUIRE (AQUatic Toxicity Information REtrieval), ECOTOX, and ASTER databases (Office of Research and Development, Duluth, Minnesota); "Screening Benchmarks for Ecological Risk Assessment" database, Oak Ridge National Laboratory, Oak Ridge, Tennessee; Chemical Evaluation Search and Retrieval System (CESAR, developed by the National Institutes of Health and EPA); ENVIROFATE (Environmental Fate) database (sponsored by EPA's Office of Toxic Substances); IRIS (Integrated Risk Information System), HSDB (Hazardous Substances Data Bank), and HEAST (Health Effects Assessment Summary Tables, National Institutes of Health and EPA); ERED (Environmental Residue-Effects Database, U.S. Army Corps of Engineers and EPA); and other sources. Information was compiled from peer-reviewed publications and reports, as well as federal and state agencies' documents and reports (gray literature), including the Agency for Toxic Substances and Disease Registry (ATSDR) toxicity profiles, a series of synoptic reviews of chemical hazards to biota (Fish and Wildlife Service), *Environmental Science and Technology*, *Environmental Chemistry and Toxicology*, *Water Research*, and *Aquatic Toxicology.* Searches were conducted at different times and by different people, depending on the chemical; thus, although every effort was made to be as comprehensive as possible, variations in search strategies and data interpretation might have affected the completeness of the data presented. Tables summarizing these findings are presented in a separate appendix to this document (EPA-823-R-00-002). Additional information on tissue residue and effects is also available in *Linkage of Effects to Tissue Residues: Development of a Comprehensive Database for Aquatic Organisms Exposed to Inorganic and Organic Chemicals* (Jarvinen and Ankley, 1999).

4.2 Rationale for Choice of Chemicals

A list of bioaccumulative compounds of potential concern was identified based on input from the Bioaccumulation Analysis Workgroup and a review of various agency documents. These chemicals are known to be found in the sediment and in animal tissues at levels associated with toxic effects and are referenced in one (usually more) of the literature items in Table 4-1. Seventeen pesticides on the list (Table 4-2) were included based on having a half-life of greater than 30 days, BCF greater than 1000, LC50 (acute fish) less than 1 ppm, and log K_{ow} greater than 4.2. Chemicals with a log K_{ow} greater than 3.5 are considered to be bioaccumulative, that is, they are likely to partition into organic materials, including the lipids of organisms and predicted and measured BAFs are correlated within the range of log K_{ow} 3.5 to 6.5 (Thomann, 1989). Of the chemicals in Table 4-2, information is reviewed for 11 metals, 1 chlorinated phenol, 10 polycyclic aromatic hydrocarbons (PAHs), 13 pesticides, selected dioxins and furans, selected Aroclors and congeners of the polychlorinated biphenyls (PCBs) group, and total PCBs (Appendix). These chemicals are marked with an asterisk in Table 4-2.

Criteria for selecting the chemicals to be researched included the following: (1) information was readily available; (2) the chemical was of immediate concern and known to bioaccumulate; (3) the chemical was representative of a group or class of compounds; and (4) the chemical was considered to be important in one or more EPA programs.

Table 4-1. Sources of Information for Selection of Important Bioaccumulative Compounds

1997 Listing of Fish and Wildlife Consumption Advisories (USEPA, 1997a).
Regional Ambient Fish Tissue Monitoring Program (RAFT) contaminants of concern (provided by EPA Region 7).
Great Lakes Water Quality Initiative Bioaccumulative Chemicals of Concern (BCCs).
Persistent Organic Pollutants (POPs) listed in “Substantiation report of the Task Force on POP,” 4th meeting, Den Haag (the Netherlands), February 21-25, 1994.
Table 9-5 in USEPA and USACE (1998) *Inland Testing Manual* (which is the same as Table 10 in USEPA and USACE (1995), *QA Guidance for Sampling and Analysis of Sediments, Water, and Tissues for Dredged Material Evaluations.* Chemical evaluations. EPA 823-B-95-001): Octanol/water partition coefficients (K_{ow}) for organic priority pollutants and 301(h) pesticides.
Recommended target analytes in USEPA (1995a), *Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories.* Volume 1. *Fish sampling and analysis.* Second edition, EPA 823-R-95-007.
List of target analytes in USEPA (1992), *National Study of Chemical Residues in Fish.* Volume 1. EPA 823-R-92-008a.
USEPA National Sediment Quality Survey (USEPA, 1997b).

Table 4-2. Important Bioaccumulative Compounds

Class	Compound	CASRN
Metals and Metallic Compounds	arsenic*[1]	7440-38-2
	cadmium*	7440-43-9
	chromium VI*	7440-47-3
	copper*	7440-50-8
	lead*	7439-92-1
	methylmercury*	22967-92-6
	nickel*	7440-02-0
	selenium*	7782-49-2
	silver*	7440-22-4
	tributyltin (oxide)*	56-35-9
	zinc*	7440-66-6
Substituted Phenols	pentachlorophenol*	87-86-5
	pentachloroanisole	1825-21-4
Low-Molecular-Weight Aromatics	acenapthylene	208-96-8
	acenaphthene*	83-32-9
	anthracene	120-12-7
	fluorene	86-73-7
	phenanthrene*	85-01-8
High-Molecular-Weight PAHs	benzo(a)anthracene*	56-55-3
	benzo(a)pyrene*	50-32-8
	benzo(b)fluoranthene*	205-99-2
	benzo(k)fluoranthene*	207-08-9
	benzo(g,h,i)perylene*	191-24-2
	chrysene*	218-01-9
	dibenzo(a,h)anthracene	53-70-3
	fluoranthene*	206-44-0
	indeno(1,2,3-c,d)pyrene	193-39-5
	pyrene*	129-00-0
Chlorinated Aromatic Hydrocarbons	1,2-dichlorobenzene	95-50-1
	1,3-dichlorobenzene	541-73-1
	1,4-dichlorobenzene	106-46-7
	hexachlorobenzene (HCB)	118-74-1
	hexachloroethane	67-72-1
	hexachlorobutadiene	87-68-3
	hexachlorocyclopentadiene	77-47-4
	octachlorostyrene	29082-74-4
	pentachlorobenzene	608-93-5
	1,2,4,5-tetrachlorobenzene	95-94-3
	1,2,3,4-tetrachlorobenzene	634-66-2
	tetrachloroethane	25322-20-7
	1,2,4-trichlorobenzene (TCB)	120-82-1
Halogenated Ethers	4-chlorophenyl phenyl ether	7005-72-3
	4-bromophenyl phenyl ether	101-55-3

Table 4-2. Continued

Class	Compound	CASRN
Pesticides	aldrin*[2]	309-00-2
	chlordane*[2]	57-74-9
	chlorpyrifos*[2]	2921-88-2
	*p,p*N-DDD*	72-54-8
	*p,p*N-DDE*	72-55-9
	*p,p*N-DDT*[2]	50-29-3
	diazinon*	333-41-5
	dicofol*[2]	115-32-2
	dieldrin*[2]	60-57-1
	disulfoton*	298-04-4
	alpha-endosulfan[2]	959-98-8
	beta-endosulfan	33213-65-9
	endrin[2]	72-20-8
	ethion[2]	563-12-2
	ethalfluralin[2]	55283-68-6
	heptachlor*[2]	76-44-8
	heptachlor epoxide	1024-57-3
	alpha-hexachlorocyclohexane (" -BHC)	319-84-6
	beta-hexachlorocyclohexane ($-BHC)	319-85-7
	delta-hexachlorocyclohexane (*-BHC)	319-86-8
	gamma-hexachlorocyclohexane ((-BHC, lindane)	58-89-9
	methoxychlor[2]	72-43-5
	mirex[2]	2385-85-5
	nitrofen (no longer in use)	1836-75-5
	oxyfluorfen*[2]	42874-03-3
	pentachloronitrobenzene (PCNB)	82-68-8
	permethrin[2]	52645-53-1
	S-fenvalerate[2]	66230-04-4
	terbufos*	13071-79-9
	toxaphene*	8001-35-2
	trifluralin[2]	1582-09-8
Dioxins/Furans	2,3,7,8-tetrachlorodibenzo-*p*-dioxin*	1746-01-6
	2,3,7,8-tetrachlorodibenzofuran*	51207-31-9
	1,2,3,7,8-pentachlorodibenzo-*p*-dioxin*	40321-76-4
	2,3,4,7,8-pentachlordibenzofuran*	57117-31-4
	1,2,3,7,8-pentachlorodibenzofuran*	57117-41-6
	1,2,3,4,7,8-hexachlorodibenzo-*p*-dioxin*	39227-28-6
	1,2,3,6,7,8-hexachlorodibenzo-*p*-dioxin*	57653-85-7
	1,2,3,4,7,8-hexachlorodibenzofuran*	70648-26-9
	1,2,3,4,6,7,8-heptachlorodibenzo-*p*-dioxin*	35822-46-9
PCBs (Aroclors)	Aroclor 1016*	12674-11-2
	Aroclor 1221	11104-28-2
	Aroclor 1232	11141-16-5
	Aroclor 1242*	53469-21-9
	Aroclor 1248*	12672-29-6
	Aroclor 1254*	11097-69-1
	Aroclor 1260*	11096-82-5
	Aroclor 1268	11100-14-4

Table 4-2. Continued

Class	Compound		CASRN
PCBs (Congeners)[3]	PCB 8	2,4N-dichlorobiphenyl	34883-43-7
	PCB 18	2,2N,5-trichlorobiphenyl	37680-65-2
	PCB 28	2,4,4N-trichlorobiphenyl*	7012-37-5
	PCB 44	2,2N,3,5N-tetrachlorobiphenyl	41464-39-5
	PCB 52	2,2N,5,5N-tetrachlorobiphenyl	35693-99-3
	PCB 66	2,3N,4,4N-tetrachlorobiphenyl	32598-10-0
	PCB 77	3,3N4,4N-tetrachlorobiphenyl*	32598-13-3
	PCB 81	3,4,4N,5-tetrachlorobiphenyl*	70362-50-4
	PCB 101	2,2N,4,5,5N-pentachlorobiphenyl	37680-73-2
	PCB 105	2,3,3N,4,4N-pentachlorobiphenyl*	32598-14-4
	PCB 118	2,3N,4,4N,5,-pentachlorobiphenyl*	31508-00-6
	PCB 126	3,3N,4,4N,5-pentachlorobiphenyl*	57465-28-8
	PCB 128	2,2N,3,3N,4,4N-hexachlorobiphenyl	38380-07-7
	PCB 138	2,2N,3,4,4N,5N-hexachlorobiphenyl	35065-28-2
	PCB 153	2,2N,4,4N,5,5N-hexachlorobiphenyl	35065-27-1
	PCB 156	2,3,3N,4,4N,5-hexachlorobiphenyl*	38380-08-4
	PCB 169	3,3N4,4N,5,5N-hexachlorobiphenyl*	32774-16-6
	PCB 170	2,2N,3,3N,4,4N,5-heptachlorobiphenyl	35065-30-6
	PCB 180	2,2N,3,4,4N5,5N-heptachlorobiphenyl	35065-29-3
	PCB 187	2,2N,3,4N,5,5N,6-heptachlorobiphenyl	52663-68-0
	PCB 195	2,2N,3,3N,4,4N,5,6-octachlorobiphenyl	52663-78-2
	PCB 206	2,2N,3,3N,4,4N,5,5N,6-nonachlorobiphenyl	40186-72-9
	PCB 209	2,2N,3,3N,4,4N,5,5N,6,6N-decachlorobiphenyl	2051-24-3

[1] Chemicals with asterisk have been researched for bioaccumulation information, which is contained in chemical-specific information tables in the Appendix.
[2] These pesticides were noted by EPA's Office of Pesticide Programs to have BCF >1000, $t_{1/2}$ (hydrolysis) > 30 days, LC50 (acute fish) <1 ppm and log K_{ow} >4.2.
[3] PCB congeners marked with an asterisk were recommended by Philip Cook, USEPA, Office of Research and Development, Duluth, Minnesota, and Richard Pruell, USEPA, Office of Research and Development, Narragansett, Rhode Island. Unmarked congeners are additional congeners measured by NOAA's National Status and Trends Program.

EPA and other agency programs have identified different bioaccumulative chemicals for different purposes. For example, the Great Lakes Water Quality Initiative limited its list of bioaccumulative chemicals of concern (BCCs) to any chemical "which, upon entering the surface waters, by itself or as its toxic transformation product, bioaccumulates in aquatic organisms by a human health bioaccumulation factor greater than 1,000, after considering metabolism and other physicochemical properties that might enhance or inhibit bioaccumulation...." (Final Water Quality Guidance for the Great Lakes System; Final Rule, 40 CFR Parts 9, 122 et al.; *Federal Register*, Thursday, March 23, 1995; pages 15387-15388, and Table 6A, page 15393). The BAF of 1,000 (log BAF = 3.00) was used to determine a limited number of chemicals for which regulatory criteria would be developed; if a lower BAF had been used, more potentially bioaccumulative chemicals would have been included on the list.

Using different criteria, other programs have identified potentially bioaccumulative chemicals not included in the Great Lakes Initiative list. EPA's Office of Pollution Prevention and Toxics developed a preliminary list of potentially persistent, bioaccumulative chemicals based on a half-life greater than 30 days, estimated BCF value exceeding 250 (log BCF = 2.40) (log K_{ow} of 3.5), molecular weight greater than 600, and production volume greater than 10,000 pounds per year (see Chapter 5 for additional information). Canada's government proposed using persistence criteria of half-life in air $ 2 days, water

$ 6 months, soil $ 6 months, or sediment $ 1 year; and bioaccumulative criteria of BCF or BAF $ 5,000 (log BCF or BAF $ 3.70) applied to freshwater fish (data for nonfish species to be used with judgment) or log K_{ow} $ 5 to screen chemicals for possible "virtual elimination" under the Canadian Environmental Policy Act Priority Substances List. (See *Toxic Substances Management Policy: Persistence and Bioaccumulation Criteria*, Final Report of the ad hoc Science Group on Criteria, Government of Canada, Environment Canada, June 1995.)

Thus, the same chemical might be of concern for one program, or within one region or state or site, and not be of concern for another because of specific program mandate(s), levels present in sediment, sources and loadings, or presence of sensitive organisms. Information was compiled on the selected chemicals to help identify gaps in our understanding of the nature of bioaccumulation and toxicity of these chemicals and to justify the inclusion of certain chemicals as bioaccumulative compounds.

4.3 Summary Data in Tables

The tables in the Appendix contain information and data on each persistent and potentially bioaccumulative organic compound or metal, including (1) chemical characteristics, human health concerns, wildlife and aquatic organism partitioning factors, and food chain multipliers, if known, and (2) a profile of the chemical's toxicity, mode of action, and potential for bioaccumulation. Data and references from laboratory and field studies on the toxicity and bioaccumulation of the chemical in invertebrates, fishes, wildlife, and humans are also presented. References are numbered and cited in full at the end of each table.

No attempt was made to screen reports to determine whether the methods used and the results obtained met some preselected level of quality or whether particular quality assurance and quality control procedures had been specified for the collection of particular types of data and for the accurate measurement of chemical concentrations in sediment, pore water, or tissues. Some data are from field studies that involved the assessment of multiple contaminants. No attempt was made to partition the reported effects among the various chemicals evaluated in these field studies. Because a variety of methods and techniques have been used to measure the bioaccumulation of different chemicals in the tissues of organisms, it is the reader's responsibility to evaluate each study carefully to determine its quality, limitations, and uncertainties in relation to the purpose for which that study might be used in interpreting sediment quality.

The chemical characteristics in the summary tables indicate the hydrophobic nature and persistence of each compound. Water solubilities were taken from the Hazardous Substances Data Bank (USEPA, 1995b). Half-lives for the chemicals were derived from data compiled in USEPA (1989; also published as Howard et al., 1991). When possible, the half-life was determined by sediment grab sample or aerobic soil die-away test data, but more often it was based on estimated unacclimated aqueous aerobic biodegradation. The latter information provides only a relative assessment of the persistence of the compound in the environment since degradation rates can be influenced by a variety of physical, chemical, and biological factors (reviewed in Rand, 1995). Shimp et al. (1990) stated that adsorption to sediment particles could rapidly remove most of a chemical from the water column compartment to the sediment compartment, but modeling chemical residence time in sediments is more difficult than modeling biodegradation in other media. Factors such as the partitioning coefficient of the chemical (which determines the proportion of a discharged mass that reaches the sediments), the rate at which sediment particles settle to the bottom, the rate at which particles can be buried, and the extent to which particles are resuspended and transported downstream can have either positive or negative effects on a chemical's biodegradation rate. Shimp et al. also noted that biodegradation of a chemical mass adsorbed

to sediment particles can change when the particle is buried and the environment becomes anoxic or anaerobic. Boethling et al. (1995) examined data for many chemicals and found that in nearly all cases incorporation into sediment enhanced biodegradability.

The log K_{ow} value provided for each nonionic organic chemical is the log of its octanol/water partition coefficient, which represents the relative ability of the chemical to dissolve in water versus octanol, as well as the likelihood of the chemical to complex or sorb to organic carbon. For most chemicals, this value is the one recommended in a draft report (Karickhoff and Long, 1995) that was based on an extensive literature review of measured and modeled data for pure compounds. The draft report is under review by an interagency committee to standardize the log K_{ow} values. Log K_{ow}s for some of the PCB congeners and dioxins and furans were obtained from Mackay et al. (1992).

Log K_{oc} was calculated from the equation:

$$\log K_{oc} = 0.00028 + 0.983(\log K_{ow}).$$

This equation was derived by Di Toro (1985) by regressing K_{oc} to K_{ow}, which demonstrated that the particle organic carbon coefficient was approximately equal to its octanol/water partition coefficient (kg/L). The higher the log K_{ow} and log K_{oc} values, the more likely the chemical will bind to sediment, especially sediment containing high concentrations of organic carbon, and to accumulate in the fatty tissues of aquatic and terrestrial biota. The accumulated chemicals can also biomagnify through the trophic levels of a food chain, as the contaminants in the consumed prey are concentrated in tissues of the predator. Thomann (1989) developed a model for predicting BAFs by using food chain multipliers generated on the basis of the log K_{ow}. For log K_{ow} values in the range of 3.5 to 6.5, model-predicted BAFs compared favorably with measured ones. Uncertainty in predicting bioaccumulation for nonpolar ionic chemicals with log K_{ow}s greater than 6.5 (e.g., superhydrophobic chemicals) increases, probably as the result of rapid metabolism, reduced bioavailability, very low water solubilities, and inhibition of transport due to the large size of the molecule (USEPA, 1991).

Daily intake levels of concern for the protection of human health were compiled from USEPA (1995c, 1995d), including estimated values for carcinogenic endpoints (slope factors) and noncarcinogenic endpoints (reference doses) for the oral ingestion exposure pathway. The carcinogenic classifications of these chemicals are the weight-of-evidence categories established by EPA's Human Health Assessment Group. The categories are defined as follows (USEPA, 1995c):

A	=	Human carcinogen
B1	=	Probable human carcinogen; limited evidence in humans but sufficient evidence of carcinogenicity in animals
B2	=	Probable human carcinogen; inadequate or lack of evidence in humans but sufficient evidence in animals
C	=	Possible human carcinogen; inadequate data for humans and limited evidence of carcinogenicity in animals
D	=	Not classifiable as to human carcinogenicity because of inadequate or no evidence
E	=	Not a human carcinogen, based on no evidence of carcinogenicity in adequate studies

Factors affecting partitioning of the chemical in relation to wildlife and aquatic organisms, food chain multipliers (biomagnification factors), toxic effects and mode of action, and other information were compiled from various sources, as referenced in the bioaccumulation summary tables.

4.4 Insights from the Chemical Summary Tables

Examination of the data compiled for the bioaccumulative chemicals included in the Appendix provides information that might be useful in addressing a number of issues pertaining to bioaccumulation. It is important to note that it is not enough to simply understand bioaccumulation and the factors that control it. Rather, one must understand that certain chemicals, when sufficiently bioavailable, can produce toxic effects in aquatic organisms and terrestrial wildlife and can pose risks to higher trophic levels (including humans) feeding on those organisms. Interpretation of the effect levels also requires consideration of the lipid levels (percent lipid) measured in the organisms. Lipid measurements can vary depending on the methodology used to extract and analyze the lipids. To facilitate comparison among results, it is necessary to convert all the values to the same units. The data in the Appendix tables were usually not converted in order to allow the end user to refer back to the original study, if necessary. However, molar unit conversions were made for three chemicals that have a nonpolar narcotic mode of toxic action—acenapthene, phenanthrene, and pyrene—because narcosis is believed to occur as the result of the presence of toxicant molecules in the organism rather than because of the absolute character of the molecules (reviewed in Rand, 1995). This conversion allows direct comparisons of the tissue residue and effects data for these chemicals.

The compilation of data for the bioaccumulative chemicals revealed that most studies only compared sediment or water concentrations to tissue levels and few studies have measured concentrations in sediment or water *and* concentrations in tissue *and* effects on the organism. No data were found for disulfoton, oxyfluorfen, and Aroclor 1248. Numerous studies did report tissue residue *and* effect concentrations (see last paragraph in this section). BCFs were obtained for various species for 24 chemicals, BAFs for 25 chemicals, BMFs for wildlife for 11 chemicals, and BSAFs for 43 chemicals. (The most BSAF values were reported for PCB congeners, chlordane, *p,p*N-DDE, *p,p*N-DDT, and dieldrin.) For some chemicals appropriate data were sparse (Aroclor 1016, Aroclor 1242, benzo(a)anthracene, benzo(b)fluoranthene, benzo(g,h,i)perylene, benzo(k)fluoranthene, chromium, chrysene, 1,2,3,7,8-pentachlorodibenzofuran, 2,3,4,7,8-pentachlorodibenzofuran, 1,2,3,4,7,8-hexachlorodibenzofuran, 1,2,3,4,7,8-hexachlorodibenzo-*p*-dioxin, 1,2,3,6,7,8-hexachlorodibenzo-*p*-dioxin,1,2,3,4,7,8-hexachlorodibenzofuran, 1,2,3,4,6,7,8-heptachlorodibenzo-*p*-dioxin, and dicofol).

Examination of the chemical summary tables might help to shed light on the following bioaccumulation issues:

- *What species are potentially available for use in testing?*

A variety of organisms have been used to determine bioaccumulation in field and laboratory situations, but not consistently among chemicals. Organisms for which data were found in the tables included those species commonly used in bioaccumulation testing (e.g., *Macoma balthica, Nereis diversicolor, Neanthes arenaceodentata, Chironomus* spp., *Pimephales promelas*) and others that had been obtained in field studies (e.g., stoneflies, caddisflies, freshwater and marine mussels, tubificid worms, carp, sole, sculpin, three-spined stickleback), including some endemic species. Further discussion of issues related to bioaccumulation testing was presented in Chapter 3.

- *How should we account for differential partitioning of bioaccumulative contaminants among tissues?*

Studies compiled in the Appendix reveal that different tissues can contain different amounts of bioaccumulative contaminants. Tissue residue measurement methodology varied greatly with the study,

from using the whole organism to obtain residues (usually invertebrates and small fishes) to examining uptake in specific organs (hepatopancreas, liver, gill, gall bladder, kidney) or in muscle. Potentially the best way to account for this partitioning is normalization to lipid, as was done in some studies. Knowledge of the "site of action" is essential for some chemicals to interpret the data. For example, nerve tissue needs to be examined in the case of some metals.

- *How can bioaccumulation methods be used to assess population-level effects?*

Evaluating endpoints that relate to the survival and success of populations in the wild could reduce uncertainties about the impacts that might occur from exposure to bioaccumulative contaminants in sediment. Percent survival or mortality was the most frequently reported effect. Other endpoints that have been examined include changes in shell thickness and tissue weight or shell deformations (bivalves), scope for growth or growth rates, impacts on reproduction (imposex, breeding impairment, deformed larvae), behavioral changes (feeding rates), immune system dysfunction, metabolic changes or enzyme induction, and histopathological changes.

Extrapolation of subcellular (e.g., enzyme induction, immune system dysfunction) and individual (e.g., growth rates, tissue weight, behavioral changes) effects to population-level effects has proven difficult. Population-level effects might be examined by analyzing the bioaccumulation of chemical contaminants of several species at different trophic levels, as well as by assessing those species' abundances, reproduction success (gonad development, viability of eggs and larvae, recruitment), and age class distributions over a period of time. Histopathological changes can be monitored to provide information on why abundances, reproduction, and age class distribution might change in relation to contaminant uptake. This information can also be incorporated into population modeling efforts to improve our ability to predict population-level effects (discussed in Chapter 3). For example, one study on fathead minnows exposed to sediment containing Aroclor 1254 reported that reproduction was inhibited and frequency and fecundity were 5 to 30 percent of values observed in unexposed fish (Melancon et al., 1989).

- *How can tissue-specific residue levels be coupled with chronic toxicity response data to develop dose-response relationships for bioaccumulative contaminants?*

The most striking lack of information concerned effects. Where sediment *and/or* water *and* tissue concentrations were found, no associated effects data were reported for organisms exposed to acenaphthene, phenanthrene, benzo(a)anthracene (invertebrates), benzo(b)fluoranthene, benzo(g,h,i)perylene, benzo(k)fluoranthene, dicofol, 1,2,3,7,8-pentachlorodibenzo-*p*-dioxin, 1,2,3,6,7,8-hexachlorodibenzo-*p*-dioxin, 2,3,7,8-tetrachlorodibenzofuran (invertebrates and fishes), silver, toxaphene, PCB 28, PCB 105, PCB 126, and PCB 156 (invertebrates and fishes). However, many of the studies found for dioxins and furans and PCB congeners reported tissue residues as the result of food exposure in association with effects on wildlife (e.g., eagle, heron, falcon, duck, and mink). References in which effects data were reported also contained tissue residue data, except for two studies in which cladocerans and amphipods were exposed to arsenic (sediment *and* water *and* effects data only). Sediment concentrations *and* tissue residues *and* effects data were reported for only nine cases. Of the metals examined, tributyltin had the most references to studies that included tissue residue *and* effects data from research on amphipods, polychaetes, mollusks, and fishes. More tissue residue *and* effects data for more species were found for 2,3,7,8-TCDD, *p,p*N-DDE, *p,p*N-DDT, and total PCBs than the other organic chemicals examined. Limited effects data (one to a few references) were found for arsenic, cadmium, chromium, lead, methylmercury, nickel, selenium, zinc, pentachlorophenol, benzo(a)pyrene, benzo(g,h,i,)perylene, fluoranthene, pyrene, *p,p*N-DDD, *p,p*N-DDE, *p,p*N-DDT, diazinon, dieldrin, heptachlor, congeners of dioxins and furans besides 2,3,7,8-TCDD, total PCBs, and Aroclors 1016,

1242, 1254, and 1260. Tissue residue concentrations and effects data for 2,3,7,8-TCDD included several fish species and birds, and for 2,3,7,8-TCDF included rainbow trout, birds, and mink.

The studies reporting sediment *and* tissue residue *and* effects data included Janssens De Bisthoven et al. (1992), which linked deformed antennae in larvae of the midge *Chironomus thummi* to tissue *and* sediment metal concentrations (copper, zinc, lead). Waite et al. (1991) found changes in shell thickness and tissue weight in relation to sediment *and* water *and* tissue concentrations of tributyltin in the oyster *Crassostrea gigas.* Sediment *and* tissue concentrations with survival data were collected for amphipods exposed to fluoranthene and pyrene (Landrum et al., 1994; Harkey et al., 1997). Whole animal wet weights were reduced in freshwater mussels exposed to a higher methylmercury sediment concentration relative to those obtained from mussels exposed to a lower sediment concentration at the least contaminated station (Salazar et al., 1995).

Tissue residues *and* effects were reported for 48 of the chemicals researched; however, for 12 of the chemicals, tissue residue and effects data were observed only for wildlife species. Mortality was a frequently reported effect for many aquatic invertebrates and fishes. Tissue concentrations of 300 µg/kg ww in muscle or liver of rainbow trout were associated with elevated EROD activity after 70 days' exposure to Aroclor 1254 (Toxscan, Inc., 1990), and eggshell thinning was associated with tissue residues of DDD, DDE, and DDT in birds. The development of dose-response relationships for bioaccumulative chemicals will require a better understanding of chronic toxicity responses and, in some cases, the internal dose or tissue residue level at the site(s) of toxic action (see review in Rand, 1995).

It is important to note that, although some chemicals have records for many species—including sediment/water *and* tissue, and tissue *and* effects—in most cases these data are for different species. For example, for benzo(a)pyrene, of 23 invertebrate species, only 1 (*Chironomus riparius*) has both types of data, and these were obtained from different studies. Though it may be possible to infer similar partitioning relationships (e.g., BSAFs) between closely related species in similar environments, data limitations clearly remain an obstacle for developing tissue residue-linked sediment chemical levels. One case in which a single species contains numerous concentration values for both categories of data (but no *single* study that reported sediment/water concentrations *and* tissue concentrations *and* effects data) is tributyltin. The mussel *Mytilus edulis* has been used in several studies to examine sediment contaminant bioaccumulation and effects of this metal. However, when trying to interpret these data, they need to be scrutinized carefully to determine, at a minimum, whether the form of tributyltin reported is consistent across the studies and whether procedures for estimating body burdens are comparable in the field and laboratory studies.

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5. SUMMARY OF AGENCY INFORMATION ON BIOACCUMULATION DATA COLLECTION AND INTERPRETATION

Bioaccumulation of toxic persistent organic contaminants by aquatic organisms is an ongoing concern for several federal agencies, including the U.S. Environmental Protection Agency (EPA), U.S. Army Corps of Engineers (USACE), National Oceanic and Atmospheric Administration (NOAA), U.S. Fish and Wildlife Service (FWS), and U.S. Geological Survey (USGS). These agencies have conducted research related to the issues presented in Chapter 1 and are involved in the development of policies and procedures pertaining to bioaccumulative chemicals. This chapter presents brief synopses of activities related to bioaccumulation data collection and interpretation in various EPA programs and in other federal agencies. Table 5-1 provides a summary of the information contained in this section.

5.1 EPA Headquarters Programs

USEPA has developed a comprehensive, multimedia Contaminated Sediment Management Strategy (59(167) FR 44879-44882, August 30, 1994; USEPA, 1994a, 1998a) describing the implementation of policies to consistently assess, prevent, and remediate contaminated sediment under existing statutory and regulatory authority. Components of the strategy address assessment, prevention, remediation, research, and outreach activities conducted to accomplish four strategic goals:

- Prevent further sediment contamination that may cause unacceptable ecological or human health risks.

- When practical, clean up existing sediment contamination that adversely affects the Nation's water bodies or their uses, or that causes other significant effects on human health or the environment.

- Ensure that sediment dredging and dredged material disposal continue to be managed in an environmentally sound manner.

- Develop and consistently apply methodologies for analyzing contaminated sediments.

EPA is using information and data on bioaccumulation in support of this strategy in its various programs. The ultimate goal of the Agency is to develop an approach to bioaccumulation assessment that will allow consistent, uniform, and robust decision making among the various Agency programs. In April 1994, the Agency participated in a consultation with the Science Advisory Board's (SAB) Bioaccumulation Subcommittee, consisting of representatives from the SAB's Ecological Processes and Effects Committee and Drinking Water Committee, to discuss approaches to estimating the bioaccumulation potential of chemicals and various mass balance/food web models. The SAB provided recommendations for modifying existing approaches for using mass balance/food web models and for prioritizing research needs related to these tools, particularly the collection of field and laboratory data that would help reduce uncertainties in these models (USEPA, 1995a). EPA has also formed a Bioaccumulation Analysis Workgroup, consisting of representatives from program and regional offices, to discuss cross-program issues in the interpretation of bioaccumulation information for the purpose of sediment quality assessment. The following subsections describe activities undertaken within EPA's program offices and regions that are related

Table 5-1. Summary of EPA Uses of Bioaccumulation Data for the Interpretation of Sediment Quality

Agency Mission and Mandates		Components of Contaminated Sediment Management					
Program	**Relevant Statutes[1]**	**Research**	**Assessment**	**Remediation**	**Dredged Material Management**	**Prevention**	**Outreach**
Office of Prevention, Pesticides, and Toxic Substances							
Office of Pesticide Programs	FIFRA		O				
Office of Pollution Prevention and Toxics	TSCA, PPA	O	O			O	O
Office of Air and Radiation	CAA		O				
Office of Research and Development							
National Health and Environmental Effects Research Laboratory		O	O				O
National Exposure Research Laboratory		O	O				
Office of Solid Waste and Emergency Response							
Office of Emergency and Remedial Response (Superfund)	CERCLA, SARA	O	O	O			
Office of Solid Waste	RCRA, HSWA, PPA	O	O	O		O	
Office of Water							
Office of Science and Technology	CWA, WRDA, GLCPA	O	O			O	O
Office of Wetlands, Oceans, and Watersheds	CWA, MPRSA, WRDA	O	O	O	O	O	O

Agency Mission and Mandates		Components of Contaminated Sediment Management					
Program	**Relevant Statutes[1]**	**Research**	**Assessment**	**Remediation**	**Dredged Material Management**	**Prevention**	**Outreach**
Office of Wastewater Management	CWA					O	

Table 5-1. Continued

Agency Mission and Mandates		Components of Contaminated Sediment Management					
Program	**Relevant Statutes[1]**	**Research**	**Assessment**	**Remediation**	**Dredged Material Management**	**Prevention**	**Outreach**
Region 1				O	O		
Region 2				O	O		
Region 3/Chesapeake Bay Program	CWA	O	O	O	O	O	O
Region 4					O		
Region 5		O	O	O	O		O
Great Lakes National Program Office	CWA as amended by GLCPA		O	O	O		
Region 6			O		O		
Region 7			O		O		
Region 8					O		
Region 9			O	O	O		
Region 10		O	O	O	O		
International Efforts			O			O	

[1]Acronyms: CAA = Clean Air Act; CERCLA = Comprehensive Environmental Response Compensation and Liability Act; CWA = Clean Water Act; FIFRA = Federal Insecticide, Fungicide, and Rodenticide Act; GLCPA = Great Lakes Critical Programs Act; HSWA = Hazardous and Solid Waste Amendments; MPRSA = Marine Protection, Research, and Sanctuaries Act; PPA = Pollution Prevention Act; RCRA = Resource Conservation and Recovery Act; TSCA = Toxic Substances Control Act; WRDA = Water Resources Development Act. Statutes are listed only for headquarters and program offices.

to research on bioaccumulative chemicals, assessment of such chemicals in the environment and potential risks from exposure, remediation and dredged material management of bioaccumulative chemicals, prevention of the manufacture or release of persistent organic pollutants, and outreach to inform and educate the public on the topic of bioaccumulative chemicals.

5.1.1 Office of Prevention, Pesticides, and Toxic Substances (OPPTS)

OPPTS develops national strategies for toxic substance control. It is responsible for the promotion of pollution prevention (P2) as the principle of first choice, as well as assessment of risk to human health and the environment from exposure to pesticides (Office of Pesticide Programs) and industrial chemicals (Office of Pollution Prevention and Toxics). Important activities of the office are to establish procedures and criteria for assessing chemical substances and to develop guidelines for chemical testing. OPPTS also develops rules and procedures for industry reporting, and develops and enforces regulations for controlling industrial chemicals and pesticides deemed hazardous to humans or the environment. Interest continues in the development of new sediment test methods for determining the environmental fate, ecotoxicity, and bioaccumulation of pesticides and industrial chemicals in sediment.

An important role of bioaccumulation test data is to support review of new and existing chemicals under the Toxic Substances Control Act (TSCA) and the registration/reregistration of pesticides under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA). In addition, bioaccumulation information may be used to provide guidance on the design of new chemicals to reduce bioavailability and partitioning to sediment.

OPPTS is in the process of harmonizing its own (OPPT and OPP) test guidelines with those of the OECD (Organization for Economic Cooperation and Development) to create a single set of guidelines that minimizes variability and unnecessary testing. The office prepared “public draft” sediment toxicity test guidelines employing chironomids (including tissue residue analysis) and amphipods (freshwater, estuarine, and marine). These have been published in the *OPPTS Test Guidelines, Series 850 Ecological Effects*, Volume I (USEPA, 1996a). These 850 guidelines are in the process of being finalized. OPPTS is also involved in developing new OECD sediment toxicity test guidelines for chironomids. An environmental fate test guideline, “Sediment/Water Microcosm Biodegradation Test” (OPPTS 835.3180), has gone final and is publicly available. Also, the office is near final approval of an OECD guideline (“Aerobic and Anaerobic Transformation in Aquatic Sediment”) that has a similar purpose.

5.1.1.1 Office of Pesticide Programs (OPP)

The mission of OPP is to safeguard public health and the environment from unreasonable pesticide risks, while ensuring that pesticides are regulated fairly and efficiently. In carrying out its responsibilities under FIFRA, OPP must consider both the risks posed by pesticides and the benefits that pesticides offer to society. State and tribal agencies and many other organizations, both public and private, are vital partners in this effort.

Assessment

Bioconcentration studies are currently required by two divisions of OPP under FIFRA for the registration of pesticides. The Environmental Fate and Effects Division requires bioconcentration testing as prescribed in 40 CFR, Part 158 under guidelines 72-6 and 165-4, -5; the Health Effects Division requires a bioconcentration test under test guideline 171-4. The purpose of these studies is to determine

if pesticide residues accumulate in fish used as human food sources, to determine the edible portions of such fish, and to characterize the fate of pesticides within the various tissue compartments of an organism.

The Environmental Fate and Effects Division of OPP requires bioaccumulation tests to support the registration of formulated end-use products intended for outdoor use (except domestic outdoor), or aquatic impact uses resulting in direct discharge into aquatic environments. Bioaccumulation testing is required when the active ingredient or its principal degradation products have a water solubility less than 0.5 mg/L, the octanol/water partition coefficient is greater than 1,000 (log K_{ow} = 3), it is persistent in water (e.g., half-life greater than 30 days), or it accumulates in organs and tissues of mammals or birds.

The studies are flow-through, preferably using radioisotopic analytical techniques, and the exposure system must maintain constant concentrations of chemical in true aqueous solution not to exceed 1/10th the 96-hour LC50 for the test species. The preferred test species is the channel catfish or the bluegill sunfish. The studies require 28 days of exposure with a depuration of 14 days. Extractable residues of 0.05 mg/L or greater must be identified in two samples of edible tissue and two samples of viscera. If pesticides are shown to bioaccumulate in fish tissue, the Environmental Fate and Effects Division may require accumulation testing with nontarget organisms.

The accumulation study required by the Health Effects Division (171-4) is a metabolism study designed to determine the magnitude of residues in fish following exposure to a pesticide. The studies are required when fish may be exposed to the pesticide or its degradation products. The test uses carbon-14 and may be static or flow-through depending on the aquatic system under consideration. Fish residue data are required for bottom feeding species (e.g., catfish), predators (e.g., bass), and shellfish (both mollusks [e.g., clams, oysters] and crustaceans [e.g., shrimp, crabs]). For pesticides used in estuarine areas, data on whole fish protein concentrate and on smoked, canned, or other processed fish products are needed to determine whether a Food Additive Regulation is necessary.

5.1.1.2 Office of Pollution Prevention and Toxics (OPPT)

The mission of OPPT is (1) to protect and improve human health and the environment, to achieve risk reduction, sustainability, and environmental justice, and to enhance the quality of life; (2) to promote safer designs and wiser use of materials, products, processes, practices, technologies, and disposal methods, using pollution prevention as the principle of first choice; and (3) to provide information, education, and technical assistance to empower the public to make informed decisions on the risks associated with toxic substances.

Prevention/Assessment/Outreach

<u>Draft Multimedia Strategy for PBT Pollutants</u>

EPA recently released a draft strategy to further reduce risks to human health and the environment from existing and future exposure to priority persistent, bioaccumulative, and toxic (PBT) pollutants. The strategy, available on the Internet at www.epa.gov/pbt/strategy.htm, reinforces and builds on existing EPA commitments related to priority PBTs, such as the 1997 Canada-U.S. Binational Toxics Strategy, the North American Agreement on Environmental Cooperation, and the recently released Clean Water Action Plan. EPA is forging a new approach to reduce risks from and exposures to priority PBT pollutants through increased coordination among EPA national and regional programs. This approach

also requires the significant involvement of stakeholders, including international, state, local, and tribal organizations, the regulated community, environmental groups, and private citizens.

The four main elements of the PBT Strategy are

- Develop and implement national action plans for priority PBT pollutants
- Screen and select more priority PBT pollutants for action
- Prevent introduction of new PBT pollutants
- Measure progress

To date, EPA actions to reduce emissions of PBT pollutants have been largely separate regulatory activities aimed at different environmental media. Such actions will now be better coordinated to ensure, for example, that regulations removing the PBT from air do not inadvertently result in transferring the pollution to the ecosystem. Developing an Agency-wide strategy enables EPA to harness all of its tools—voluntary, regulatory, international, enforcement, compliance, and research—and direct them at a set of priority pollutants of common concern to all EPA program offices. EPA's first 12 priority PBT pollutants, from the Canada-U.S. Binational Toxics Strategy, are aldrin/dieldrin, benzo(a)pyrene, chlordane, DDT (+ DDD + DDE), hexachlorobenzene, alkyl-lead, mercury and compounds, mirex, octachlorostyrene, PCBs, PCDD (dioxins) and PCDF (furans), and toxaphene.

The strategy outlines a number of actions that EPA will take to reduce exposures to and uses of PBT chemicals. Some of the near-term actions include the following:

- Preventing the introduction of new PBT chemicals in commerce
- Encouraging voluntary reductions of priority PBT chemicals in hazardous waste
- Giving the public information on mercury emissions from utilities
- Increasing the public's right to know about local sources of PBT pollutant emissions
- Evaluating fish in U.S. water bodies for PBT chemical contamination

Research/Prevention/Assessment

Screening the TSCA Inventory

In the early 1990s OPPT screened discrete organic chemicals on the TSCA Chemical Substances Inventory to provide a preliminary list of potentially persistent and bioaccumulative chemicals for further evaluation. This activity identified approximately 80 chemicals with estimated half-lives exceeding 30 days, estimated BCFs greater than 250, molecular weights greater than 600, and production volumes greater than 10,000 pounds per year. Substances having such physical properties often partition significantly to the sediment compartment in the aquatic environment. This list is being used by the Canadian government (joint project of Health Canada/Environment Canada) in activities aimed at prioritizing Canada's Domestic Substances List (DSL), the Canadian equivalent of the TSCA inventory, and identifying PBT chemicals.

Waste Minimization Prioritization Tool (WMPT)

The goal of the Waste Minimization National Plan (WMNP), developed by EPA's Office of Solid Waste (OSW) and mandated by the U.S. Congress, is to reduce the volume of the most persistent, bioaccumulative, and toxic chemicals in the nation's hazardous wastes at least 25 percent by the year 2000 (50 percent by 2005). In pursuing this goal, source reduction is preferred over recycling and a central purpose is to avoid cross-media transfers of constituents. The Waste Minimization Prioritization

Tool (WMPT) was developed jointly by OSW and OPPT to assist in the implementation of the WMNP. The WMPT ranks chemicals based on persistence, bioaccumulation, and toxicity potential ("PBTness"). To accomplish this, it classifies (bins) chemicals as high, medium, or low for persistence and bioaccumulation potential. As noted above, substances having such properties often demonstrate a marked tendency to accumulate in aquatic sediments, in addition to fatty tissue of exposed organisms. The WMPT is flexible, Windows-based software designed for screening only.

Toxics Release Inventory (TRI): PBT Rulemaking

OPPT maintains the Toxics Release Inventory (TRI) under the Emergency Planning and Community Right-to-Know Act (EPCRA). Proposed rulemaking that specifically targets PBTs was published in the *Federal Register* on January 5, 1999 (64 FR 687). As noted above, substances having such properties often demonstrate a marked tendency to accumulate in aquatic sediments, in addition to fatty tissue of exposed organisms. The rulemaking proposes to:

- Set persistence and bioaccumulation criteria for reporting on chemicals under EPCRA section 313.
- Add some chemicals that meet toxicity criteria for listing and are also persistent and bioaccumulative.
- Lower reporting thresholds for PBTs that are already on the TRI.

In so doing the rule provides general technical guidance on the types of persistence and bioaccumulation data needed to determine "PBTness." It also sets half-life criteria of 2 months for water/sediment and soil and 2 days for air, as well as a bioaccumulation factor/bioconcentration factor (BAF/BCF) of 1,000, for purposes of defining which substances are and which are not persistent and bioaccumulative under the rule. Further, although compartment-specific half-life criteria are the primary means of determining persistence, the rule indicates that multimedia fate modeling will be used to confirm findings based on compartment-specific half-lives, and it may override compartment-specific data if key model inputs are judged reliable.

TSCA: Proposed PBT Policy for Premanufacture Review Chemicals

Under TSCA, a Premanufacture Notice (PMN) must be submitted 90 days prior to manufacture of any new industrial chemical. For many years OPPT has used chemical "categories," based on toxicity, to streamline regulatory review, and until now categories (of which there are now more than 45) have been defined by structure/activity relationships (SARs). Taking a new approach, OPPT recently developed and proposed a category for persistent, bioaccumulative, and toxic (PBT) substances for purposes of PMN review. As noted above, substances having such properties often demonstrate a marked tendency to accumulate in aquatic sediments, in addition to fatty tissues of exposed organisms.

Proposed half-life criteria for persistence and bioaccumulation are tiered to reflect level of concern:

Criterion	Possible Actions
Persistence: > 2 mo for water/sed or soil; Bioaccumulation: fish BCF $ 1,000	Exposure/release controls; triggered testing
Persistence: > 6 mo for water/sed or soil; Bioaccumulation: fish BCF $ 5,000	More stringent controls; ban pending testing

To determine potential risk, EPA may use multimedia models to account for all loadings, transformation processes, and intermedia transfer in an integrated fashion. Implementation of the new PBT policy might also lead to testing to fill critical data gaps if EPA is unable to adequately determine the potential for persistence and bioaccumulation using existing information. The proposed testing strategy is tiered and includes sediment/water persistence test guidelines of increasing complexity in tiers 2 and 3. Analogously, laboratory testing for bioaccumulation potential would be conducted if tier 1 testing (Ready Biodegradability testing and measured log K_{ow}) confirms suspicion that a substance is potentially persistent and bioaccumulative. Standard fish bioconcentration testing is conducted in tier 2, and, potentially, bioaccumulation testing using sediment-dwelling aquatic organisms in tier 3.

Development of this TSCA new PBT chemicals policy has occurred in coordination with US national, US/Canada binational, and international efforts to identify and control the environmental release of persistent organic pollutants (POPs). The PBT category description, in the form of an October 5, 1998 proposed *Federal Register* policy statement (63 FR 53417), was provided to the United Nations Environment Program (UNEP) Governing Council's Criteria Expert Group (CEG) for POPs, established at the first session of the Intergovernmental Negotiating Committee (INC). The final policy statement on this PBT category responded to comments received on the proposed policy and was published in the Federal Register on November 4, 1999 (64 FR 60194). This final notice represents the first formal statement of US national policy regarding new chemical POPs. Under our domestic TSCA program, the policy statement provides guidance criteria for persistence, bioaccumulation and toxicity for new chemicals and advises the industry about our regulatory approach for chemicals meeting the criteria. Internationally, the October 5, 1998 proposed policy statement alerted the parties involved in negotiation of the POPs Convention to the need for inclusion of a new chemicals provision in the Convention. The issuance of the final policy statement reaffirms US leadership on this issue and serves as a model for other countries in taking steps to discourage the introduction of POPs as new chemicals and pesticides.

5.1.2 Office of Air and Radiation (OAR)

OAR incorporates information on bioaccumulation of air toxics in certain analyses when such information is available. For example, in the Urban Air Toxics Strategy, finalized in July 1999, OAR ranked air toxics to derive a list of the pollutants posing the greatest risk to public health in urban areas. As one part of this analysis, OAR ranked air toxics by relative potential for oral toxicity and food-chain bioaccumulation. Using estimated national emissions, in tons per year, OAR adjusted the emissions by multiplying a bioconcentration factor (BCF) and dividing it by the oral risk-based dose (RBD) for chronic effects. RBDs for chronic oral exposure were expressed as a milligram of an air toxic per kilogram of body mass per day. Measured and estimated BCFs for air toxics were obtained from EPA's draft Waste Management Prioritization Tool (WMPT). The WMPT is intended to allow EPA to rank relative hazards from the list of hazardous substances regulated under the Resource Conservation and Recovery Act, and it has been judged to be a reasonably comprehensive resource for the purpose of ranking air toxics. The BCF is an estimate of the proportion of a substance that will partition into aquatic

organisms relative to ambient water, at equilibrium. Well-developed BCFs consider both relative solubility in water and lipid and the ability of biological systems to metabolically alter the contaminant.

In addition, in March 1999 OAR released a Report to Congress presenting its methodology for conducting "residual risk" assessments. After setting technology-based standards for major industrial sources of air toxics, OAR must evaluate the remaining health and environmental risks (i.e., the residual risks) and set more stringent regulations, if necessary. For each of these assessments, OAR will identify the exposure pathways of concern, taking into consideration the environmental persistence and bioaccumulation potential of certain air toxics. For a limited subset of air toxics, assessments may include noninhalation exposure (e.g., food chain, other environmental media).

5.1.3. Office of Research and Development (ORD)

ORD provides scientific and technological expertise to remediate environmental and human health problems. Its three headquarters' offices, three national research laboratories, and two national centers work with other EPA program and regional offices, the states, and tribes. ORD conducts basic, peer-reviewed research and implements cost-effective, common-sense technology, including providing extramural research grants and fellowships to develop the sound environmental research necessary to ensure effective policy and regulatory decisions.

5.1.3.1 National Health and Environmental Effects Research Laboratory (NHEERL)

NHEERL in Research Triangle Park, North Carolina, is responsible for investigating the effects of environmental pollutants and other anthropogenic stressors on human health and ecosystems, using toxicological, clinical, epidemiological, ecological, and biogeographic research methods. Long-term research at NHEERL is defining and characterizing toxicological hazards, quantifying dose-response and other important cause-effect relationships, and assessing the integrity and sustainability of ecosystems. The Atlantic Ecology Division (AED) in Narragansett, Rhode Island; Mid-Continent Ecology Division (MCED) in Duluth, Minnesota; Gulf Ecology Division (GED) in Gulf Breeze, Florida; and Western Ecology Division (WED) in Corvallis, Oregon, are charged with developing methods and techniques for examining bioaccumulation of persistent organic chemicals and metals, evaluating bioaccumulation under field and laboratory conditions, developing models and other analytical procedures for assessing risks to biota from bioaccumulative chemicals, and assisting in the preparation of guidance for interpreting bioaccumulation data.

Research/Assessment

NHEERL has conducted numerous studies of bioaccumulation in marine biota. Examples include comparisons of field data to the 28-day bioaccumulation test for uptake of the pesticides DDT and dieldrin in the clam *Macoma nasuta* (WED) and measurement of the accumulation of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD), 2,3,7,8-tetrachlorodibenzofuran (TCDF), and polychlorinated biphenyls (PCBs) from Passaic River, New Jersey, sediment samples by sandworms, clams, and grass shrimp (AED) (Pruell et al., 1993). Another project involves the bioaccumulation and trophic transfer of polychlorinated dibenzo-*p*-dioxins (PCDDs), polychlorinated dibenzofurans (PCDFs), PCBs, and other compounds in a laboratory food chain consisting of sediments, polychaetes, and lobster (AED). A second area of research involves the use of surrogate materials mixed into sediment to determine the bioavailability of sediment-associated contaminants. The ability of equilibrium-based and kinetic approach bioaccumulation models to predict tissue residues as a

cost-effective alternative to direct measurement of tissue residues has also been evaluated (WED) (Lee, 1992).

MCED is conducting research on assessing bioaccumulative chemicals found in freshwater sediments, as well as improving methods for evaluating the effects of such compounds on aquatic and terrestrial biota and developing tissue residue criteria for a variety of contaminants. Because relationships between complex mixtures of sediment contaminants and bioaccumulation and toxicity in benthic organisms are often uncertain, the Division is conducting controlled toxicity and bioaccumulation tests. Current research includes further investigation of the effects of pore water chemistry on bioavailability and toxicity identification evaluation (TIE) methods development.

The Great Lakes Water Quality Initiative (GLWQI) required bioaccumulation factors (BAFs) for fish that incorporated site-specific bioavailability, biomagnification/food chain effects, and metabolism. Additional complications were the need for BAFs for chemicals such as TCDD, which currently do not have measured concentrations in water, and a concern for variation over time and space, and in the chemical properties of the sediment-water distribution coefficient. Most of the potential variability was eliminated by basing BAFs on trophic level, lipid-normalized concentrations in tissues, and concentrations of freely dissolved chemicals in water. Freely dissolved concentrations in water are based on octanol-water partition coefficient (K_{ow})-correlated partitioning to particulate and colloidal organic carbon in the water. The ability of biota-sediment accumulation factor (BSAF) ratios to measure bioaccumulation and metabolism differences between chemicals was incorporated into a procedure for estimating BAFs for chemicals below analytical detection limits. Food chain models that incorporate benthic food chain linkages to pelagic fish were also evaluated and included in the GLWQI bioaccumulation methods. These methods need to be evaluated further in other aquatic ecosystems.

At MCED, laboratory methods for toxicity and bioaccumulation tests for a variety of freshwater benthic invertebrate species have been developed and validated. These include toxicity test methods for the amphipod *Hyalella azteca* and the midge *Chironomus tentans*, and a 28-day bioaccumulation test method for the oligochaete *Lumbriculus variegatus* (USEPA, 1994g). Field validations of bioaccumulation predictions have been accomplished (e.g. Ankley et al., 1992). Nonchemical factors that increase sediment contaminant toxicity, such as activation of PAHs in organisms by ultraviolet radiation, have been documented and incorporated into toxicity predictions.

MCED is also working on sediment-based ecological risk assessment methods. Fish early life stage toxicity from exposure to complex mixtures of PCDDs, PCDFs, PCBs, and other polyhalogenated aromatic chemicals that share an aryl hydrocarbon receptor-mediated mode of action has been related to sediment contamination in the Great Lakes through use of chemical-specific BSAFs, TCDD toxicity equivalence factors (TEFs), and dose-response relationships based on concentrations of these chemicals in fish eggs. Through retrospective analysis and correlation with epidemiological data, this work provides an initial validation of the integrated application of sediment-based bioaccumulation models for pelagic fish, chemical concentration in tissue-based dosimetry, and an additive toxicity equivalence model. The same methods may be applied to wildlife. Staff scientists from MCED also prepared an “interim report on data and methods for assessment of TCDD risks to aquatic life and associated wildlife” (USEPA, 1993). The report reviewed and evaluated data and models available for analyzing the effects of exposure to and bioaccumulation of TCDD in aquatic life and wildlife, and it addressed areas of uncertainty that limit how well risks can be characterized.

Scientists at MCED are participating in the Lake Michigan Bioaccumulation and Toxic Effects Characterization Study (LMBTECS), the purpose of which is to incorporate all of the data, models, and

methods of the Division's projects into the interpretation of a high-quality bioaccumulation data set obtained in conjunction with the ongoing Lake Michigan Mass Balance Study for the chemicals PCBs, *trans*-nonachlor, and atrazine. Besides the collection of sediment, plankton, benthos, fish, and water samples to evaluate bioaccumulation and toxic effects models, LMBTECS is unique because of the extensive list of analytes (approximately 1,000 chemicals) and the use of high-resolution gas chromatography/high-resolution mass spectrometry with stable isotope standards and maximum instrument sensitivity. This sensitivity, coupled with large-volume water samples (1,000 L) extracted by three different techniques, will maximize detection of hydrophobic organic chemicals in water. Much of the study involves interpretation of chemical concentrations in sediments and suspended solids. Core analyses, BSAFs, tissue dosimetry-effects relationships, and toxicity equivalence models for complex mixtures of chemicals with the same mode of action will provide a basis for retrospective analysis of exposures and associated toxicity risks.

GED’s Estuarine Assessment program is working to develop, improve, and validate realistic diagnostic procedures to determine the ecological condition of Gulf of Mexico estuaries. Research related to bioconcentration/bioaccumulation is part of this program. Some biota are transplanted and exposed in cages for periods of 21 to 42 days, after which tissues are analyzed for metals, pesticides, PAHs, and PCBs. The exposures are conducted at many stations and seasonally to determine the spatial and temporal variability in the bioresidues. In other cases, indigenous species are analyzed from the same locations to determine the relative difference in tissue quality using the two assessment techniques. The types of biota that have been analyzed include freshwater bivalves, oysters, blue crabs, fishes, sea grasses, rooted vascular plants, algal mats, and periphyton. The study areas are those associated with Superfund sites, wastewater, urban and storm water runoff, agricultural runoff, dredging activities, and golf course runoff.

The residue concentrations have been compared to enzyme content, blood proteins, pigment content, biomass, and community structure for several of the biota to determine the impact of the residues. The chemical quality of the surface water and sediment has been determined during the analyses and compared to the tissue burdens to determine the bioconcentration factors from these different media. In addition, the effect of salinity on the magnitude of the residues has also been investigated. Currently, several GED personnel are developing a data set describing the tissue quality in several biota inhabiting reference coastal areas. The data from this study will be used to determine the relative impact of the anthropogenic activities on tissue quality determined in previous studies.

Outreach/Technical Guidance

The Pacific Ecosystems Branch in Newport, Oregon, produced a guide to the methods used for assessing pollutant bioaccumulation from bedded sediment in sediment-dwelling organisms (Boese and Lee, 1992). The guide assists researchers in selecting the best laboratory or field assessment method or bioaccumulation model based on the available data and the goals of the project.

AED developed a Standard Operating Procedure for the extraction of lipids from marine tissues (Pruell, 1995). Contaminant concentrations normalized to lipids measured using this SOP have been shown to correlate well with toxicity and contaminant depuration rate. The technique is also relatively simple and produces precise data.

Outreach/Database Development

MCED has developed databases to assist in the interpretation of bioaccumulation data. ECOTOX version 1.0 is now available for government users (Russom, 1996). Developed at MCED with support by the Department of Defense's Strategic Environmental Research and Development Program, ECOTOX allows users to search across three existing EPA databases—AQUIRE, PHYTOTOX, and TERRETOX—which contain ecotoxicological effects information for aquatic life, plants, and terrestrial wildlife, respectively. Version 1.0 also provides access to the Office of Pesticide Programs' Ecological Effects Database of aquatic and terrestrial effects data reviewed and categorized as acceptable for pesticide registration and reregistration guideline requirements explained under FIFRA Subdivision E, Parts 158.145 and 158.150. ECOTOX user's manuals can be obtained from the Scientific Outreach Program by phone, (218) 720-5602; fax, (218) 720-5539; or e-mail, outreach@du4500.dul.epa.gov. Data are grouped in the AQUIRE database according to chemical, aquatic organism, exposure conditions, and effect endpoint, and include BCF test results. TERRETOX is a terrestrial wildlife toxicity database that provides data linking quantified chemical exposures with observed toxic effects, providing data to quantify the relationships between chemical concentrations in environmental media or wildlife foods and residues in wildlife tissues. This database identifies sources of alternative data (domestic or laboratory animal toxicity and bioaccumulation information) when there is a lack of information on wildlife species.

MCED is currently developing a database of toxic effects to aquatic life associated with concentrations of bioaccumulative chemicals in tissues. To date, approximately 450 references from the scientific literature have provided data on 180 chemicals for freshwater and saltwater invertebrates and vertebrates, including amphibians. The data will allow prediction of toxicity risks or establishment of tissue residue-linked sediment chemical levels for single chemicals when concentrations of sediment contaminants in tissues of susceptible organisms can be determined through use of equilibrium partitioning, BSAFs, site-specific exposure models, or direct measurement. Tissue chemical concentrations associated with no observable effects, mortality, or reduction in growth or reproduction are most commonly noted.

5.1.3.2 National Exposure Research Laboratory (NERL)

NERL is responsible for characterizing the sources of environmental stressors and the compartments of the environment in which they reside or move, studying the pathways through compartments that lead to exposure of receptors to stressors, investigating intra- and intercompartmental stressor transfers and their transformations, and studying and characterizing receptors to predict or measure stressor exposure. The Ecological Exposure Research Division in Cincinnati, Ohio, and the Ecosystems Research Division, in Athens, Georgia, have been involved in research to develop and implement models to provide risk assessors with better and more refined estimates of exposure and dose.

Research/Assessment

Scientists at the Athens laboratory developed a model to analyze the bioaccumulation of PCBs in Lake Ontario alewife (*Alosa pseudoharengus*), coho salmon (*Oncorhynchus kisutch*), rainbow trout (*Oncorhynchus mykiss*), brown trout (*Salmo trutta*), and lake trout (*Salvelinus namaycush*) (USEPA, 1991a). The model addresses fish gill morphometry; feeding and growth rate; fractional aqueous, lipid, and nonlipid organic composition; and chemical properties used to estimate aqueous diffusivity and partitioning to fish lipid and nonlipid organic fractions such as molar volume and – octanol/water partition coefficient (K_{ow}).

5.1.4 Office of Solid Waste and Emergency Response (OSWER)

OSWER is responsible for controlling hazardous wastes and remediating hazardous waste sites under the Resource Conservation and Recovery Act (RCRA) and the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA), including ash, mining wastes, medical wastes, underground petroleum storage tanks, oil spills, municipal solid wastes, industrial solid wastes, and household hazardous wastes.

5.1.4.1 Office of Emergency and Remedial Response (Superfund/Oil Programs) (OERR)

OERR manages the Superfund program, which was created to protect citizens from the dangers posed by abandoned or uncontrolled hazardous waste sites. Congress established Superfund in 1980 by passing CERCLA, which gives the federal government the authority to respond to hazardous substance emergencies and to develop long-term solutions for the nation's most serious hazardous waste problems.

Research/Assessment

Under CERCLA, EPA has established a comprehensive program for identifying, investigating and remediating sites contaminated with hazardous substances. The Hazard Ranking System (HRS) is a scoring system used to assess the relative threat associated with actual or potential releases of hazardous substances at sites. The HRS is the primary way of determining whether a site is to be included on the National Priorities List (NPL), EPA's list of sites that are priorities for long-term evaluation and remedial response. The HRS score is the result of an evaluation of four pathways: (1) ground water migration, (2) surface water migration, (3) soil exposure, and (4) air migration. Bioaccumulation is evaluated for two threats in the surface water migration pathway, the human food chain and the environmental food chain. The bioaccumulation potential of each hazardous substance is determined using a tiered system employing (1) BCF data, (2) the logarithm of the n-octanol-water partition coefficient (log K_{ow}) data, and (3) water solubility data (55 FR 51532).

Risk assessments for Superfund sites involve a longer and more costly process than an HRS evaluation. Under CERCLA, EPA carries out a detailed analysis of risks posed by contaminants at the site to human health and the environment, and the feasibility of various response action alternatives to reduce risk. The *Risk Assessment Guidance for Superfund* (RAGS) (USEPA, 1989, 1991b, 1991c,1997c) provides a framework for the assessment of human health and environmental impacts. Various EPA publications, including guidance in RAGS, Ecological Updates, and fact sheets, are used to develop assessments that are presented as a part of the Remedial Investigation/Feasibility Study (RI/FS) of a CERCLA site. The process is not designed specifically for sediments, but rather for the purpose of assessing all exposure routes from contamination at CERCLA sites. Nine criteria are used in the FS to evaluate options for remedial actions at CERCLA sites: (1) overall protectiveness of human health and the environment; (2) compliance with ARARs, i.e., national and state standards and criteria; (3) long-term effectiveness and permanence; (4) reduction of toxicity, mobility, or volume through treatment; (5) short-term effectiveness; (6) implementability; (7) cost; (8) state acceptance; and (9) community acceptance.

There is a widely recognized need for Agency-wide guidance for determining which chemicals have the potential to bioaccumulate. There is regional variation in the log K_{ow} cutoff for determining chemicals that have the potential to bioaccumulate, with some using log K_{ow} greater than 5 and others using log K_{ow} greater than 4. There is also no consensus on the degree of bioaccumulation of heavy metals and

lipophilic organic halogens. Other issues concerning bioaccumulation data and their interpretation in the Superfund Program are summarized below.

Estimates and Measurements of Bioaccumulation

Regions prefer to use site-specific information on bioaccumulation whenever possible, using models and literature values for preliminary or screening assessments. Site-specific data are usually collected for more complex sites or sites requiring further investigation. Types of empirical data used to evaluate bioaccumulation include

- Fish and mammal tissue sampling and analysis
- Toxicity testing
- Field and *in situ* (caging) bioaccumulation studies
- Surface water and sediment chemical analyses
- Sediment leachability tests

In one region, the U.S. Fish and Wildlife Service is using an "artificial" fish with a lipid-filled membrane bag to conduct bioaccumulation studies. Bioaccumulation models being used by the regions include a K_{ow} model and a fish-gill exchange transfer model (USEPA, 1991a). Literature values used by the regions include chemical-specific bioaccumulation factors (BAFs).

Effects Concentrations for Chemicals

To identify effects concentrations, the regions are using values from peer-reviewed journal articles, AQUIRE, USFWS Hazard Reviews, information from other sites, and the Oak Ridge National Laboratory database. A number of regions mentioned using toxicity tests to measure effects concentrations. Most Regions estimated effects concentrations using NOEL and LOEL values, the hazard quotient method, and food web models. Partitioning coefficients (K_{ow} and K_{oc}) are obtained from USEPA and USFWS documents, the Superfund Chemical Data Matrix, and the ASTER database (part of AQUIRE). Approaches to identify effects concentrations and partitioning coefficients vary among the regions.

Use of Background Data to Assess Bioaccumulation

Most regions use background data as a reference for comparison to site conditions. One region uses background data to evaluate non-site-related stressors such as widespread pesticide use, agricultural input, and other nonpoint sources of pollution. Some regions screen out potential chemicals of concern (COCs) based on their presence in background or reference samples, whereas others take background information into account during the risk management, rather than the risk assessment phase. Background data may be particularly relevant during remediation, when cleanup levels are determined. The regions agree that background data should be collected and presented with site-specific data, but none suggest that empirical data collected to assess the effects of bioaccumulation should be used on both background and site-impacted samples.

Remediation

Cleanup and Endpoints Based on Bioaccumulation

Several regions reported that cleanups at sites within their jurisdiction had been driven by risks associated with bioaccumulation of chemicals. Generally, numerical cleanup for COCs was back-calculated, using either site-specific BAFs or published values for representative species. In two cases, sites were remediated to a predetermined numerical standard, and tissue sampling and analysis of the local habitat followed cleanup. Overall, the examples mentioned by the regions involved cleanups of soil, not sediment. The Regions agreed that endpoints for bioaccumulation effects should protect and preserve local habitats, species, or populations. Target species for protection were generally fish-eating mammals and birds such as minks, shrews, wrens, raptors (hawks, eagles), and raccoons. Reproduction endpoints in these species were a major concern, followed by mortality and growth. One region suggested specific endpoints for DDT (avian reproduction) and mercury (fish-eating mammals and birds).

Assessment

Bioassay procedures can be valuable tools that provide efficiency and realism in assessing contamination. This was demonstrated in Puget Sound, Washington, where caged mussels were used to assess the bioavailability of sediment contaminants at the Harbor Island Superfund site and at a Carr Inlet reference site (Salazar et al., 1995). The caged mussels were held one meter from the bottom for an exposure period of 82 days. Accumulation of contaminants in adult mussel tissues was used to identify the extent and magnitude of contamination in overlying water, while reduced growth in juveniles was used to indicate adverse bioeffects. The analysis revealed a statistically significant inverse relationship between growth rate, toxicity-normalized tissue accumulation, and toxicity-normalized sediment contamination. Based on these results, the researchers recommended an integrated approach for evaluating contaminated sediments that includes conducting *in situ* bioassays, estimating bioaccumulation in addition to bioeffects, and evaluating water overlying sediments.

5.1.4.2 Office of Solid Waste (OSW)

OSW manages a complex regulatory program addressing solid waste disposal and hazardous waste management under RCRA. RCRA regulates the identification, transportation, treatment, storage, and disposal of solid and hazardous wastes. The act regulates such matters as hazardous waste generators and transporters; land disposal restrictions (LDR); federal procurement of products that contain recycled materials; municipal solid waste landfill criteria; solid and hazardous waste recycling; treatment, storage, and disposal facilities; and waste minimization and hazardous waste combustion.

Assessment/Prevention

Currently, OSW is conducting several multimedia risk analyses to support regulatory development efforts under the Resource Conservation and Recovery Act (RCRA). Examples of these include determining chemical-specific waste stream concentrations that represent a threshold below which Subtitle C disposal will not be required and thus the waste stream may exit the hazardous waste system; determining whether specific industrial wastes should be listed as hazardous under Subtitle C; and evaluating the risks of special types of wastes to determine the appropriate level of waste management required. As part of these efforts, OSW is developing a risk assessment modeling tool in coordination

with the Office of Research and Development. The tool will address multimedia exposures through several pathways for multiple receptors, including various types of human and ecological receptors.

Major components of the risk assessment methodology are various food chain modules in both the terrestrial and aquatic environments; within each environment several different types of habitats are being investigated. The key function of the aquatic food chain module is to determine suitable BAFs for chemical constituents and use these values to predict concentrations in the tissues of aquatic biota. Mechanistic models, regression equations, EPA analyses, and empirical data are used in a weight-of-evidence approach to determine the appropriate BAFs and, where possible, distributions of these factors. The approach is organized around five types of constituents—hydrophobic organics, hydrophilic organics, ionizable organics, PAHs, and metals (including mercury). The module involves a weight-of-evidence approach that considers the appropriateness of simulation modeling and regression equations to derive suitable BAFs, as well as measured BAFs from field studies. Although the Gobas model (and other similar models) adapted for use in the tool have been validated (e.g., for Lake Ontario), they have not been validated across all of the aquatic systems included in this analysis, particularly small streams (i.e., stream order 2 and 3). In addition, estimates of fish tissue concentrations are limited by the quality and quantity of data on bioaccumulation.

For farm food chain exposures, waste constituent concentrations are estimated in major plant and animal categories. For most organic constituents, regression equations relate simulated air and soil concentrations to plant tissue concentrations and, in turn, soil and plant concentrations to beef/milk concentrations (as described in the IEM [1997]). Steady-state and equilibrium are assumed between soil and roots, between soil and aerial parts, and between air and aerial parts. No flow between compartments is considered. A simple partition coefficient between the plant and an environmental medium (air or soil) is used to estimate the concentrations in vegetables and forage grasses. Each of the mechanisms considered for plant uptake is represented by a biotransfer factor and includes the following:

- Root uptake and translocation
- Air-to-plant transfer of vapor-phase contaminants
- Deposition of particle-bound contaminants on plant surfaces

Similarly, biotransfer factors are developed for each of the mechanisms considered for uptake into beef and dairy cattle, including

- Uptake from ingestion of contaminated forage, silage, and grain
- Uptake from incidental ingestion of contaminated soil
- Uptake from ingestion of contaminated surface water (e.g., local pond)

For all organic constituents except dioxins (and congeners), selected PAHs, and several hydrophobic constituents that are well studied, biotransfer factors are predicted using the empirical relationships represented by the regression equations. For these organic constituents, values are selected that are specific to both the chemical and, in some cases, the category of plant or cattle (i.e., beef or dairy). For metals, including mercury, plant uptake factors are derived from field data, including but not limited to values reported in EPA's sludge risk assessment work.

Biotransfer factors are used to represent major categories of plants (and animal tissues) without regard to site-based conditions such as soil type, plant species, application matrix, or environmental conditions (e.g., temperature). As a result, predicted concentrations in plants and beef/dairy products may be

associated with substantial uncertainty. For example, the biotransfer factors for metals may vary over several orders of magnitude depending on the study conditions.

In the terrestrial environment, uptake and accumulation in food items (flora and fauna) are used to estimate concentrations in higher trophic levels found in the terrestrial habitat. Four major categories are evaluated: (1) plants, (2) earthworms, (3) soil invertebrates, and (4) vertebrates. Each major category is divided into several subcategories that reflect significant differences in the dietary habits of receptors; for example, forage grasses and forbs would not be treated the same as nuts and berries. Generally speaking, mechanistic models and regression equations are lacking for terrestrial systems, particularly models that address variability in the environmental setting (e.g., differences in soil characteristics).

The same limitations noted above for the aquatic and farm food chain estimates also apply to the terrestrial environment with respect to predicting plant concentrations (e.g., an empirically based approach). In addition, the lack of data on uptake and accumulation of constituents in other terrestrial food items introduces significant uncertainty. In many cases, only a single point estimate may be available to determine the bioaccumulation potential in a given category.

Remediation

Subtitle C of RCRA provides EPA with the authority to assess whether releases from a hazardous waste treatment, storage, or disposal facility have contaminated sediments and to require corrective action, including possible remediation, if contamination is discovered. RCRA corrective action authorities apply to, among other things, all releases of hazardous waste or constituents from any solid waste management unit at a treatment, storage, and disposal facility seeking a RCRA permit, regardless of when the waste was placed in the unit. EPA assesses hazardous waste facilities that have RCRA permits. These assessments are called RCRA Facility Assessments (RFAs). If an RFA suggests that a release has occurred, hazardous waste permit writers can require facility operators or owners to conduct extensive RCRA Facility Investigations (RFIs) to determine the extent of any contamination. In the corrective action program, EPA sets priorities using the National Corrective Action Prioritization System (NCAPS). NCAPS priorities are generally based on information gathered during the RFA. EPA's policy is to focus its corrective action resources first on facilities and areas at facilities that present the greatest relative risk to human health and the environment. Accordingly, NCAPS considers the environmental setting of a facility and potential receptors, actual and potential releases of hazardous wastes or constituents from the facility, and the toxicity of constituents of concern to group facilities into high, medium, and low priority groups. NCAPS rankings are based on risk, but NCAPS does not involve a traditional site-specific risk assessment. NCAPS is a resource management tool that EPA and authorized states use to set relative priorities among corrective action sites to focus limited agency resources (61 FR 19432).

Prevention/Research

In November 1994, EPA released the Waste Minimization National Plan (WMNP). The WMNP focuses on reducing the generation and subsequent release to the environment of the most persistent, bioaccumulative, and toxic chemicals in hazardous wastes. It establishes three goals:

- To reduce, as a nation, the presence of these chemicals in hazardous wastes by 25 percent by the year 2000 and by 50 percent by the year 2005.
- To avoid transferring these chemicals across environmental media.

- To ensure that these chemicals are reduced at their source whenever possible, or, when not possible, that they are recycled in an environmentally sound manner.

The first goal has also been included as a Government Performance and Results Act (GPRA) objective for the RCRA program.

Stakeholders involved in the development of the WMNP emphasized the need to prioritize source reduction and recycling activities based on risk and requested a flexible screening tool that would assist them in identifying priorities. EPA subsequently committed in the WMNP to develop a tool that would prioritize chemicals based on persistence, bioaccumulation potential, toxicity, and quantity. EPA's Office of Solid Waste (OSW) and Office of Pollution Prevention and Toxics (OPPT) developed this tool, the Waste Minimization Prioritization Tool (WMPT), and released a Windows-based beta-test version for public review in June 1997 (EPA530-C-97-003). Based on the public comments received, a number of improvements were made to the WMPT, and a modified spreadsheet version was released in November 1998 in support of OSW's draft RCRA PBT List (described below).

Bioaccumulation scoring in the revised WMPT is based on measured bioaccumulation factors, measured bioconcentration factors, predicted bioaccumulation factors, and predicted bioconcentration factors, in order of preference. Chemicals with bioaccumulation or bioconcentration factors greater than or equal to 1,000 are classified as high concerns for bioaccumulation, chemicals with values below 250 are classified as low concerns, and the rest are classified as medium concerns. Additional information on the revised WMPT and its bioaccumulation scoring approach is provided in the *Waste Minimization Prioritization Tool Spreadsheet Document for the RCRA Waste Minimization PBT Chemical List Docket* located in RCRA docket F-98-MMLP-FFFFF and on EPA's homepage at www.epa.gov/wastemin.

The revised WMPT was used as the starting point for selecting chemicals for the RCRA PBT List, a list of chemicals that will serve as the focus of national hazardous waste minimization program activities. Four equally weighted criteria were used to score and rank candidate chemicals for the draft list, including the higher of human health and ecological concern scores from the revised WMPT, the quantity and prevalence of chemicals in RCRA hazardous waste, the presence of chemicals in the environment, and RCRA programmatic concerns associated with the chemicals. Additional information on the list is provided in RCRA docket F-98-MMLP-FFFFF and on EPA's homepage at www.epa.gov/wastemin.

5.1.5 Office of Water (OW)

OW is responsible for EPA's water quality activities, which represent a coordinated effort to restore the nation's waters. The functions of this program include developing national programs, technical policies, and regulations relating to drinking water, water and sediment (including dredged material) quality, and ground water; establishing environmental and pollution source standards; and providing for the protection of wetlands. In addition, the Office furnishes technical direction, support, and evaluation of regional water activities; enforces standards; and develops programs for technical assistance and technology transfer. The Office oversees the provision of training in the fields of water quality, economic and long-term environmental analysis, and marine and estuarine protection.

5.1.5.1 Office of Science and Technology (OST)

OST is responsible for developing sound, scientifically defensible standards, criteria, and advisories and effluent guidelines, effluent limitations, and standards guidelines under the Clean Water Act and the Safe

Drinking Water Act. OST is also responsible for developing risk assessment methodologies and for providing risk assessment support for the Office of Water.

Research

In August 1998, OST proposed revisions to its human health methodology for deriving ambient water quality criteria (see 63 FR 43755, EPA-822-Z-98-001 for Federal Register notice; see EPA-822-B-98-005 for Technical Support Document). The existing methodology uses BCFs to predict exposure from the consumption of contaminated aquatic species. The revisions to the methodology propose to use BAFs instead of BCFs. BAFs are preferred over BCFs because BAFs are a better predictor of the concentration of a chemical within aquatic organisms since they include consideration of the uptake of contaminants from all routes of exposure, not just uptake from water. EPA's human health methodology includes a methodology for establishing BAFs. The BAF methodology consists of four different approaches. Each approach is ranked (i.e., tiered) in order of preference. Using the proposed methodology, OST is developing National Default BAFs for 28 chemicals to assist states and tribes in updating their ambient water quality criteria. OST is also developing guidance on how to plan and collect field data for deriving BAFs.

OST has developed a simulation tool called AQUATOX, for evaluating bioaccumulation risks. AQUATOX is an ecosystem model that has the ability to model the combined environmental fate and ecological effects of pollutants in aquatic ecosystems. It can simulate the fate of pollutants, including bioaccumulative organic compounds, beginning with their input into the water body, partitioning to the water, sediments, and biotic components, and transfer throughout the food web, and culminating in the tissues of fish that might be consumed by humans or wildlife. Significant ecological processes are simulated, including primary and secondary productivity, trophic structure and dynamics, predator/prey interactions, toxicity, and nutrient dynamics. The model has undergone several validation studies (including one on bioaccumulation of PCBs in Lake Ontario), and is expected to be released in early 2000.

Another EPA model, WASTOX (Water Quality Analysis Simulation for Toxics), is composed of sequential models for calculating the fate and bioaccumulation of toxic chemicals in surface water systems (rivers, lakes, estuaries). The fate model computes time-variable or steady-state concentrations of toxic chemicals in the water column and sediments based on mass balance equations for advective-dispersive transport, phase transfers, and chemical transformations within a multidimensional, segmented water-sediment domain. The bioaccumulation model uses water column and sediment exposure concentrations from the fate model to compute time-variable or steady-state transfers of toxic chemicals through pelagic and/or benthic food webs. In this calculation, toxic chemical accumulations in various compartments of the food web are determined from information on the bioenergetic structure of the food web, gill transfer rates, chemical uptake through ingestion of contaminated prey, chemical egestion/excretion rates, chemical metabolism, and fish migration behavior.

WASTOX was originally developed under cooperative agreements with EPA's ORD laboratory in Gulf Breeze, Florida, and the Large Lakes Research Station of EPA's ORD laboratory in Duluth, Minnesota. The model has been applied to toxic contamination problems at various sites including kepone in the James River (through striped bass), PCBs in Lake Michigan and Green Bay (through lake trout), and PCBs in the Hudson River (through striped bass). A Windows version of the WASTOX model is being developed under a cooperative agreement with EPA's Office of Water, Washington, DC.

EPA is developing a series of analytical tools to screen environmental samples (i.e., sediment, tissue, effluent, ambient water) for chemicals that have the potential to bioaccumulate and move through the food web. The screening methods and background information were first released as draft guidance in 1993. These methods isolate acid-stable, nonpolar organics that have log partition coefficients (log P) values greater than 3.5. Detection levels associated with the different environmental media are 100 ng/L for aqueous samples (e.g., effluent or ambient water) and 50 Fg/kg for sediment and tissue samples.

The screening methods have undergone field validation at two discharge sites. The results of the field validation have been published in scientific journals and as an addendum to the draft guidance. In addition, the analytical procedures have also been the focus of a round robin study to evaluate inter- and intra-laboratory variability. Participants in the round robin study were from 16 national, industrial, and academic laboratories. EPA is currently analyzing the data from the round robin study. As EPA completes the data analysis for each medium, EPA will publish the results in scientific journals and revise the screening methods to address any deficiencies identified in the round robin exercise.

These screening methods will provide an alternative to the "target list" approach for evaluating environmental samples. Instead of looking for specific chemicals, the screening methods generate a list of any compound present in the sample that has a potential to bioaccumulate and its approximate concentration. This approach allows environmental managers to quickly identify chemicals of potential concern and focus on them. EPA recommends that the screening protocol be followed up by chemical-specific analysis to confirm the identification of a chemical and to more precisely determine its concentration.

Assessment

National Study of Chemical Residues in Lake Fish Tissue

The Office of Water is conducting a National Survey of Chemical Residues in Lake Fish Tissue. This study will meet objectives presented in the President's Clean Water Action Plan (CWAP, February 1998) and provide information about persistent, bioaccumulative, and toxic chemicals for the Agency's PBT Initiative.

- CWAP Key Action — EPA and NOAA will conduct a national survey of mercury and other contaminant levels in fish and shellfish throughout the country and will coordinate the effort with states and tribes to maximize geographic coverage. The shellfish survey will be based on the data obtained by NOAA's ongoing Mussel Watch Project.

- The PBT Initiative, begun by EPA's Office of Prevention, Pesticides, and Toxic Substances, seeks to identify areas of concern for human or ecological health. The lake fish tissue study may reveal where PBTs not previously considered a problem are present at levels of concern.

A workshop was held on October 27 and 28, 1998, in Crystal City, Virginia, to review the survey design. More than 40 workshop attendees representing states, other federal agencies, and EPA headquarters, regional offices, and labs reviewed, commented on, and added to the survey design components, including the statistical design, target analytes, field sampling procedures, and data management. This study expands the scope of the 1987 study (USEPA, 1992b) which focused on chemical residues in fish tissue near point source discharges. This new study will

- Provide information on the national distribution of selected persistent, bioaccumulative, and toxic chemical residues (PBTs) in game fish and bottom-dwelling fish in lakes and reservoirs of the continental United States (excluding the Great Lakes).

- Include lakes and reservoirs selected according to a probability design.

- Involve the collection of fish from those randomly selected lakes and reservoirs over a 3-year survey period.

- Include the analysis of fish tissue for PBT chemicals selected from the Agency's multimedia candidate PBT list of 451 chemicals and a list of 130 chemicals from several contemporary fish and bioaccumulation studies.

The study will not be used to set fish consumption advisories. However, states and Native American tribes may choose to initiate a detailed fish study in a particular lake based on the screening contaminant concentrations provided by the national study.

Contaminants monitored in fish tissue will include mercury, PCBs, dioxin, and pesticides as well as other PBT chemicals. The workshop participants form the initial study workgroup consisting of EPA program offices, state and tribal agencies, and federal agencies. EPA will work closely with the state/tribal/federal partners to carry out the study.

National Sediment Quality Survey

The National Sediment Quality Survey (USEPA, 1997d) was prepared in response to the requirement in the Water Resources Development Act of 1992 that EPA conduct a comprehensive national survey of data regarding sediment quality in the United States and report to Congress biennially on the assessment results. For calculations related to bioaccumulation, the survey makes use of fish tissue residue data and models bioaccumulation from sediment using the theoretical bioaccumulation potential approach. Data were compiled from 11 regional and nationwide databases to identify locations throughout the United States where accumulated persistent organic chemicals could pose a threat to aquatic and terrestrial biota and humans. OST used screening-level assessment to identify watersheds for further assessment and management actions to protect sediment quality. As part of the peer-reviewed evaluation methodology, OST compiled benthic and pelagic BSAFs from information provided by NHEERL-Narragansett and NHEERL-Duluth, worked with NERL-Athens to review chemical-specific K_{ow}s, compiled final chronic values (FCVs) from various sources, and developed sediment quality advisory levels (SQALs) for nonionic organics using an equilibrium partitioning methodology. By substituting a protective tissue reside value (TRV) for human or wildlife consumers for a theoretical bioaccumulation potential (TBP; see Section 4.3.2.5), sediment guidelines can be derived for more than 100 chemicals by using the TBP model:

$$\text{TBP-based sediment guideline } (\mu g/g_{oc}) = TRV(\mu g/g_{tissue}) / [f_{lipid}(g_{lipid}/g_{tissue}) * BSAF(g_{oc}/g_{lipid})]$$

Field-measured BSAFs vary depending on food chain biomagnification, metabolism, and many site-specific environmental variables. In most cases, USEPA (1997d) used median values from frequency distributions of field-measured BSAFs by chemical class reported by Tracey and Hansen (1996). Risk-based TRVs for protection of human consumers are available in USEPA (1997a). Information needed to calculate TBP-based sediment guidelines is available in the National Sediment Quality Survey appendices online at www.epa.gov/ost/cs/report.html.

Assessment of Mercury and PCB Contamination

To address a current public health concern, OST is collecting fish tissue data from state and federal agencies to develop a more detailed national picture of the nature and extent of mercury contamination. The project is ongoing, with the initial phase (data collection and compilation) completed in late FY 1997. A data summary report will be published in early 1999. Future directions of this project involve statistical analysis of the spatial patterns of fish tissue residue levels and related factors such as atmospheric deposition, local mercury sources, watershed size, pH, dissolved organic carbon, and acid-neutralizing capacity using GIS. OST also sponsored a technical assistance conference that addressed concerns about mercury in fish (September 1994) and another on PCBs in fish (May 1993).

Assessment/Prevention

OST's Standards and Applied Science Division uses bioaccumulation data and modeling in support of effluent guidelines for industries that discharge persistent organic pollutants in significant amounts, such as the promulgation of effluent limitations guidelines for the pulp, paper, and paperboard industry. In-stream pollutant concentrations are estimated for various treatment technologies based on estimated pollutant loadings. Potential fish tissue concentrations are calculated by multiplying the pollutant-specific BCF by the estimated in-stream concentrations of all pollutants evaluated, except dioxins and furans. Fish tissue levels of 2,3,7,8-TCDD and 2,3,7,8-TCDF are estimated using EPA/ORD's draft Dioxin Reassessment Evaluation (DRE) model (USEPA, 1994f), which estimates fish tissue levels by including the amount of dioxin adsorbed to the organic carbon fraction of sediments suspended in the water column. Carcinogenic risks and noncancer hazards to recreational, subsistence, and Native American anglers are estimated based on different rates of consumption of these potentially contaminated fish.

Dredged Material Management (with Office of Wetlands, Oceans, and Watersheds)

See "Dredged Material Management" under Office of Wetlands, Oceans, and Watersheds (Section 5.1.5.2).

Outreach/Technical Guidance

Guidance for Conducting Fish and Wildlife Consumption Surveys (USEPA, 1998b) provides explicit instructions for selecting a survey approach and designing a survey to obtain consumption rate information. A statistician should also be consulted to provide advice on the specific sampling and statistical analysis considerations for each fish consumption rate assessment project. The survey methods presented in this document may be used by regional, state, tribal, or local agencies to obtain information on the consumption of noncommercially obtained fish and wildlife. This information can then be used to estimate risks to persons who could consume organisms that might contain bioaccumulative and potentially dangerous levels of toxicants, and to develop consumption advisories and point-source discharge loads to protect human health. Such surveys can also provide demographic information about a population for which advisories are issued, which might assist in the communication of risks and advisory recommendations.

OST developed a four-volume series of documents titled *Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories. Volume I: Fish Sampling and Analysis (*second edition) (USEPA, 1995d) provides information on sampling strategies for a contaminant monitoring program. In addition, information is provided on selection of target species, selection of chemicals as target analytes, development of human health screening values, sample collection and analysis procedures, and data

reporting and analysis. *Volume II: Risk Assessment and Fish Consumption Limits* (second edition) (USEPA, 1997a) provides guidance on the development of risk-based meal consumption limits for the high-priority chemical fish contaminants (target analytes). It also contains a discussion of risk assessment methods used to derive the consumption limits, as well as a discussion of methods to modify the limits to reflect local conditions. *Volume III: Risk Management* (USEPA, 1996b) provides guidance on risk management procedures regarding the selection and implementation of various options for reducing health risks associated with the consumption of chemically contaminated fish and shellfish. A tiered approach to developing fish advisories is discussed. Templates are included to enable risk managers to organize their information to evaluate needs and to identify the optimal group of options and consumption limits. *Volume IV: Risk Communication* (USEPA, 1995e) provides guidance on risk communication as a process for sharing information with the public on the health risks of consuming chemically contaminated fish and shellfish.

Outreach/Database Management

The *Listing of Fish and Wildlife Consumption Advisories* (USEPA, 1997b) is a database of all fish and wildlife consumption advisories issued by various state, federal, and local agencies. Included in the database is information regarding types of advisories; species and chemical included in the advisory; contaminant levels; segments of the population affected; percentage of water bodies under advisory by state, region, or nationally; georeferenced locations of advisories; dates of issue; and agency contacts with phone numbers. The database can also generate maps that illustrate any combination of these parameters. The database is updated annually and is available on the Internet at www.epa.gov/ost/fish.

5.1.5.2 Office of Wetlands, Oceans, and Watersheds (OWOW)

OWOW was created to integrate the protection and management of our nation's watersheds, coastal and marine waters, and wetlands. OWOW is made up of three Divisions—the Oceans and Coastal Protection division, the Wetlands Division, and the Assessment and Watershed Protection Division—all three of which manage national programs that use bioaccumulation testing.

OWOW plays a significant role at regional, national, and international levels in efforts to manage dredged material. The Oceans and Coastal Protection Division (OCPD) manages the disposal of dredged material in the ocean under the Marine Protection, Research and Sanctuaries Act (MPRSA). Under MPRSA, OCPD develops regulations that set forth the criteria for ocean dumping, which include criteria for designating ocean dumping sites, issuing ocean dumping permits, and testing material proposed for ocean dumping. The Wetlands Division (WD) manages the discharge of dredged material in waters of the United States under section 404 of the Clean Water Act. WD develops regulations that set forth the criteria for such discharge, which include criteria for choosing disposal sites, issuing permits, and testing material proposed for disposal in inland waters.

OWOW's responsibilities include development of the environmental criteria used by the U.S. Army Corps of Engineers (USACE, the permitting authority for dredged material) in evaluating dredged material for proposed disposal. In addition, OWOW develops national guidance to the regions (jointly with the USACE) regarding dredged material management, and it provides technical assistance to the regions on site selection or designation, testing, and permitting issues.

The Assessment and Watershed Protection Division (AWPD) serves as the national program manager for EPA's nonpoint source control efforts. AWPD assists states in implementing programs that target watersheds for Total Maximum Daily Loads (TMDLs), thus helping to control potential sources of

sediment contamination, including bioaccumulative material; develops nonpoint source management practices; and assesses the conditions of our nation's watersheds.

Dredged Material Management

OWOW manages the national dredged material management program for EPA. OCPD and WD regulate and manage dredged material disposed of in ocean and inland waters, respectively. OCPD manages dredged material disposal in ocean waters under MPRSA and the Ocean Dumping Regulations. The regulations describe the criteria that must be met for materials disposed of in the ocean (dredged material or other materials). The national testing manual (the "Green Book"), developed jointly with the USACE, describes the tests and procedures recommended for use in determining whether dredged materials meet the regulatory requirements (USEPA and USACE, 1991). Bioaccumulation of contaminants is one of the factors described in the regulatory criteria and is one of the factors for which test procedures and evaluation guidance are contained in the Green Book. As provided in the Green Book, each EPA region or USACE district involved in ocean dumping is to use the national guidance in developing local testing and evaluation procedures based on the contaminants of concern and species existing in a given area, and the levels of contaminants in the sediments already existing in the area of the disposal site (the reference). Based on the regulatory requirements and the 1991 Green Book guidance, the regions follow a tiered approach to testing that includes conducting 28-day bioaccumulation tests on the dredged material with at least two species, and comparison of the resultant data with FDA action limits, bioaccumulation levels observed in the same species exposed to reference sediments in 28-day bioassays, and other factors listed in the Green Book such as magnitude of bioaccumulation above reference and toxicological importance of the resultant bioaccumulation. Toxicological importance of the 28-day bioaccumulation results is evaluated by comparison to regionally appropriate values of specific contaminants in tissue that have been shown to indicate tissue concentrations associated with significant undesirable effects.

WD regulates dredged material disposed in inland waters under section 404 of the Clean Water Act (CWA). These 404(b)(1) guidelines, regulations promulgated by EPA under the CWA, describe the criteria that must be met for materials disposed of in inland waters. WD is also responsible for implementing the testing procedures in the Inland Testing Manual (USEPA/USACE, 1998), jointly developed by EPA and the USACE.

Assessment/Prevention

Section 301(h) of the 1977 Clean Water Act allows EPA, with concurrence from states, to issue NPDES permits to Publicly Owned Treatment Works (POTWs) for the discharge of less-than-secondary-treated effluent. The marine discharge waiver program under section 301(h) provides an opportunity for waiver of the secondary treatment requirements for sewage discharged by cities and towns to marine waters and unstressed estuaries. Applicants must demonstrate that the discharge will not degrade water quality from levels that ensure the protection of public water supplies, the protection and propagation of balanced, indigenous populations of shellfish, fish and wildlife, and the protection of recreational activities on or in the water. OCPD develops the criteria used to evaluate such waiver requests. The deadline for 301(h) applications closed in 1982, and 45 applicants/permittees remain in the program.

OCPD also developed a revised technical support document for modifications of secondary treatment requirements for POTWs under the National Pollutant Discharge Elimination System (NPDES) permitting program (USEPA, 1994a). The support document provides technical guidance for implementing the 301(a) requirements and the accompanying regulations (40 CFR Part 125, Subpart G).

Among other things, the support document explains the toxic control requirements of section 301(h), which include consideration of bioaccumulation of sediment contaminants. POTWs are required to provide a schedule for development and implementation of nonindustrial toxic control programs if their discharges contain these contaminants. The law also requires POTWs to describe their public education programs for minimizing entrance of nonindustrial toxic pollutants and pesticides into their treatment systems and to describe industrial pretreatment requirements, if applicable.

Section 403(c) requires that discharges from point sources into ocean waters cause "no unreasonable degradation" to the marine environment. OCPD develops the environmental criteria used under section 403 in evaluating requests for NPDES permits for discharges to ocean waters. Both the 403(c) and 301(h) programs evaluate the ecological impacts of direct discharges to the marine environment and provide the Agency with a geographically targeted approach toward managing risk and protecting sensitive habitats that need the greatest protection.

Under both the 301(h) and 403 programs, the Agency considers the quantities of and potential for bioaccumulation of discharged pollutants, the potential for pollutant transport, potential harm to biological communities, and direct and indirect effects on humans. The 301(h) program developed a series of technical guidance documents to provide state-of-the-art methods of marine environmental monitoring and assessment to 301(h) applicants/permittees to aid in preparing 301(h) waiver applications, making waiver decisions, and designing and evaluating marine environmental monitoring programs.

OCPD has prepared a bibliography of documents that address methods for monitoring toxic substances and investigating bioaccumulation of toxic substances by marine and estuarine organisms (USEPA, 1994c). Bioaccumulation monitoring guidance documents address the qualities of target species and methods for assessing bioaccumulation; monitoring program design, including sampling of caged or indigenous indicator species; the type of tissue to be analyzed in invertebrates and fishes; and techniques for extracting and analyzing chemical contaminants. For example, the technical documents provide guidance on selecting target species for bioaccumulation studies in coastal areas (USEPA, 1987a). Tissue chemistry data for the target species recommended in the first volume were compiled in the second volume (USEPA, 1987b).

The 301(h) technical guidance has generally been well received and adapted as a basis of marine monitoring in other EPA marine-related programs, including programs related to section 403 ocean discharges and other marine discharges and marine disposal activities. OCPD developed *CWA Section 403: Procedural and Monitoring Guidance* (USEPA, 1994d) and similar guidance for the National Estuary Program, including the design and conduct of bioaccumulation monitoring studies to link exposure and effects and to examine risks to target species and humans (USEPA, 1992a). States have also adapted the 301(h) technical guidance for use in marine/ocean discharge programs.
OCPD also supports studies of environmental conditions at point sources and ocean dumping sites to manage impacts and protect the marine environment. Field surveys are conducted to support the designation of ocean disposal sites and to assess impacts of past and ongoing ocean disposal operations and discharges.

Remediation

The MPRSA and CWA regulate all discharge of material dredged from navigable waters and set forth the environmental criteria to be used in making such permit decisions. For example, when existing contaminated sediment in navigable waters requires *in situ* remediation using dredged material, the

proposed remediation material is evaluated based on applicable environmental criteria set forth in the MPRSA and CWA regulations. The criteria include bioaccumulation assessment when appropriate.

Research

There are ongoing research efforts in dredged material management and testing. For example, EPA, jointly with the USACE, is continuing to develop the Environmental Residue Effects Database (ERED), which contains bioaccumulation data from published research linked with concentration and its effects (USACE, 1997). ERED has been developed to reduce the level of uncertainty associated with interpreting bioaccumulation data for the purpose of making regulatory decisions regarding dredged material.

Outreach

OWOW and the EPA regions are involved in many forms of outreach regarding dredging-related issues. OCPD's outreach efforts include educating the general public about the ocean and coastal programs and making program information readily available to the public. Dredged material management-related testing and evaluation manuals, which include *Evaluating Environmental Effects on Dredged Material Management Alternatives - a Technical Framework*, *Overview of Dredged Material Testing Framework*, *Ocean Dumping Testing Requirements*, and the Green Book are available on the OCPD homepage (http://www.epa.gov/owow/oceans/).

The National Dredging Team (NDT) is an interagency group cochaired by EPA and the USACE. It includes the U.S. Fish and Wildlife Service (USFWS), the Maritime Administration, the National Oceanic and Atmospheric Administration's Office of Ocean and Coastal Resource Management, and NOAA's National Marine Fisheries Service. It was established in 1995 to promote national and regional consistency on dredged material management issues and to provide a forum for conflict resolution and information exchange. NDT's homepage is located at http://www.epa.gov/owow/oceans/ndt/.

OWOW and the USACE developed a guidance document, *Identifying, Planning and Financing Beneficial Use Projects Using Dredged Material.* Beneficial use of dredged material is considered an environmentally and economically sound way of disposing of dredged material. This document provides information to the general public on how to seek opportunities for beneficial uses of dredged material.

Information (fact sheets) regarding other marine and coastal programs (e.g. "Clean Water Act Section 403 - a Framework for Ecological Risk Assessment") are available on the OCPD homepage.

5.1.5.3 Office of Wastewater Management (OWM)

OWM oversees a range of programs contributing to the well-being of our nation's waters and watersheds. Through its programs and initiatives, OWM promotes compliance with the requirements of the Federal Water Pollution Control Act (Clean Water Act).

Prevention

Section 402 of the CWA authorizes the National Pollutant Discharge Elimination System (NPDES) permitting program, administered by the Office of Wastewater Management (OWM) within OW, to regulate the discharge of pollutants from point sources into navigable waters. Bioaccumulation screening methods can be used to identify chemicals of potential concern in the sediments, followed by chemical-

specific analysis for confirmatory purposes. Until the states adopt numeric criteria into their standards for sediment contaminants based on bioaccumulation, the NPDES program will not require permitting authorities to include, in their NPDES permits, sediment bioaccumulation-based numeric limits. However, states have the discretion to include such limits in permits based on an interpretation of their narrative standards for toxic substances. To establish such permit limits, it will be necessary for permitting authorities to develop Waste Load Allocations (WLAs) for the relevant sediment contaminants. After reviewing available fate, transport, and effect models, EPA's Office of Science and Technology (OST) has identified models that will allow development of WLAs. EPA's Office of Water will supply technical support for users of these models. Current resources for addressing the challenges of sediment bioaccumulative contaminants are the Technical Support Document (TSD), the Great Lakes Initiative (GLI), the draft User's Guide for Multi-program Implementation of Sediment Quality Criteria in Aquatic Ecosystems (under development by EPA OST), and the watershed permitting approach.

5.2 EPA Regions

Regional Administrators develop, propose, and implement regional programs for comprehensive and integrated environmental protection activities. The regional offices support the Agency's overall mission by translating technical program direction and evaluation for various Assistant Administrators and heads of headquarters staff offices into effective operating programs at the regional level. In addition, regional offices ensure that such programs are executed efficiently, that approval authority for proposed state standards and implementation plans is exercised appropriately, and that overall and specific evaluations of regional programs are provided. All EPA regions implement the dredging programs under the CWA and MPSRA as described in Section 5.1.5.2 of OWOW programs.

5.2.1 Region 1

The Region 1 office oversees environmental protection issues in the states of Connecticut, Maine, Massachusetts, New Hampshire, Rhode Island, and Vermont.

Remediation

In EPA New England (Region 1) various data have been collected to assess the potential for bioaccumulation during the evaluation of ecological risks at several stages within the CERCLA process, which include the Remedial Investigation, Feasibility Study, and long-term monitoring as part of the Record of Decision (ROD) requirements.

During screening-level ecological risk assessments, predictive food chain models have been calculated using literature-derived BAFs. If the results of the literature-derived food chain models demonstrate an unacceptable risk to the ecological receptors, a risk management decision is usually made to revise risk analysis and decrease the uncertainties carried through risk characterization since contaminant-specific BAFs are quite variable. At this point within the ecological risk assessment process, revisions to risk analysis may include the collection of site-specific biota such as fish and invertebrates in order to develop a site-specific BAF to integrate into an avian or mammalian food chain model to provide a more realistic and confident assessment of exposure.

Over the last several years, through the efforts of the regional Biological Technical Advisory Group (BTAG) and the assistance of USFWS, the majority of bioaccumulation studies have focused on the collection and chemical analysis of various fish species as part of the baseline ecological risk assessment within the Remedial Investigation phase. In general, fish species are collected with electrofishing methods

to represent the three major feeding guilds—bottom feeders (i.e., brown bullheads), forage feeders (i.e., pumpkinseeds or bluegills), and predators (i.e., largemouth bass)—so as to be able to evaluate both piscivorous avian (i.e., great blue heron) and mammalian (i.e., river otter) receptors of concern. Once the fish species are collected, fish may be analyzed as whole bodies or separately as fillet and offal (remainder of fish minus the fillet) portions and then fillet and offal information added together so that both the human health and ecological risk assessments can benefit from this information. The greatest confidence with these types of bioaccumulation field efforts is associated with the integration of site-specific tissue residue concentration into the ecological risk assessment. In contrast, the greatest amount of uncertainty is associated with the inability to evaluate the potential adverse effects on the fish population itself from the exposure data. Therefore, tissue residue contaminant concentrations are predominately used to indirectly evaluate the effects on secondary consumers. Unfortunately, there is a paucity of effects data that can be correlated with exposure data to be able to determine the likelihood of ecological risk, especially for contaminant concentrations for terrestrial vegetation, submerged aquatic vegetation, and nonvascular plants such as seaweeds.

Following the collection and analysis of these data, along with other measures of exposure and effects (which could include the laboratory results of a toxicity test or the analysis of the benthic community), the weight of this evidence is discussed in the final phase of the ecological risk assessment, risk characterization. As part of the Feasibility Study process in which the remedial alternatives are evaluated, the results of the ecological risk assessment, which may be supported through the collection and interpretation of bioaccumulation data, are used to select a potential remedial action.

For sites in which the major contaminants of concern have been compounds known to bioaccumulate, such as DDT and its breakdown products DDD and DDE along with methylmercury, site-specific food chain models have been generated. These models have been used to establish the preliminary remedial goal (i.e., cleanup number) for contaminated sediments if both the sediment and biota were collected and analyzed so as to be able to develop the site-specific BSAF.

The collection and use of bioaccumulation has also been used as a component of the long-term monitoring at sites where actions have occurred at source areas and measures such as fish tissue residue data are needed to ensure the effectiveness of the remedial action. In contrast, these activities may be undertaken to monitor the consequences of a no-further-action risk management decision.

5.2.2 Region 2

EPA's Region 2 includes the states of New Jersey and New York, as well as Puerto Rico and the U.S. Virgin Islands.

Remediation

In Region 2, bioaccumulation testing is being used during the evaluation of ecological risks at the Remedial Investigation stage of the CERCLA process. Such testing is also a condition of the Record of Decision (ROD) to ensure that long-term monitoring is conducted when contaminants of concern are left in place following the completion of a remedial action. Tissue data are used to determine whether there is a risk to ecological receptors; they have not been used to calculate sediment clean-up numbers. Further, tissue numbers in themselves are not used as clean-up numbers or preliminary remedial goals.

Bioaccumulation Studies and the Remedial Investigation

During Step 4 of the ecological risk assessment process (*Ecological Risk Assessment Guidance for Superfund: Process for Designing and Conducting Ecological Risk Assessments*, EPA 540-R-97-006), it is recommended that tissue be collected to gain a better understanding of the bioavailability of contaminants. These site-specific data are also employed in models to reduce the uncertainty associated with calculating the dose to higher-trophic-level receptors. In situations where a water body might be affected by site contaminants, the BTAG often recommends the collection of whole body fish. In some instances, to take advantage of the human health data being collected, it is requested that fish fillets collected for human health purposes be recombined (e.g., the carcass and the fillet are analyzed and mathematically combined) to enable data to be used for ecological purposes. Fish recommended for collection include bottom dwellers, those with a limited home range, and those most likely to be consumed by piscivorous receptors of concern (such as blue heron or mink). In some studies EPA recommends that caged bivalves (placed in contact with the sediment) be used to gain a better location-specific understanding of the system. The study is designed so that sediment chemistry, benthic community assessments, toxicity studies, and caged bivalves are collected from one location.

Although not frequently requested, benthic tissue data collection may also be used to gain a better understanding of site-specific characteristics. These data also help to reduce uncertainty by allowing the risk assessor to use a real bioaccumulation factor (between sediment and organism) in models.

Tissue studies of other organisms include small mammals such as shrews, voles, and mice. The data are then used to calculate a dose to upper-trophic-level receptors such as fox, mink, weasel, woodcock, owl, and hawk. The calculated dose is then compared to literature values and hazard quotients are computed to determine whether there is a potential for risk. These types of studies have been conducted at a number of Superfund sites.

Bioaccumulation Studies at the ROD Stage and Beyond

To determine the effectiveness of the remedy proposed at some CERCLA sites, long-term monitoring, which may include tissue collection, is being recommended more frequently. This approach is particularly useful at those sites where contaminants left in place have the potential to adversely affect the environment. In some cases it has been determined that the value of the habitat outweighs the risk of leaving contaminants in place. At two Superfund sites, PCBs are being left in place because of the value of the wetland habitat. Baseline fish tissue will be collected, and fish tissue will be collected following remedial action at one site and after a couple of years at the other. In the event that fish body burden levels do not drop after a period of time, further remedial action (i.e., sediment excavation) might be warranted.

At one site, postremedial monitoring includes tissue residue analyses for PCBs in field-collected fiddler crab and mummichog, as well as in clams exposed to site sediments in the laboratory. The goal is to demonstrate a negative trend in biotic PCB concentrations by the 5-year ROD review.

At one site EPA is proposing that amphibians (frogs) be used to determine whether soil and sediment removal for lead has successfully removed the contaminant from the ecosystem. In this situation, the availability of an adequate database to which tissue values could be compared is not critical. The study will examine lead body burdens and will look for a reduction over a period of time. At another site, the need to remediate the Westerly Wetlands will depend on the detection of significant reduction in growth or survival, or significant impact on higher-trophic-level receptors based on tissue residues (which are

then compared to literature values). Some organisms that might be included for studies are shrews and voles, which have been previously collected.

5.2.3 Region 3

Region 3 is composed of Delaware, the District of Columbia, Maryland, Pennsylvania, Virginia, and West Virginia.

Assessment/Outreach

On December 10, 1992, the Chesapeake Bay Program's (CBP) Toxics Subcommittee sponsored a "Chesapeake Bay Contaminated Sediment Critical Issue Forum" as part of the ongoing reevaluation of the Chesapeake Bay Basinwide Toxics Reduction Strategy. In 1993, the Chesapeake Executive Council designated three areas as toxic "hot spots" or "Regions of Concern," due in large part to sediment contamination: Baltimore Harbor, Anacostia River, and Elizabeth River.

The CBP's Toxics Subcommittee's Regional Focus Workgroup is currently conducting a toxics characterization outside these three Regions of Concern to identify any additional chemical contamination problems in the tidal tributaries of the bay. This characterization is based on an analysis and integration of water/sediment/fish tissue contaminant concentration data, water/sediment toxicity data, benthic community structure data, and any other biological effects data available. Based on a weight-of-evidence approach, the CBP will place segments of the bay's tidal tributaries into one of four categories, depending on the level of contamination: Regions of Concern (hot spots); Areas of Emphasis (warm spots); Areas with Low Probability for Adverse Effects (no problem spots); and Areas with Insufficient Data. The CBP has developed a set of "decision rules" for how to interpret the data. Sediment contaminant concentration data are compared to a variety of sediment thresholds (e.g., equilibrium-partitioning sediment guidelines [ESGs], NOAA effects range-low [ER-L] and effects range-median [ER-M] values; Environment Canada threshold effects level [TEL] and probable effects level [PEL] values). Fish tissue contaminant concentrations are compared to FDA action levels, FDA levels of concern, and EPA screening levels. Based on these comparisons and any associated water chemistry data and effects data, the Regional Focus Workgroup uses best professional judgement to characterize an area into one of the four categories. The workgroup is composed of scientists, regulators, industries, and managers. This characterization will be used as an outreach tool to put information in the hands of the public, scientists, and managers to target the appropriate areas for additional monitoring and management activities. This characterization will be updated every 3 years.

Prevention

The Toxics Subcommittee has also developed a risk-based chemical ranking system to evaluate chemical contaminant data in order to identify a short list of chemicals causing or having the potential to cause an adverse impact on the Bay's living resources. This chemical ranking system was used to revise the Chesapeake Bay Toxics of Concern chemical list. This revised list is still under review. The list was developed considering the source (loadings), fate (bioconcentration and environmental persistence), and exposure/effects (water column, sediment, and fish tissue) of chemical contaminants measured in the Chesapeake Bay.

Research/Remediation

The NOAA Chesapeake Bay Office (NOAA CBO) has a committee called the Chesapeake Bay Environmental Effects Committee, which sponsors research on environmental effects of toxicants, some of which includes bioaccumulation and food web work. In 1996 the program funded sediment transport research to provide managers with critical information to aid them in decisions regarding how to effectively deal with contaminated sediment in the Regions of Concern. This work may help managers make wise decisions regarding remediation, dredging, and capping projects. The CBP is working in concert with the NOAA CBO to develop a 5-year plan for the research program that will be finalized in late 1998.

5.2.4 Region 5

Region 5 consists of Illinois, Indiana, Michigan, Minnesota, Ohio, and Wisconsin.

Assessment/Remediation

Water Division and Superfund/RCRA-driven sediment remediations tend to use site-specific risk assessment information in deriving sediment clean-up goals. For bioaccumulative contaminants such as PCBs, the main exposure and risk pathways are ingestion of contaminated fish and incidental ingestion of contaminated sediment. To assess the fish ingestion pathway, a model/algorithm (BSAF) is used to derive acceptable levels in sediment that will allow for safe fish consumption, based on site-specific exposure assumptions. In addition, best professional judgment and negotiations will shape data issues (and resulting clean-up goals) such as defining area, averaging methods, conservatism of assumptions, and use of background contaminant data. The most recent regional thinking on how to approach these clean-up decisions is the subject of the draft S2F document (see Research below) and has been applied at some sites. In some cases the incidental ingestion pathway-driven clean-up goal (vs. fish ingestion) can result in lower clean-up goals. In another example, fish consumption, coupled with RCRA-benzene determinations and nonbioaccumulative NOELs/LOELs, is being used to leverage more removal.

Situations without PCBs can also be problematic. Dioxin sediment contamination can also result in fish contaminant concerns and thus lead to examination of sediment levels and effluent discharges. Control of existing effluents has usually been determined to be the most appropriate remedy, with sediment remediation not deemed necessary.

Research

The Region 5 Sediments to Fish (S2F) Workgroup, a joint project of the Region 5 In Place Pollutant Task Force and Health Effects Forum, reviewed relevant material and deliberated on risk and sediment issues. Because fish consumption might be a significant exposure pathway for humans and fish-eating mammals and birds, the workgroup agreed that a sound methodological approach for sediment-to-biota modeling was needed to provide support for both human health and ecological risk assessments. Results of the workgroup's study are compiled in a report entitled *Predicting Bioaccumulation of Sediment Contaminants to Fish* (USEPA, 1994h).

Research/Outreach

The *Great Lakes Water Quality Initiative Technical Support Document for the Procedure to Determine Bioaccumulation Factors* (USEPA, 1995c) provides technical information and a rationale in

support of the methods to determine BAFs. BAFs, together with the quantity of aquatic organisms eaten and the percent lipid, determine the extent to which wildlife and humans are exposed to chemicals through the consumption of aquatic organisms. The more bioaccumulative a pollutant is, the more important the consumption of aquatic organisms becomes as a potential source of contaminants to humans and wildlife. BAFs are needed to determine both human health and wildlife Tier I water quality criteria and human health Tier II values, as well as to define bioaccumulative chemicals of concern, which were identified for the GLI to focus pollution prevention and remediation efforts.

Great Lakes National Program Office (GLNPO)

Assessment/Remediation

Thirty-one problem harbors and tributaries in the Great Lakes have been identified and labeled as Areas of Concern (AOCs). Contaminated bottom sediments have been identified as a significant problem that must be addressed to attain beneficial uses at all of these AOCs. The Assessment and Remediation of Contaminated Sediments (ARCS) Program was a 5-year demonstration project to help address the contaminated sediment problem at these locations. The goals of the ARCS Program were to develop an integrated, comprehensive approach to assess the extent and severity of sediment contamination; to assess the risks associated with that contamination; and to select appropriate remedial responses. This information was developed to help support implementation of Remedial Action Plans at the AOCs. Major findings of the ARCS Program are presented in a summary report (USEPA, 1994b).

As part of the ARCS Program, baseline human health risk assessments were conducted for five AOCs, and baseline aquatic and wildlife risk assessments were performed for the Buffalo River, New York. Comparative human health risks associated with direct and indirect exposures to contaminated sediments in the lower Buffalo River under selected remedial alternatives were estimated for carcinogenic and noncarcinogenic effects (Crane, 1995). Additionally, noncarcinogenic risks of ingestion of PCB-contaminated carp were estimated for mink, as an indication of ecological risks to piscivores. It was shown that risks could be reduced under the different remedial alternatives compared to no action, particularly if dredging was the selected option.

Currently, as a follow-up to the ARCS Program, GLNPO is conducting sediment assessments in many of the Great Lakes AOCs. As part of the assessment package, sediment bioaccumulation work is conducted on a case-by-case basis using the *Lumbriculus* bioaccumulation test. To date, this test has been conducted in Waukegan Harbor, Illinois; White Lake, Michigan; and Duluth Harbor, Minnesota.

In a number of locations around the Great Lakes, high contaminant concentrations in fish tissue have been key factors in moving forward with remedial activities. In the Ottawa River in Toledo, Ohio, PCB concentrations up to 510 ppm were found in carp. Fish consumption advisories are in effect for the entire river. Due in large part to these extremely high fish contaminant concentrations, a sediment remediation project was conducted in the spring of 1998. A total of 10,000 cubic yards of PCB-contaminated sediment (including 56,000 pounds of PCBs) was removed from a tributary to the Ottawa River. Fish will continue to be monitored to determine the success of the remedial project. High PCB concentrations in fish are also driving a second sediment remedial project on the Hayton Mill Pond of the Manitowoc River in Wisconsin. Fish tissue concentrations in the vicinity of the Hayton Mill Pond average 16 ppm PCB and range up to 77 ppm. Fish advisories have been in effect on the river since 1991. These bioaccumulation findings are helping move the site forward toward remediation, which is currently scheduled for the spring of 1999.

5.2.5 Region 6

EPA's Region 6 consists of the states Arkansas, Louisiana, New Mexico, Oklahoma, and Texas.

Assessment

EPA Region 6 performs very few bioaccumulation tests other than those required for the ocean dumping program. Typically, the region and states conduct chemical analysis of sediment and fish tissue to assess the degree of bioaccumulation and whether the sediment could be a source. A number of years ago, Region 6 required 28-day bioaccumulation tests performed with effluent, as well as collection of ambient water and sediment data, to evaluate whether effluent limits for several facilities discharging PCBs were appropriate and adequately protective. The EPA freshwater methodology (USEPA, 1994g) will aid in future sediment bioaccumulation work. The most easily implemented approach to assess bioaccumulation would be the development of chemical-specific guidelines for sediments that address bioaccumulative effects.

5.2.6 Region 7

Region 7 consists of the midwestern states Iowa, Kansas, Missouri, and Nebraska.

Assessment

Fish tissue analysis is the only bioaccumulation analysis being conducted in the Environmental Services Division. Two programs are involved in the collection and analysis of fish tissue—the Regional Ambient Fish Tissue Monitoring Program (RAFT) and the Regional Environmental Monitoring and Assessment Program (R-EMAP).

5.2.7 Region 9

Region 9 consists of Arizona, California, Hawaii, Nevada, American Samoa, and Guam.

Monitoring

Bioaccumulation testing and data are important components in numerous state and federal programs taking place within the region, such as the following: the San Francisco Bay Regional Monitoring Program, the Southern California Bight Regional Monitoring Program, and the routine monitoring of ocean disposal sites. In addition, several dischargers collect bioaccumulation data as part of their regular permit monitoring under the NPDES program.

Assessment/Remediation

The Region 9 Superfund program and Federal Facilities Cleanup Office (FFCO) routinely collect and use sediment bioaccumulation data. These programs use whole sediment, and occasionally chemical-specific, bioaccumulation data in their project-specific risk assessments. For example, Superfund's United Heckathorn remediation and Palos Verdes Shelf EE/CA involve comprehensively evaluating both human health and ecological risk endpoints, primarily for DDT compounds. The FFCO's numerous base closure-related projects typically evaluate a variety of contaminant mixtures in whole sediments.

5.2.8 Region 10

Alaska, Idaho, Oregon, and Washington are included in Region 10.

Assessment

The Columbia River Inter-Tribal Fish Commission (CRITFC) Exposure Study is a multiphase study sponsored by EPA to assess the exposure of the Nez Perce, Warm Springs, Umatilla, and Yakama Tribes to contaminants through fish consumption (USEPA, 1995b). The first phase of the study determined fish consumption patterns of the tribes. A study design for the second phase has been developed and includes collection and contaminant analysis of fish tissue at a number of sites. Tissue will be collected from resident and anadromous fish species consumed by tribal members and caught from tribal fisheries in the Columbia River Basin.

Remediation/Assessment

For bioaccumulative contaminants, sediment remediations tend to use site-specific field measurements as well as laboratory test data as the basis for risk assessment/management evaluations in deriving sediment clean-up goals. The main exposure and risk pathways are ingestion of contaminated fish or shellfish and incidental ingestion of contaminated sediment, although dermal contact and respiratory contact have been issues at some sites. PCBs, tributlytin (TBT), and mercury are the most frequent bioaccumulative chemicals of concern encountered. The Superfund program and the state of Washington's Toxic Cleanup Program coordinate routinely with the technical staff of the interagency Cooperative Sediment Management Program (CSMP) and frequently include them as part of the remediation study team as well as on special studies.

Region 10 Superfund published a final report on TBT, *Recommendations for Screening Values for Tributyltin in Sediments at Superfund Sites in Puget Sound, Washington* (USEPA, 1996c). An extensive literature search was conducted to develop screening values, including review of published research, regulatory standards and criteria, calculations of apparent effects threshold (AET) values and effects ranges, and examination of sediment-tissue and sediment-water partitioning. Bioaccumulation testing is recommended, in conjunction with bioassays and use of a sublethal effects endpoint, to confirm the ecological significance of interstitial water TBT concentrations. The approach will be used to identify TBT-contaminated sediments that should be addressed in remedial actions.

Assessment/Research

Human Health Sediment Quality Criteria

Taking the lead for the CSMP, the Washington State Department of Ecology ("Ecology") is developing human health-based criteria for sediments in Puget Sound. Technical development efforts have resulted in a number of technical reports, which are available from Ecology. (Many of the reports are available through the Internet at http://www.wa.gov/ecology.) The most significant issues facing the agency are determination of a fish consumption rate, development of chemical-specific BSAFs, and implementation within the structure of the State Sediment Management Standards. See the discussion in Section 3.5 for more information about Ecology's approach.

Quantifying Mercury Bioaccumulation

Mercury bioaccumulation presents a potential risk to human health at several sites in Puget Sound. Since mercury is of human health concern, there is a need to quantify the relationship between tissue mercury concentrations and sediment concentrations so that health-protective clean-up and source control decisions can be made. However, surprisingly few data are available in the literature that describe this relationship. As a result, Ecology has been evaluating a number of statistical approaches for determining an acceptable mercury sediment concentration given available data sets in Puget Sound. The Puget Sound Ambient Monitoring Program (PSAMP) has provided the most robust data set currently available.

5.3 International Efforts

The United States is engaged in a number of international efforts to reduce human and environmental exposure to persistent toxic compounds. Current efforts include a wide variety of activities, but the greatest focus is on efforts aimed at the reduction of persistent organic pollutants such as PCBs and selected heavy metals, most notably mercury and lead. Longstanding measures taken by the United States, Canada, and other western nations to reduce the domestic use and emissions of many of the most harmful of these substances have recently been recognized as insufficient to fully control the risks posed by these substances. This is largely due to the proclivity of such substances to travel long distances through environmental media and thereby pose risks of a transboundary or even global nature. In response, the United States and other countries have promoted a growing number of international efforts to assess and manage the transboundary risks.

Years of work in Scandinavia, Canada, and the United States has focused international attention on a group of chemicals known as “persistent organic pollutants,” or POPs. The precise definition of these pollutants is a matter of scientific and even political debate, but there is general agreement on four basic characteristics—persistence, toxicity, bioaccumulation potential, and ability to be transported long distances through the atmosphere or water. A more detailed discussion of the relevant characteristics is presented under Section 4.4.1. International fora at both the regional and global level are faced with four essential tasks: selecting an initial list of POPs for joint action; agreeing on needed control measures, which could be of either a legally binding or voluntary nature; establishing a process for selecting additional POPs for control; and determining the efficacy of joint action. The United Nations Environment Programme’s (UNEP) list of 12 POPs is generally accepted as the principal focus of international action in the near term. The list of 12 includes aldrin, chlordane, DDT, dieldrin, endrin, heptachlor, hexachlorobenzene, mirex, toxaphene, PCBs, and dioxins and furans. Separate, but related international efforts are also under way to address lead, mercury, and cadmium.

Each of the persistent organic pollutants identified above, as well as lead, mercury, and cadmium, is a potential bioaccumulative chemical of concern in contaminated sediments. Consequently, actions taken in the international context to reduce or eliminate emissions of these substances are critical to reducing sediment loadings over the long term.

In light of the continued use and release of persistent toxic substances in many countries and the effect on U.S. ecosystems and human health (including sediment bioaccumulation), the United States is pursuing international action in many international fora. The principal fora are described in the subsections that follow:

5.3.1 U.S.-Canada Great Lakes Water Quality Agreement (GLWQA)

[EPA leads: Great Lakes National Program Office, OW] In keeping with the objective of the Revised Great Lakes Water Quality Agreement of 1978, as amended by the Protocol signed November 18, 1987 (1987 GLWQA) to restore and protect the Great Lakes, Canada and the United States signed "The Great Lakes Binational Toxics Strategy" on March 27, 1997. The Strategy sets forth a collaborative process by which Environment Canada and the United States Environmental Protection Agency, in consultation with other federal departments and agencies, Great Lakes states, the Province of Ontario, tribes, and first nations, will work in cooperation with their public and private partners toward the goal of virtual elimination of persistent toxic substances resulting from human activity, particularly those which bioaccumulate, from the Great Lakes Basin, so as to protect and ensure the health and integrity of the Great Lakes ecosystem. In cases where the strategy addresses a naturally occurring substance, it is the anthropogenic sources of pollution that, when warranted, will be targeted for reduction through a life-cycle management approach so as to achieve naturally occurring levels. An underlying tenet of this strategy is that the governments cannot by their actions alone achieve the goal of virtual elimination. The strategy challenges all sectors of society to participate and cooperate to ensure success. The Binational Toxics Strategy will protect human health and ecosystem health, as well as further the environmental justice objectives of the Agency, through the targeting of PCBs and mercury, the contaminants primarily responsible for fish consumption advisories. Successful implementation of the Binational Toxics Strategy will assist in eliminating fish advisories in the Great Lakes. Recognizing the long-term nature of virtual elimination, the strategy provides the framework for actions to achieve quantifiable goals in the timeframe 1997 to 2006 for specific persistent, bioaccumulative toxic pollutants. Flexibility is provided in the strategy to allow for the revision of targets, time frames, and the list of substances.

5.3.2 North American Commission for Environmental Cooperation (NACEC)

[EPA leads: OPPTS, ORD, OIA] The NACEC Council Resolution on the "Sound Management of Chemicals," adopted in October 1995, establishes a general framework for United States-Canada-Mexico cooperation on chemicals, including a high-level working group to direct this cooperation. Under the resolution, priority for work is to be given to persistent toxic substances. Substantive work began with the development of regional action plans (NARAPs) for PCBs, DDT, chlordane, and mercury, as well as the development of and agreement on a process for selecting additional substances for joint action. At present, work is focused on implementation of the DDT and chlordane NARAPs and screening of other persistent toxics for eventual submission to the formal substance selection process.

5.3.3 United Nations Economic Commission for Europe/Convention on Long-Range Transboundary Air Pollution (LRTAP)

[EPA lead: OAR] Based on 5 years of technical substantiation work by the United States, Canada, western Europe, and most of the former Soviet bloc states, the LRTAP Executive Body agreed in November 1995 that its Working Group on Strategies should begin work on legally binding protocols on POPs and heavy metals in early 1996. Both protocols were concluded in spring 1998 and signed by most LRTAP parties in June 1998. The POPs protocol (Economic Commission for Europe, 1994) addresses 16 substances (the UNEP short list, plus chlordecone, hexabromobiphenyl, HCH, and PAHs), which have in large measure already been addressed by the United States, Canada, and Western Europe. The protocol allows flexibility in dealing with specific POPs; for example, certain pesticides such as DDT would be subjected to use restrictions and gradually phased out while others would be banned outright and at a much earlier date. For stationary sources (e.g., sources of dioxin and furan emissions), best available technology or best environmental practice requirements would be applied.

Among other things, the protocol also provides differentiated control action timelines for western countries and former Soviet bloc countries, establishes a variety of reporting and monitoring efforts, and sets forth a mechanism for selecting other POPs for joint action. To promote widespread compliance with this protocol, the United States has initiated, and won significant political and financial support from the other arctic countries for, a PCB identification and phase-out project in the Russian Federation. The heavy metals protocol focuses on lead, mercury, and cadmium and establishes a substance selection mechanism, monitoring and reporting requirements, and a variety of legally binding and voluntary provisions for reducing the release of and exposure to these heavy metals.

5.3.4 United Nations Environment Program (UNEP), and Intergovernmental Forum on Chemical Safety (IFCS)

[EPA leads: OPPTS, OIA] The May 1995 UNEP Governing Council Decision on POPs (GC.18/32) invited the Inter-Organizational Program for Sound Chemical Management working with the International Program on Chemical Safety and the IFCS, to initiate a process to assess the chemistry, toxicology, transport, and socioeconomic factors associated with the UNEP short list of POPs (PCBs, dioxins and furans, aldrin, dieldrin, DDT, endrin, chlordane, hexachlorobenzene, mirex, toxaphene, and heptachlor). Based on the results of this assessment, the decision invited the IFCS to develop recommendations for international actions on POPs, including an appropriate international legal mechanism. This assessment report was the basis for the decision of the 19th session of the UNEP Governing Council to mandate the development of a legally binding global instrument aimed at the reduction and/or elimination of emission and discharges and, where appropriate, the manufacture and use of these pollutants. The first negotiating session was held in June-July 1998, the second session was held in late January 1999, and the negotiations are to be concluded before the end of calendar year 2000. As with the LRTAP POPs protocol, the UNEP convention will establish a process by which additional substances can be selected for joint action. Compared to the LRTAP protocol, the UNEP convention will greatly emphasize the capacity building needs of developing countries.

5.3.5 Arctic Council

[EPA lead: OIA] The Arctic Council is a ministerial-level body that brings together the eight countries with arctic territory (United States, Canada, Denmark, Norway, Sweden, Finland, Russian Federation) to cooperate voluntarily to protect human health and the environment in the Arctic. Although it was established only in 1997-98, the Council subsumes and continues all of the work initiated in 1991 by these same countries under the auspices of the Arctic Environmental Protection Strategy. Of particular interest is the priority work of the Arctic Monitoring and Assessment Program (AMAP) on POPs and heavy metals, which is not surprising given the proclivity for these substances to migrate to and settle into the Arctic. The AMAP work offers the United States an additional opportunity to develop and exchange information on the transport and fate of persistent toxics, especially with respect to sensitive human subpopulations and ecological areas. The voluntary nature of the Council's work dictates that risk management measures on POPs and heavy metals must be undertaken voluntarily by the arctic countries or prompted by legally binding international agreements to which the eight countries, and others farther to the south, are already a party (e.g., the LRTAP POPs and heavy metals protocols).

At the North Sea Conferences, ministers representing countries bordering the North Sea agreed to the reduction and elimination of hazardous chemical contaminants, as stated in the Esbjerg Declaration, June 1995. The Helsinki Convention on the Protection of the Marine Environment of the Baltic Sea Area (HELCOM) is developing activities pertaining to concerns about DDT, PCBs, PCTs, mercury, cadmium, organotin compounds, and pesticides. A ministerial meeting was held in 1998 to decide on the

program of action. The Paris Commission-OSPAR (Oslo-Paris Convention for the Prevention of Marine Pollution from Land-based Sources to the North-East Atlantic) (PARCOM 1974 and 1992) is working to adopt the targets of the Esbjerg Declaration in 1997 and examining PAHs, chlorinated paraffins, nonylphenols and related substances, suspected endocrine disruptors, and mercury, cadmium, and lead. The European Union has issued several directives on the use, marketing, discharge, and management of persistent organic pollutants, in particular, nonylphenol/ethoxylate, phthalates, organotin compounds, polybrominated diphenylethers/biphenyls, and short-chain chlorinated paraffins. The United States and the European Union will be working together to examine a number of complex scientific and political issues related to their own and other international efforts to better assess and manage the known and suspected risks associated with persistent toxic substances.

5.4 Summary of Regional and Headquarters Activities

This review has identified a variety of activities under way in EPA that are important for interpreting bioaccumulation data in relation to sediment quality. Most of the programs represent individual and different approaches that address specific statutory mandates as well as the goals of EPA's Contaminated Sediment Management Strategy. Following are the highlights of the bioaccumulation-related activities that are ongoing in the EPA.

Research on bioaccumulation and the use of bioaccumulation to assess sediment contamination are ongoing activities in OPPT, NHEERL, NERL, OERR, OSW, OST, OWOW, the Chesapeake Bay Program, and all EPA regions. OPPT, OSW, OST, and OERR have been working on identifying, based on various chemical properties, chemical structure, and structure-activity relationships, which chemicals might bioaccumulate and cause adverse environmental effects. Intra-Agency agreement on which chemicals should be considered to be bioaccumulative and how risks to humans and ecosystems should be assessed is still developing, and consensus is building through various workgroups and activities. NHEERL has conducted numerous studies of bioaccumulation in freshwater and marine biota, examining conditions and factors affecting bioaccumulation, determination of BAFs and BSAFs in numerous species, trophic transfer, and metabolism of bioaccumulative chemical contaminants, and developing and validating toxicity and bioaccumulation test procedures and sediment-based ecological risk assessment methods. NERL has used data obtained from the field and laboratory studies to model bioaccumulation processes to examine risks to higher tropic levels. Bioaccumulation of sediment contaminants in fish that might be consumed by humans is a major concern in the development of water quality criteria for the protection of human health, and research is under way on the most appropriate methods for predicting bioaccumulation in fish (Region 5) and developing chemical-specific BSAFs for setting tissue residue-linked sediment chemical levels for the protection of human health, particularly for mercury (Region 10). The Chesapeake Bay Program, in concert with NOAA, is developing a 5-year plan for a toxics research program that will include work on bioaccumulation and food webs.

Assessment procedures are of concern to or used by OERR, OPP, OPPT, OAR, ORD, OST, OSW, OWOW, GLNPO, the Chesapeake Bay Program, and all EPA regions. OERR is working on issues such as improving consistency in methods used to determine potential bioaccumulative chemicals at Superfund sites, effects concentrations for these chemicals, and use of background data to assess bioaccumulation. OPP uses bioconcentration and bioaccumulation tests to evaluate whether and under what conditions pesticides and other chemical products pose risks to aquatic and terrestrial biota, and OPPT can require manufacturers to conduct such tests on compounds that might pose risks based on chemical characteristics, suspected toxicities, and loadings information. OAR incorporates information on bioaccumulation of air toxics in certain analyses (e.g., ranking chemicals for the draft Urban Air Toxics Strategy). OST is developing methods for quickly screening effluent discharges for

bioaccumulative contaminants. OST has used bioaccumulation modeling procedures to assess potential risks to human health in support of industrial effluent discharge limitation guidelines and to help identify sites around the nation having potentially bioaccumulative contaminants in sediments that might present risks to aquatic and terrestrial biota and humans. The Office of Water is conducting a National Survey of Chemical Residues in Lake Fish Tissue. This study will meet objectives presented in the President's Clean Water Action Plan and provide information about persistent, bioaccumulative, toxic chemicals (PBTs) for EPA's PBT Strategy. OPP uses a 28-day test of channel catfish or bluegill to assess bioaccumulation, whereas ORD assesses sediment bioacccumulation with invertebrates (such as *Hyalella*, *Chironomus*, and *Lumbriculus*) and freshwater and marine fish species. OSW is developing a rule establishing chemical-specific waste stream concentrations that represent a threshold below which Subtitle C disposal will not be required and thus the waste stream may exit the hazardous waste system. Under development (with ORD) is a risk-based methodology that will be used as the basis for the waste stream concentrations. OWOW developed guidance for conducting bioaccumulation tests required under section 403 of the CWA. GLNPO and the regions are using bioaccumulation tests and fish tissue concentrations of bioaccumulative contaminants to assess the extent and magnitude of sediment contamination and evaluate ecological and human health risks. In addition, they are evaluating BAF and BSAF models, the incorporation of site-specific data, and new techniques such as semipermeable membrane devices. The Chesapeake Bay Program is conducting a toxics characterization to identify chemical contamination problems in the tidal tributaries of the Chesapeake Bay. This characterization is based, in part, on an analysis and integration of water, sediment, and fish tissue contaminant concentration data. A variety of assessment procedures are thus being developed, applied at various sites, and used under various statutes to reduce or eliminate exposures to bioaccumulative contaminants.

OERR, OSW, GLNPO, and a number of international organizations are focusing on the use of bioaccumulation information to identify and evaluate sediments for remediation efforts. Clean-up levels at several Superfund sites have been derived based on the presence of bioaccumulative contaminants. Subtitle C of RCRA provides EPA (OSW) with the authority to assess whether releases from a hazardous waste treatment, storage, or disposal facility have contaminated sediments and to require corrective action, including possible remediation, if contamination is discovered. The regions are using different approaches to target remediation of contaminated sediments; many are evaluating the fish ingestion pathway and various factors influencing clean-up decisions. Region 2 employs bioaccumulation tests with fishes and other organisms (small mammals, clams, amphibians) during the evaluation of ecological risk. GLNPO operated the ARCS Program to identify AOCs and to help select appropriate remedial actions in harbors and tributaries of the Great Lakes. Western Europe, Canada, and the North Sea countries have begun talks and developed agreements that pertain to the remediation of sediment-associated bioaccumulative contaminants.

Preventing the release of bioaccumulative contaminants is of concern to OPPTS, OSW, OST, OWM, OWOW, the Chesapeake Bay Program, and numerous countries around the world. OPPT is using its screening and prioritizing procedures to determine those chemicals that might accumulate in sediments and pose risks so that dangerous levels of loadings can be targeted. Under the Waste Minimization National Plan, OSW and OPPT are developing a tool that would prioritize chemicals in hazardous waste based on persistence, biaoaccumulation potential, toxicity, and quantity. OWOW and OST use bioaccumulation data in the control of discharges of bioaccumulative chemicals under the CWA Several international conferences have been held to examine methods for reducing or eliminating persistent organic and metal pollutants, and some have resulted in agreements.

The dissemination of information pertaining to the bioaccumulation of chemicals has been important for OPPT, OST, OWOW, the Chesapeake Bay Program, NHEERL, and all EPA regions. OPPT recently

released a draft multimedia strategy to reduce risks from exposures to priority persistent, bioaccumulative, and toxic pollutants through increased coordination among EPA national and regional programs. This approach also requires the significant involvement of stakeholders, including international, state, local, and tribal organizations, the regulated community, environmental groups, and private citizens. OST has published a series of documents on the analysis of fish tissue concentrations of contaminants and the use of such data to determine risks to humans from consumption of fish, as well as providing a database listing all fish and wildlife consumption advisories currently operating in the states. OWOW's outreach efforts include educating the public about the ocean and coastal programs and making program information readily available to the public. The toxics characterization of tidal tributaries of the Chesapeake Bay being performed by the Chesapeake Bay Program will be used as an outreach tool to put information in the hands of the public, scientists, and managers to target appropriate areas for additional monitoring and management activities. NHEERL has prepared guidance documents pertaining to bioaccumulation methods and has developed several databases to assist in the interpretation of bioaccumulation data. Region 5 developed guidance for determining BAFs that are used in the evaluation of risks to wildlife and humans from bioaccumulative chemicals under the Great Lakes Water Quality Initiative.

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6. ISSUES AND RESEARCH NEEDS FOR INTERPRETING BIOACCUMULATION DATA FOR THE PURPOSE OF SEDIMENT QUALITY ASSESSMENT

This chapter contains issues touched on previously, as well as research needs, for interpreting bioaccumulation data for the purpose of assessing sediment quality. The interpretation of bioaccumulation data will require an understanding of the relationship between environmental media concentrations, tissue concentrations, and effects on specific organisms or consumers of those organisms. The role of physical, chemical, and biological variables in mediating toxic effects should also be considered. "Research needs" were taken from the proceedings of EPA's National Sediment Bioaccumulation Conference, held September 11-13, 1996 (USEPA, 1998), and input from EPA's Bioaccumulation Analysis Workgroup.

It is anticipated that the research needs presented below will be used as a starting point to identify future work. Cost and feasibility considerations were not factored into the development of these research needs. Due to differences in mandates among EPA offices, the issues and needs presented here might not be applicable to all EPA programs.

Results of multivoting on the research needs indicate that 11 are of high priority (denoted by **H**), 12 are of intermediate priority (denoted by **I**), and 7 are of low priority (denoted by **L**).

Methods

Issue: Laboratory and field methods for assessing bioaccumulation (Sections 3.1, 3.2)

Needs:

H 1. Conduct round-robin tests to determine the precision and variability of new and existing methods and how well laboratories are conducting bioaccumulation tests

H 2. Identify additional species for sediment bioaccumulation test methods, and develop the methods

H 3. Conduct research to provide better understanding of how reference sites can be selected.

I 4. Standardize the use of microlipid analytical methods

I 5. Continue to field-validate laboratory bioaccumulation methods, particularly for PAHs and metals

L 6. Conduct further research on the development and use of formulated sediments and sediment spiking techniques

L 7. Standardize the sampling and handling procedures for assessing bioaccumulation (field-collected samples)

L 8. Determine most appropriate means of storing sediment samples

Chemical Identification

Issue: Identification of bioaccumulative chemicals (Chapter 4)

Needs:

H 9. Determine additional BAF and BSAF values for known persistent bioaccumulators or identify potential persistent bioaccumulators

H 10. Reach a better understanding of bioavailability processes of chemicals occurring in sediments

I 11. Develop a forensic chemical approach to assist in identification of bioaccumulative compounds

Species Considerations

Issue: Species selection for bioaccumulation testing (Sections 3.2.1.1, 3.2.2.2, 4.4)

Needs:

H 12. Determine tissue residue values for terrestrial and avian wildlife

I 13. Provide guidance on further identification or development of contaminant-specific receptors of choice, where relevant, so that appropriate target species can be selected

Toxicology

Issue: Dose-response relationships for bioaccumulative contaminants (Section 4.4, Chapter 3)

Needs:

H 14. Determine the relationship of tissue residue concentrations and adverse effects

H 15. Determine the applicability of the critical body residue (CBR) approach to multiple classes of chemical mixtures

I 16. Determine the additivity of chemicals for specific modes of action

I 17. Determine differential partitioning of bioaccumulative contaminants among tissues

I 18. Develop the appropriate guidance for selecting species/effects endpoints that should be used

I 19. Confirm exposure route independence (e.g. dietary, sediment)

L 20. Provide procedures to evaluate experimental data that are suitable for use in generating CBRs

Further research is needed on how to resolve or compensate for uncertainties related to the assessment of bioaccumulation of sediment-associated contaminants. Specific areas of research required to address uncertainty in bioacccumulation testing include the following:

I 21. Environmental factors that affect toxic responses

I 22. Rates at which contaminants are metabolized or eliminated

L 23. The modes and time course of toxic action

L 24. The toxicities of intermediate metabolites relative to parent compounds

L 25. Dose-related induction of enzymatic systems

Models

Issue: Development, selection, application, and interpretation of models (Sections 2.4.2, 3.2.1.3, 3.3, 4.4)

Needs:

H 26. Continue work on developing food chain multipliers and trophic models. Further refine the relationship of BAFs to BCFs (Predicted BAF may be derived by multiplying the laboratory BCF by a food chain multiplier)

H 27. Develop extrapolation models that will take us from single-species endpoints to more ecologically relevant endpoints that involve community and population-level impacts

H 28. Determine how the toxicity risks of complex mixtures of PBTs, bioaccumulated by organisms with no direct exposure to sediments, can be related to sediment contamination levels

I 29. Develop a better understanding of physiologically based models or kinetic models. When are time-dependent exposure models preferable to steady-state BSAFs?

I 30. Determine whether the BAF methodology applied to the Great Lakes region can be applied to Toxic Release Inventory (TRI) PBT chemicals that may be manufactured and released at sites in several states, in one or more regions, or even throughout the United States.

Reference

USEPA. 1998. *National Sediment Bioaccumulation Conference proceedings.* U.S. Environmental Protection Agency, Office of Water, Washington, DC.

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

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Chemical	Page
Acenaphthene	1
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1,2,3,7,8-PentaCDF	412
2,3,4,7,8-PentaCDF	421
2,3,7,8-TCDF	431
Fluoranthene	443
Heptachlor	455
Lead	465
Methylmercury	485
Nickel	525
Oxyfluorfen	533
PCB 28	536
PCB 77	547
PCB 81	561
PCB 105	571
PCB 118	585
PCB 126	599
PCB 156	609
PCB 169	621
Pentachlorophenol	631
Phenanthrene	649
Pyrene	659
Selenium	667
Silver	685
Tributyltin	693
Terbufos	745
Total PCBs	751
Toxaphene	787
Zinc	801

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 = $\frac{[\text{Organism}]_{(\text{ng/g lipid})}}{[\text{Sediment}]_{(\text{ng/g organic carbon})}}$
	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)				0.96	[43]	BSAFs are lipid and TOC normalized values reported in text.
			Liver = 12.0* µg/kg, (24.8% lipids)				1.4		
			**median concentration*						
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: 3×10^{-4} mg/kg/day [2] **Confidence:** Medium, uncertainty factor = 3

Critical Effect: Hyperpigmentation, keratosis, and possible vascular complications

Oral Slope Factor: $1.5 \times 10^{+0}$ 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 x 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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Chemical Category: POLYNUCLEAR AROMATIC HYDROCARBON (high molecular weight)

Chemical Name (Common Synonyms): BENZO(B)FLUORANTHENE **CASRN:** 205-99-2

Chemical Characteristics

Solubility in Water: 0.0012 mg/L [1]

Half-Life: 360 days - 1.67 yrs based on aerobic soil die-away test data [2]

Log K_{ow}: 6.20 [3]

Log K_{oc}: 6.09 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(b)fluoranthene in wildlife were not found in the literature.

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

Aquatic Organisms

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

Food Chain Multipliers: Food chain multipliers for benzo(b)fluoranthene in aquatic organisms were not found in the literature.

Toxicity/Bioaccumulation Assessment Profile

The acute toxicity of hydrocarbons, including benzo(b)fluoranthene, 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].

Bioavailability of sediment-associated polynuclear aromatic hydrocarbons (PAHs), e.g., benzo(b)fluoranthene, has been observed to decline with increased contact time [6]. 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. The apparent effects threshold concentration of 4,500 ng/g was established for benzo(b)fluoranthene based on effects observed in the marine amphipod *Rhepoxynius abronius* [7].

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(b)fluoranthene [8]. Meador et al. [8] 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(b)fluoranthene

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	18 ng/g		18 ng/g					[9]	F
	2.9 ng/g		27 ng/g					[9]	F
	9.9 ng/g		40 ng/g					[9]	F
Diporeia spp, Amphipod	27 nmol/g		321 nmol/g					[6]	L

[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: POLYNUCLEAR AROMATIC HYDROCARBON (high molecular weight)

Chemical Name (Common Synonyms): BENZO(G,H,I)PERYLENE **CASRN:** 191-24-2

Chemical Characteristics

Solubility in Water: Insoluble in water [1]

Half-Life: 590 d - 650 days based on aerobic soil die-away test data at 30°. [2]

Log K_{ow}: 6.70 [3]

Log K_{oc}: 6.59 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(g,h,i)perylene in wildlife were not found in the literature.

Food Chain Multipliers: Food chain multipliers for benzo(g,h,i)perylene in wildlife were not found in the literature.

Aquatic Organisms

Partitioning Factors: Partitioning factors for benzo(g,h,i)perylene in aquatic organisms were not found in the literature.

Food Chain Multipliers: An ecotoxicological in situ study conducted at the Baltic Sea, showed that the tissue residue concentration of benzo(g,h,i)perylene decreased with increasing trophic level [5]. The relatively high theoretical flux through the food chain was not possible to detect.

Toxicity/Bioaccumulation Assessment Profile

The acute toxicity of hydrocarbons, including benzo(g,h,i)perylene, 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].

The majority of investigations have shown that aquatic organisms are able to release polynuclear aromatic hydrocarbons (PAHs), e.g., benzo(g,h,i)perylene, from their tissues rapidly when they were returned to clean environment. Tanacredl and Cardenas [6] reported that *Mercenaria mercenaria* exposed to PAHs accumulated them to high levels in their tissues and failed to release them when returned to clean seawater over the 45-day depuration period. Unlike other marine organisms, this "sequestering" in molluscs may support the apparent inability to metaboilize PAHs to more water soluble and thus easily secreted polar metabolites.

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(g,h,i)perylene [7]. Meador et al. [7] 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(g,h,i)perylene

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									
Mytilus edulis, Mussels			13 ng/g					[8]	F
Crassostrea virginica, Oyster	0.4 ng/g		10 ng/g					[10]	
	122.1 ng/g		16 ng/g					[10]	F
	31.1 ng/g		27 ng/g					[10]	F
	75.1 ng/g		12 ng/g					[10]	F
	5.4 ng/g		14 ng/g					[10]	F
	5.7 ng/g		18 ng/g					[10]	F
	6.2 ng/g		10 ng/g					[10]	F
	6.7 ng/g		10 ng/g					[10]	F
	0.4 ng/g		10 ng/g					[10]	F
	16.1 ng/g		16 ng/g					[10]	F
Pontoporeia hoyi, Amphipod	400 ng/g	5 ng/mL	BDL					[9]	L
Fishes									
Cyprinus carpio, Common carp			29.6 mg/kg (liver)[4]	Physiological, NOED				[11]	L; no significant increase in EROD enzyme and P450 1a protein content

Summary of Biological Effects Tissue Concentrations for Benzo(g,h,i)perylene

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]**
Wildlife									
Somateria mollissima, Eider duck			2 ng/g					[8]	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(K)FLUORANTHENE **CASRN:** 207-08-9

Chemical Characteristics

Solubility in Water: Insoluble in water [1]

Half-Life: 2.49 yrs - 5.86 yrs based on aerobic soil die-away test data [2]

Log K_{ow}: 6.20 [3]

Log K_{oc}: 6.09 L/kg organic carbon

Human Health

Oral RfD: No data [4]

Confidence: —

Critical Effect: —

Oral Slope Factor (Reference): Not available [4]

Carcinogenic Classification: B2 [4]

Wildlife

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

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

Aquatic Organisms

Partitioning Factors: The only partitioning factors for benzo(k)fluoranthene in aquatic organisms found in the literature were log BAFs of -0.68 to 0.01 for the clam *Macoma nasuta* [9].

Food Chain Multipliers: An ecotoxicological in situ study conducted at the Baltic Sea [5] showed that the tissue residue concentration of benzo(k)fluoranthene decreased with increasing trophic level. The relatively high theoretical flux through the food chain was not possible to detect.

Toxicity/Bioaccumulation Assessment Profile

The acute toxicity of hydrocarbons, including benzo(k)fluoranthene, 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 [6].

The majority of investigations have shown that aquatic organisms are able to release polynuclear aromatic hydrocarbons (PAHs), e.g., benzo(k)fluoranthene, from their tissues rapidly when they were returned to clean environment. The apparent effects threshold concentration of 4500 ng/g for benzo(k)fluoranthene was established based on effects observed in the marine amphipod *Rhepoxynius abronius* [7].

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(k)fluoranthene [8]. Meador et al. [8] 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(k)fluoranthene

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									
Mytilus edulis, Blue mussel			44 ng/g					[5]	F
Crassostrea virginica, Eastern oyster	1.5 ng/g		14 ng/g					[10]	F
	36 ng/g		85 ng/g					[10]	F
	59.6 ng/g		65 ng/g					[10]	F
	127.5 ng/g		61 ng/g						
Macoma nasuta, Clam	14.1 ng/g		92 ng/g			0.009 or 0.01		[9]	F
	17 ng/g		24 ng/g			-0.66		[9]	F
	121 ng/g		59 ng/g			-0.48		[9]	F
	156 ng/g		87 ng/g			-0.39		[9]	F
	390 ng/g		128 ng/g			-0.51		[9]	F
	610 ng/g		96 ng/g			-0.68		[9]	F
Wildlife									
Somateria mollissima, Eider duck			4.3 ng/g					[5]	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: METAL (Divalent)

Chemical Name (Common Synonyms): CADMIUM **CASRN:** 7440-43-9

Chemical Characteristics

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

Log K_{ow}: – **Log K_{oc}:** –

Human Health

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

Critical Effect: Significant proteinuria, presence of protein in urine

Oral Slope Factor: Not available [2] **Carcinogenic Classification:** B1 [2]

Wildlife

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

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

Aquatic Organisms

Partitioning Factors: Cadmium in the water column can partition to dissolved and particulate organic carbon. The more important issues related to water column concentrations of cadmium are water hardness (i.e., calcium concentration), pH, and metal speciation since the divalent cadmium ion is believed to be responsible for observed biological effects. Cadmium speciation yields primarily the divalent form of the metal, Cd^{+2}, between pH values of 4.0 and 7.0 [3]. In addition, the concentration of acid-volatile sulfides is known to be an important factor controlling the toxicity and bioaccumulation of cadmium in sediments.

Food Chain Multipliers: Most studies reviewed contained data which suggest that cadmium is not a highly mobile element in aquatic food webs, and there appears to be little evidence to support the general occurrence of biomagnification of cadmium within marine or freshwater food webs [4,5,6,7].

Toxicity/Bioaccumulation Assessment Profile

Cadmium does not appear to be a highly mobile element under typical conditions in most aquatic habitats [4]. Additional studies reviewed by Kay [4] indicated that no maternal transfer of cadmium was observed in zebrafish and that the cadmium content of bird eggs did not appear to be a good indicator of environmental exposure to cadmium. Tissue residue-toxicity relationships can also be variable because organisms might sequester metal in various forms that can be analytically measurable as tissue residue but might actually be stored in unavailable forms within the organism as a form of detoxification [8]. Whole body residues might also not be indicative of effects concentrations at the organ level because concentrations in target organs, such as the kidneys and liver, may be 20 times higher than whole body residues [9]. The application of "clean" chemical analytical and sample preparation techniques is also critical in the measurement of metal tissue residues. After evaluating the effects of sample preparation techniques on measured concentrations of metals in the edible tissue of fish, Schmitt and Finger [10] concluded that there was little direct value in measuring copper, zinc, iron, or manganese tissue residues in fish because they do not bioaccumulate to any appreciable extent. Cadmium and lead were the only ones found to be of potential concern in edible fish tissue based on the results from Schmitt and Finger's study of "clean" chemical techniques, although Wiener and Stokes [11] suggested that cadmium did not generally accumulate to any appreciable extent in the edible muscle tissue of fish.

Rule and Alden [26] studied the relationship between uptake of cadmium and copper from the sediment by the blue mussel (*Mytilus edulis*), grass shrimp (*Palaemonetes pugio*), and hard clam (*Mercenaria mercenaria*). The uptake of cadmium by the blue mussel significantly increased as a function of increasing cadmium concentration in sediment. However, the uptake of cadmium increased when copper was added to the sediments. The uptake of cadmium by the grass shrimp exhibited a pattern similar to that of the mussel, while the uptake of cadmium by the hard clam was low compared to the other two species and related only to the cadmium concentration in sediment.

The experiments performed by Meador [28] revealed that the response of the amphipods *Rhepoxynius abronius* and *Eohaustorius estuarius* to cadium decreased two- to threefold for animals held in the laboratory for several weeks compared to organisms recently collected from the field.

Summary of Biological Effects Tissue Concentrations for Cadmium

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]**
Plants									
Scenedesmus obliquus, Freshwater colonial green algae			2,340 mg/kg (whole body)[5]	Growth, LOED				[31]	L; significant inhibition of growth (27% reduction from control)
			658 mg/kg (whole body)[5]	Growth, LOED				[31]	L; 39% reduction in population growth from controls
			3,030 mg/kg (whole body)[5]	Growth, NOED				[31]	L; no significant inhibition of growth
Eichhornia crassipes, Water hyacinth			11.4 mg/kg (leaf)[5]	Growth, LOED				[47]	F; reduced growth rate, chlorosis
			262 mg/kg (root)[5]	Growth, LOED				[47]	F; reduced growth rate, chlorosis
			49.6 mg/kg (stem)[5]	Growth, LOED				[47]	F; reduced growth rate, chlorosis

Summary of Biological Effects Tissue Concentrations for Cadmium

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]
			11.4 mg/kg (leaf)[5]	Morphology, LOED				[47]	F; chlorosis, browning, necrosis, waterlogging of tissues
			262 mg/kg (root)[5]	Morphology, LOED				[47]	F; chlorosis, browning, necrosis, waterlogging of tissues
			49.6 mg/kg (stem)[5]	Morphology, LOED				[47]	F; chlorosis, browning, necrosis, waterlogging of tissues
			20.8 mg/kg (leaf)[5]	Growth, NA				[47]	F; reduced growth rate, chlorosis
			45.8 mg/kg (leaf)[5]	Growth, NA				[47]	F; reduced growth rate, chlorosis
			578 mg/kg (root)[5]	Growth, NA				[47]	F; reduced growth rate, chlorosis
			1,300 mg/kg (root)[5]	Growth, NA				[47]	F; reduced growth rate, chlorosis

Summary of Biological Effects Tissue Concentrations for Cadmium

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]
			84.8 mg/kg (stem)[5]	Growth, NA				[47]	F; reduced growth rate, chlorosis
			159 mg/kg (stem)[5]	Growth, NA				[47]	F; reduced growth rate, chlorosis
			20.8 mg/kg (leaf)[5]	Morphology, NA				[47]	F; chlorosis, browning, necrosis, waterlogging of tissues
			45.8 mg/kg (leaf)[5]	Morphology, NA				[47]	F; chlorosis, browning, necrosis, waterlogging of tissues
			578 mg/kg (root)[5]	Morphology, NA				[47]	F; chlorosis, browning, necrosis, waterlogging of tissues
			1,300 mg/kg (root)[5]	Morphology, NA				[47]	F; chlorosis, browning, necrosis, waterlogging of tissues

Summary of Biological Effects Tissue Concentrations for Cadmium

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]
			84.8 mg/kg (stem)[5]	Morphology, NA				[47]	F; chlorosis, browning, necrosis, waterlogging of tissues
			159 mg/kg (stem)[5]	Morphology, NA				[47]	F; chlorosis, browning, necrosis, waterlogging of tissues
			8 mg/kg (leaf)[5]	Growth, NOED				[47]	F; no effect on growth
			142 mg/kg (root)[5]	Growth, NOED				[47]	F; no effect on growth
			27.8 mg/kg (stem)[5]	Growth, NOED				[47]	F; no effect on growth
			8 mg/kg (leaf)[5]	Morphology, NOED				[47]	F; no effect on plant appearance
			142 mg/kg (root)[5]	Morphology, NOED				[47]	F; no effect on plant appearance
			27.8 mg/kg (stem)[5]	Morphology, NOED				[47]	F; no effect on plant appearance

Summary of Biological Effects Tissue Concentrations for Cadmium

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									
Lumbriculus variegatus, Oligochaete			670 mg/kg (whole body)[5]	Mortality, LOED				[32]	L; 40% mortality
			310 mg/kg (whole body)[5]	Mortality, NOED				[32]	L; no effect on mortality
Najas quadulepensis, Southern naiad			10.3 mg/kg (whole body)[5]	Development, LOED				[35]	L; reductions in chlorophyll and stolon development
Neanthes arenaceodentata, Polychaete			67 mg/kg (whole body)[5]	Reproduction, ED100				[46]	L; reproductive failure
			67 mg/kg (whole body)[5]	Behavior, LOED				[46]	L; reduced tube building, sluggish behavior
			4.5 mg/kg (whole body)[5]	Behavior, NOED				[46]	L; no effect on behavior
			0.22 mg/kg (whole body)[5]	Behavior, NOED				[46]	L; no effect on behavior
			0.028 mg/kg (whole body)[5]	Behavior, NOED				[46]	L; no effect on behavior

Summary of Biological Effects Tissue Concentrations for Cadmium

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]
			0.0028 mg/kg (whole body)[5]	Behavior, NOED				[46]	L; no effect on behavior
			67 mg/kg (whole body[5]	Mortality, NOED				[46]	L; no effect on survival
			4.5 mg/kg (whole body)[5]	Mortality, NOED				[46]	L; no effect on survival
			0.22 mg/kg (whole body)[5]	Mortality, NOED				[46]	L; no effect on survival
			0.028 mg/kg (whole body)[5]	Mortality, NOED				[46]	L; no effect on survival
			0.0028 mg/kg (whole body)[5]	Mortality, NOED				[46]	L; no effect on survival
			4.5 mg/kg (whole body)[5]	Reproduction, NOED				[46]	L; no effect on reproduction
			0.22 mg/kg (whole body)[5]	Reproduction, NOED				[46]	L; no effect on reproduction
			0.028 mg/kg (whole body)[5]	Reproduction, NOED				[46]	L; no effect on reproduction
			0.0028 mg/kg (whole body)[5]	Reproduction, NOED				[46]	L; no effect on reproduction

Summary of Biological Effects Tissue Concentrations for Cadmium

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]**
Neanthes virens, Polychaete - Sandworm			106 mg/kg (whole body)[5]	Behavior, LOED				[33]	L; lethargy
			78 mg/kg (whole body)[5]	Physiological, LOED				[33]	L; total glycogen reduced, increase in ascorbic acid
			290 mg/kg (whole body)[5]	Physiological, LOED				[33]	L; increase in ascorbic acid content
Helisoma sp., Snail			625 mg/kg (whole body)[5]	Mortality, ED50				[32]	L; 50% mortality
			300 mg/kg (whole body)[5]	Mortality, NOED				[32]	L; no effect on mortality
			460 mg/kg (whole body)[5]	Mortality, NOED				[32]	L; no effect on mortality
Dreissena polymorpha, Zebra mussel			Day 27: 539-598 µg/g 0.96-1.06 mmol/kg	50% mortality				[19]	L
Mytilus edulis, Blue mussel			30 mg/kg (whole body)[5]	Growth, NOED				[53]	L

Summary of Biological Effects Tissue Concentrations for Cadmium

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]**
			30 mg/kg (whole body)[5]	Mortality, NOED				[53]	L; highest body burden reported
			6.45 mg/kg (whole body)[5]	Mortality, NOED				[26]	L; estimated wet weight
			4.22 mg/kg (whole body)[5]	Mortality, NA				[60]	L; decreased anoxic survival time (Control 10.7 days)
			8.06 mg/kg (whole body)[5]	Mortality, NA				[60]	L; decreased anoxic survival time (Control 10.7 days)
			3.74 mg/kg (whole body)[5]	Mortality, NA				[60]	L; decreased anoxic survival time (Control 13 days)
			8.06 mg/kg (whole body)[5]	Mortality, NA				[60]	L; decreased anoxic survival time (Control 10.7 days)
			8.06 mg/kg (whole body)[5]	Physiological, NOED				[60]	L; no significant changes in adenylate energy charge or glycogen content

Summary of Biological Effects Tissue Concentrations for Cadmium

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]**
Mytilus galloprovincialis, Mussel			0.57-0.92 mg/kg				0.416	[27]	F
Crassostrea virginica, Oyster			18.2 mg/kg (whole body)[5]	Reproduction, NOED				[62]	L; no reduced viability of gametes after exposure of adults in 21 ppt seawater
			54 mg/kg (whole body)[5]	Reproduction, NOED				[62]	L; 24% reduction in viability of gametes after exposure of adults in 21 ppt seawater
Daphnia magna, Cladoceran			Day 21: 2.36 µg/g	LOEC				[20]	F
			Week 20: 17.4 µg/g	LOEC				[17]	L
			Day 21: 2.0 mmol/kg	10% mortality				[21]	L

Summary of Biological Effects Tissue Concentrations for Cadmium

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			1.7 mg/kg (whole body)[5]	Reproduction, ED10				[21]	L; 10% reduction in number of offspring
			221 mg/kg (whole body)[5]	Mortality, ED50				[21]	L; lethal body burden after 21-day exposure
Daphnia galeata mendotae, Cladoceran			10.3 mg/kg (whole body)[5]	Growth, LOED				[48]	L; increased weight of individual animals
			3.5 mg/kg (whole body)	Mortality, LOED				[48]	L; reduced longevity, increased prenatal mortality
			5.7 mg/kg (whole body)[5]	Mortality, NA				[48]	L; reduced longevity, increased prenatal mortality
			8.6 mg/kg (whole body)[5]	Mortality, NA				[48]	L; reduced longevity, increased prenatal mortality

Summary of Biological Effects Tissue Concentrations for Cadmium

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]**
			10.3 mg/kg (whole body)[5]	Mortality, NA				[48]	L; reduced longevity, increased prenatal mortality
			3.5 mg/kg (whole body)[5]	Growth, NOED				[48]	L; no effect on individual weight
			5.7 mg/kg (whole body)[5]	Growth, NOED				[48]	L; no effect on individual weight
			8.6 mg/kg (whole body)[5]	Growth, NOED				[48]	L; no effect on individual weight
Folsomia candida, Cladoceran			60 µg/g	LOEC				[22]	F
Gammarus fossarum, Amphipod			Day 14: 60-70 µg/g	50% mortality				[18]	L
Moina macrocopa, Cladoceran			16.4 mg/kg (whole body)[5]	Reproduction, ED100				[42]	L; no reproduction after 12 days
			16.4 mg/kg (whole body)[5]	Growth, LOED				[42]	L; reduced growth

Summary of Biological Effects Tissue Concentrations for Cadmium

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]**
			16.4 mg/kg (whole body)[5]	Mortality, LOED				[42]	L; reduced survival
			10.6 mg/kg (whole body)[5]	Reproduction, LOED				[42]	L; reduced brood size
			10.6 mg/kg (whole body)[5]	Mortality, NOED				[42]	L; no effect on survival
			8 mg/kg (whole body)[5]	Reproduction, NOED				[42]	L; no effect on brood size
Hyallela azteca, Amphipod			Week 6: 15.2 µg/g	LOAEC				[17]	L
Pontoporeia affinis, Amphipod (juveniles, 105-460 d)			Day 460: 80-90 µg/g (0.14 mmol/kg)	LOEC				[16]	L
Pontoporeia affinis, Amphipod			11 mg/kg (whole body)[5]	Mortality, LOED				[58]	L
			6 mg/kg (whole body)[5]	Reproduction, LOED				[58]	L; percent malformed eggs
			6 mg/kg (whole body)[5]	Mortality, NOED				[58]	L

Summary of Biological Effects Tissue Concentrations for Cadmium

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]**
			3 mg/kg (whole body)[5]	Reproduction, NOED				[58]	L; Percent malformed eggs
			2 mg/kg (whole body)[5]	Mortality, NOED				[59]	L; body burden estimated from graph
			10 mg/kg (whole body)[5]	Mortality, NOED				[59]	L; body burden estimated from graph
Balanus crenatus, Barnacle			52 mg/kg (whole body)[5]	Behavior, NOED				[55]	L; regulation of metals endpoint - summer experiment
Mysidopsis bahia, Mysid			1.29 mg/kg (whole body)[5]	Growth, LOED				[34]	L; reduction in growth, mean dry weight of animals
			1.29 mg/kg (whole body)[5]	Physiological, LOED				[34]	L; altered O:N ratio, shift towards lipid utilization with increasing cadmium concentration

Summary of Biological Effects Tissue Concentrations for Cadmium

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]**
			2.38 mg/kg (whole body)[5]	Growth, NA				[34]	L; reduction in growth, mean dry weight of animals
			4.36 mg/kg (whole body)[5]	Growth, NA				[34]	L; reduction in growth, mean dry weight of animals
			2.38 mg/kg (whole body)[5]	Physiological, NA				[34]	L; altered O:N ratio, shift towards lipid utilization with increasing cadmium concentration
			4.36 mg/kg (whole body)[5]	Physiological, NA				[34]	L; altered O:N ratio, shift towards lipid utilization with increasing cadmium concentration
Oniscus asellus, Isopod			Day 91: 8.15 mmol/kg	50% mortality				[23]	F
Porcellio scaber, Isopod			Day 63: 5.40 mmol/kg 3.77 mmol/kg	50% mortality 50% mortality				[23]	F

Summary of Biological Effects Tissue Concentrations for Cadmium

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]**
Palaemonetes pugio, Grass shrimp			0.9 mg/kg (whole body)[5]	Mortality, NOED				[26]	L; estimated wet weight
			2.6 mg/kg (whole body)[5]	Mortality, NA				[61]	L; 20% increased mortality over control in 5 ppt water; no statistical analysis
			5.8 mg/kg (whole body)[5]	Mortality, NA				[61]	L; 22% increased mortality over control in 5 ppt water; no statistical analysis
			7 mg/kg (whole body)[5]	Mortality, NA				[61]	L; 25% increased mortality over control in 5 ppt water; no statistical analysis
Palaemonetes pugio, Grass shrimp			Day 21: 4.0 µg/g	25% mortality				[14]	L

Summary of Biological Effects Tissue Concentrations for Cadmium

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]**
Callianassa australiensis, Mole shrimp			Day 14: 4.8 µg/g	50% mortality				[15]	L
Cambarus latimanus, Crayfish			14.9 mg/kg (whole body)[5]	Growth, NOED				[13]	L; no significant difference from control growth at lowest test concentration
			14.9 mg/kg (whole body)[5]	Mortality, NOED				[13]	L; no significant difference from control mortality
			14.9 mg/kg (whole body)[5]	Physiological, NOED				[13]	L; no significant difference from control temperature sensitivity at lowest test concentration
Cambarus latimanus, Crayfish			Month 5: 4.4 µg/g	LOEC				[13]	L

Summary of Biological Effects Tissue Concentrations for Cadmium

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 virilis, Crayfish			Day 14: 5.6 µg/g	25% mortality				[12]	L
Orconectes propinquus, Crayfish			534 mg/kg (whole body)[5]	Mortality, NOED				[39]	L; 7% mortality after 190.5 hours, probably not significant
Chironomus gr. thummi, Midge			0.156 mg/kg (whole body)[5]	Morphology, NOED				[45]	F; 4th instar larvae
Glyptotendipes pallens, Midge			20 mg/kg (whole body)[5]	Behavior, LOED				[44]	L; modified feeding behavior, reduced net spinning activity
			20 mg/kg (whole body)[5]	Growth, LOED				[44]	L; reduced biomass
			30 mg/kg (whole body)[5]	Behavior, NA				[44]	L; modified feeding behavior, reduced net spinning activity
			50 mg/kg (whole body)[5]	Behavior, NA				[44]	L; lethargy

Summary of Biological Effects Tissue Concentrations for Cadmium

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]
			72 mg/kg (whole body)[5]	Behavior, NA				[44]	L; lethargy
			138 mg/kg (whole body)[5]	Behavior, NA				[44]	L; lethargy
			30 mg/kg (whole body)[5]	Growth, NA				[44]	L; reduced biomass
			50 mg/kg (whole body)[5]	Growth, NA				[44]	L; reduced biomass
			72 mg/kg (whole body)[5]	Growth, NA				[44]	L; reduced biomass
			138 mg/kg (whole body)[5]	Growth, NA				[44]	L; reduced biomass
			10 mg/kg (whole body)[5]	Behavior, NOED				[44]	L; no effect on feeding behavior or activity level
			18 mg/kg (whole body)[5]	Behavior, NOED				[44]	L; no effect on feeding behavior or activity level
			10 mg/kg (whole body)[5]	Growth, NOED				[44]	L; no effect on biomass
			18 mg/kg (whole body)[5]	Growth, NOED				[44]	L; no effect on biomass

Summary of Biological Effects Tissue Concentrations for Cadmium

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)[5]	Mortality, NOED				[44]	L; no effect on mortality in 96 hours
			18 mg/kg (whole body)[5]	Mortality, NOED				[44]	L; no effect on mortality in 96 hours
			20 mg/kg (whole body)[5]	Mortality, NOED				[44]	L; no effect on mortality in 96 hours
			30 mg/kg (whole body)[5]	Mortality, NOED				[44]	L; no effect on mortality in 96 hours
			50 mg/kg (whole body)[5]	Mortality, NOED				[44]	L; no effect on mortality in 96 hours
			72 mg/kg (whole body)[5]	Mortality, NOED				[44]	L; no effect on mortality in 96 hours
			138 mg/kg (whole body)[5]	Mortality, NOED				[44]	L; no effect on mortality in 96 hours
Orchesella cincta, Springtail			Day 49: 0.07 mmol/kg	50% mortality				[23]	F
Tomocerus minor, Springtail			Day 63: 0.13 mmol/kg	50% mortality				[23]	F

Summary of Biological Effects Tissue Concentrations for Cadmium

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]**
Platynothrus peltifer, Oribatid mite			Day 63: 0.42 mmol/kg	50% mortality				[23]	F
Classenia sabulosa, Stonefly	<0.3 µg/g 3.5 µg/g 6.6 µg/g		0.1 µg/g ND 1.4 µg/g					[24]	F
Hesperoperla pacifica, Stonefly	<0.3 µg/g 3.5 µg/g 6.6 µg/g		0.2 µg/g ND 1.0 µg/g					[24]	F
Isogenoides sp., Stonefly	<0.3 µg/g 3.5 µg/g 6.6 µg/g		<0.4 µg/g 1.4 µg/g 1.8 µg/g					[24]	F
Pteronarcys californica, Stonefly	<0.3 µg/g 3.5 µg/g 6.6 µg/g		0.1 µg/g ND 1.0 µg/g					[24]	F
Hydropsyche sp., Caddisfly			9.8 mg/kg (whole body)[5]	Mortality, LOED				[38]	L; mortality in one day
			17.4 mg/kg (whole body)[5]	Mortality, LOED				[38]	L; mortality in two days
			29.8 mg/kg (whole body)[5]	Mortality, LOED				[38]	L; mortality in four days

Summary of Biological Effects Tissue Concentrations for Cadmium

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]**
			0.118 mg/kg (whole body)[5]	Mortality, LOED				[38]	L; mortality in one day
			0.0934 mg/kg (whole body)[5]	Mortality, LOED				[38]	L; mortality in two days
			16 mg/kg (whole body)[5]	Mortality, NOED				[38]	L; no effect on mortality in one day
			24.8 mg/kg (whole body)[5]	Mortality, NOED				[38]	L; no effect on mortality in one day
			41.8 mg/kg (whole body)[5]	Mortality, NOED				[38]	L; no effect on mortality in one day
			0.202 mg/kg (whole body)[5]	Mortality, NOED				[38]	L; no effect on mortality in one day
			0.284 mg/kg (whole body)[5]	Mortality, NOED				[38]	L; no effect on mortality in one day
Hydropsyche spp., Caddisfly	<0.3 µg/g 3.5 µg/g 6.6 µg/g		0.2 µg/g 2.2 µg/g 2.8 µg/g					[24]	F
Arctopsyche grandis, Caddisfly	<0.3 µg/g 3.5 µg/g 6.6 µg/g		0.2 µg/g ND[4] 1.4 µg/g					[24]	F

Summary of Biological Effects Tissue Concentrations for Cadmium

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			0.03 mg/kg (gonad)[5]	Development, LOED				[37]	combined, estimated wet weight adult males
			0.14 mg/kg (gonad)[5]	Development, LOED				[37]	combined, estimated wet weight adult females
Fishes									
Oncorhynchus mykiss, Rainbow trout			16.4 mg/kg (whole body)[5]	Mortality, ED100				[29]	L; complete mortality of alevins within 10 hours
			101 mg/kg (whole body)[5]	Mortality, ED100				[29]	L; complete mortality of eggs within 32 hours
			0.84 mg/kg (whole body)[5]	Mortality, ED100				[29]	L; complete mortality of alevins within 320 hours
			0.71 mg/kg (whole body)[5]	Behavior, LOED				[29]	L; erratic swimming
			0.21 mg/kg (whole body)[5]	Morphology, LOED				[29]	L; deformed vertebrae, blood clots in fins

Summary of Biological Effects Tissue Concentrations for Cadmium

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]
			0.21 mg/kg (whole body)[5]	Mortality, LOED				[29]	L; hatching alevins unable to break free from egg membrane, died
			10 mg/kg (whole body)[5]	Physiological, LOED				[30]	L; induction of metallothionein
			0.0599 mg/kg (whole body)[5]	Mortality, NOED				[41]	L; no effect on mortality
			6.4 mg/kg (whole body)[5]	Mortality, ED50				[51]	L; hardness: 279 mg/L $CaCO_3$
			3.74 mg/kg (whole body)[5]	Mortality, ED50				[51]	L; Hardness: 279 Mg/L $CaCO_3$
			4 mg/kg (whole body)[5]	Mortality, ED50				[51]	L; hardness: 70 mg/L $CaCO_3$
			2.2 mg/kg (whole body)[5]	Mortality, ED50				[51]	L; hardness: 70 mg/L $CaCO_3$

Summary of Biological Effects Tissue Concentrations for Cadmium

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]**
Salmo salar, Atlantic Salmon			0.26 mg/kg (yolk sac/stomach)[5]	Growth, LOED				[52]	L; yolk sac/stomach weight - graph and table interpretation
			0.26 mg/kg (yolk sac/stomach)[5]	Mortality, LOED				[52]	L; yolk sac/stomach weight - graph and table interpretation
			0.05 mg/kg (yolk sac/stomach)[5]	Growth, NOED				[52]	L; yolk sac/stomach weight - graph and table interpretation
			0.05 mg/kg (yolk sac/stomach)[5]	Mortality, NOED				[52]	L; yolk sac/stomach weight - graph and table interpretation
Salvelinus fontinalis, Brook trout		3.4 µg/g	Week 38: 10 µg/g, kidney 2 µg/g, liver					[25]	L

Summary of Biological Effects Tissue Concentrations for Cadmium

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]**
Salvelinus fontinalis, Brook Trout			0.175 mg/kg (liver)[5]	Mortality, LOED				[40]	L; significant mortality in 10.5 µg/L at 15 days and 1.91 µg/L at 7 days, but no body burdens measured
			0.232 mg/kg (liver)[5]	Growth, NA				[40]	L; no significant effect on growth
			0.203 mg/kg (liver)[5]	Mortality, NOED				[40]	L
			144 mg/kg (whole body)[5]	Mortality, LOED				[40]	L; significantly reduced survival at lowest test concentration, exp_conc = <3.6

Summary of Biological Effects Tissue Concentrations for Cadmium

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]**
			0.742 mg/kg (liver)[5]	Physiological, LOED				[40]	L; significantly increased metallothionein in whole body tissues at lowest test concentration; no correlation between metallothionein concentration and mortality or whole body tissue residues, exp_conc = < 3.6
			144 mg/kg (whole body)[5]	Physiological, LOED				[40]	L; significantly increased metallothionein in whole body tissues at lowest test concentration; no correlation between metallothionein and mortality or whole body tissue residues, exp_conc = < 3.6

Summary of Biological Effects Tissue Concentrations for Cadmium

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]**
Jordanella floridae, American flagfish			0.4 mg/kg (whole body)[5]	Mortality, LOED				[56]	L; body burden estimated from graph, fish initially exposed as embryos
			0.4 mg/kg (whole body)[5]	Mortality, LOED				[56]	L; body burden estimated from graph, fish not exposed as embryos
			6 mg/kg (whole body)[5]	Growth, NOED				[56]	L; body burden estimated from graph
			0.4 mg/kg (whole body)[5]	Mortality, NOED				[56]	L; body burden estimated from graph, fish not exposed as embryos
			0.09 mg/kg (whole body)[5]	Mortality, NOED				[56]	L; body burden estimated from graph, fish initially exposed as embryos
			6 mg/kg (whole body)[5]	Reproduction, NOED				[56]	L; body burden estimated from graph

Summary of Biological Effects Tissue Concentrations for Cadmium

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 mg/kg (whole body)[5]	Growth, LOED				[57]	L; total length of females
			10 mg/kg (whole body)[5]	Growth, NOED				[57]	L; total length of females
			35 mg/kg (whole body)[5]	Mortality, NOED				[57]	L
Poecilia reticulata, Guppy			8 mg/kg (whole body)[5]	Mortality, ED50				[43]	L; 50% reduction in survival
			0.5 mg/kg (whole body)[5]	Growth, LOED				[43]	L; reduction in body length within 10 days
			1.2 mg/kg (whole body)[5]	Mortality, LOED				[43]	L; 14% reduction in survival
			0.8 mg/kg (whole body)[5]	Growth, NA				[43]	L; reduction in body length within 10 days
Cyprinodon variegatus, Sheepshead minnow			0.9 mg/kg (whole body)[5]	Development, LOED				[49]	L; decreased time to hatch

Summary of Biological Effects Tissue Concentrations for Cadmium

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]**
Platichthys flesus, European flounder			17.2 mg/kg (kidney)[5]	Biochemical, LOED				[54]	L; females - Cd + estradiol injection: RNA:DNA ratio
			21.6 mg/kg (liver)[5]	Biochemical, LOED				[54]	L; females - Cd + estradiol injection: RNA:DNA ratio
			1.82 mg/kg (ovary)[5]	Biochemical, LOED				[54]	L; females - Cd + estradiol injection: RNA:DNA ratio
			33.2 mg/kg (kidney)[5]	Biochemical, NOED				[54]	L; males - Cd + estradiol injection: RNA:DNA ratio
			43.8 mg/kg (liver)[5]	Biochemical, NOED				[54]	L; males - Cd + estradiol injection: RNA:DNA ratio

Summary of Biological Effects Tissue Concentrations for Cadmium

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]**
			4.66 mg/kg (ovary)[5]	Biochemical, NOED				[54]	L; males - Cd + estradiol injection: RNA:DNA ratio
			17.2 mg/kg (kidney)[5]	Mortality, NOED				[54]	L; females - Cd + estradiol injection: survival
			33.2 mg/kg (kidney)[5]	Mortality, NOED				[54]	L; males - Cd + estradiol injection: survival
			43.8 mg/kg (liver)[5]	Mortality, NOED				[54]	L; males - Cd + estradiol injection: survival
			21.6 mg/kg (liver)[5]	Mortality, NOED				[54]	L; females - Cd + estradiol injection: survival
			4.66 mg/kg (ovary)[5]	Mortality, NOED				[54]	L; males - Cd + estradiol injection: survival
			1.82 mg/kg (ovary)[5]	Mortality, NOED				[54]	L; females - Cd + estradiol injection: survival

Summary of Biological Effects Tissue Concentrations for Cadmium

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]**
Pleuronectes americanus, Winter flounder			1 mg/kg (whole body)[5]	Physiological, LOED				[36]	L; induction of metallothionein
Wildlife									
Ambystoma gracile, Salamander			140 mg/kg (whole body)[5]	Behavior, LOED				[50]	L; significant reduction in regurgitation/ food retention
			6.28 mg/kg (whole body)[5]	Growth, LOED				[50]	L; significant reduction in both length and weight
			4.7 mg/kg (whole body)[5]	Growth, LOED				[50]	L; significant reduction in both length and weight
			71.7 mg/kg (whole body)[5]	Behavior, NOED				[50]	L; no significant increase in regurgitation/ food retention

Summary of Biological Effects Tissue Concentrations for Cadmium

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]
			43.5 mg/kg (whole body)[5]	Growth, NOED				[50]	L; no significant reduction in length or weight at highest test concentration
			3.75 mg/kg (whole body)[5]	Growth, NOED				[50]	L; no significant reduction in length or weight
			145 mg/kg (whole body)[5]	Growth, NOED				[50]	L; no significant reduction in length or weight at highest test concentration
			1.62 mg/kg (whole body)[5]	Growth, NOED				[50]	L; no significant reduction in length or weight
			43.5 mg/kg (whole body)[5]	Mortality, NOED				[50]	L; no significant increase in mortality at highest test concentration

Summary of Biological Effects Tissue Concentrations for Cadmium

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]**
			145 mg/kg (whole body)[5]	Mortality, NOED				[50]	L; no significant increase in mortality at highest test concentration
			4.13 mg/kg (whole body)[5]	Mortality, NOED				[50]	L; no significant increase in mortality at highest test concentration

[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] ND = not detected.
[5] 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: PESTICIDE (ORGANOCHLORINE)

Chemical Name (Common Synonyms): CHLORDANE **CASRN:** 57-74-9

Chemical Characteristics

Solubility in Water: 0.1 mg/L at 20 - 30°C [1]

Half-Life: 283 days - 3.8 yrs based on unacclimated aerobic river die-away test and reported soil grab sample data [2]

Log K_{ow}: 6.32 [3]

Log K_{oc}: 6.21 L/kg organic carbon

Human Health

Oral RfD: 6×10^{-5} mg/kg/day [4]

Confidence: Low, uncertainty factor = 1000

Critical Effect: Regional liver hypertrophy in female rats; hepatocellular carcinomas in mice

Oral Slope Factor: $1.3 \times 10^{+0}$ per (mg/kg)/day [4]

Carcinogenic Classification: B2 [4]

Wildlife

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

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

Aquatic Organisms

Partitioning Factors: The major components of technical chlordane include gamma chlordane (24 percent), alpha chlordane (19 percent), and *trans*-nonachlor (7 percent). Alpha chlordane is environmentally more stable and therefore more persistent than gamma chlordane. Oxychlordane is an epoxide metabolite formed in mammalian liver. It is persistent and much more toxic than its parent chemicals [5].

Food Chain Multipliers: In a marine ecosystem the chlordane compounds (nonachlor and oxychlordane) increased significantly with trophic levels from zooplankton to marine mammals [6]. Although the results of the study reported by Kawano et al. [6] indicated a small difference in the chlordane composition in zooplankton from the North Pacific, Bering Sea, and Antarctic, they also revealed a significant difference in chlordane composition between Dall's porpoise and the Weddell seal. *Trans*-chlordane was present in the seal but not in the porpoise, and the percentage composition of oxychlordane in the seal was larger than that in the porpoise. Furthermore, the compositional percentage

of oxychlordane in the Adelie penguin and thick-billed murre was much higher than that in the other organisms. Marine mammals and seabirds accumulated chlordane via food. Biomagnification of total chlordanes through the food chain is strongly evident in marine mammals. Chlordanes are concentrated gradually from zooplankton, through squid and fish, to porpoises and dolphins [7,8]. Chlordane residues in marine mammals are positively correlated with lipid content and not with the age of the animal [9]. Food chain multipliers (FCMs) for *cis*- or *trans*-chlordane for trophic level 3 aquatic organisms were 21.7 (all benthic food web), 1.6 (all pelagic food web), and 13.2 (benthic and pelagic food web). FCMs for trophic level 4 aquatic organisms were 49.5 (all benthic food web), 3.5 (all pelagic food web), and 23.3 (benthic and pelagic food web) [26].

Toxicity/Bioaccumulation Assessment Profile

Chlordane adversely affected sensitive species of fish and aquatic invertebrates at concentrations of 0.2 to 2.0 µg/L. Specifically, survival of shrimp and crabs was reduced at water concentrations of 0.2 to 2.0 µg/L, while survival of freshwater and marine fishes was reduced between 1.7 and 3.0 µg/L. Generally, the uptake of chlordane by aquatic organisms is high, ranging from 216.8 µg organic carbon cleared per gram organism per hour for *Diporeia* spp. to 358 µg organic carbon cleared per gram organism per hour for *Chironomus riparius* [10]. Accumulation of chlordane by *Diporeia* spp., *C. riparius,* or *Lumbriculus variegatus* from whole sediment exposures was greater than that from the elutriate or pore water. Neither species was able to metabolize chlordane. A study by Wilcock et al. [11] has shown that the bivalve *Macomona liliana* can accumulate chlordane bound to sediment at depths below 2 cm. Animals constantly exposed to contaminated sediment accumulated more (5,728 ppb lipid) than those able to feed alternatively on contaminated and uncontaminated sediments (3,617 and 2,756 ppb). An in situ study of the uptake and elimination by adult intertidal benthic infauna of chlordane from contaminated sediment has shown large differences in accumulation between deposit- and suspension-feeding species [12]. In the case of surface feeders, these differences can be attributed to direct exposure to high initial concentration of chlordane in surficial sediments. The extract from the chlordane residues obtained from Lake Michigan lake trout was significantly more toxic (3 to 5 times) than the chlordane used in agricultural applications. Gooch et al. [13] suggested that the increased toxicity of these extracts was due to the presence of the stable metabolite heptachlor epoxide and oxychlordane. Chlordane is persistent in the environment; measurable residues in sediment were found 2.8 years after application to the overlying water column [5]. More than 80 percent of the fish sampled from the Kansas River had detectable chlordanes in their tissue [14]. Residues of *cis*-chlordane and *trans*-chlordane were the most abundant and persistent of the chlordane components measured in fish tissues in a U.S. study conducted aproximately 10 years after the termination of the agricultural use of chlordanes [15]. In birds, technical chlordane and its metabolite oxychlordane are frequently elevated in tissues with high lipid content. In northern gannets, the half-time persistence of *cis*-chlordane, *cis*-nonachlor, and oxychlordane was estimated at 11, 199, and 35 years [16].

Summary of Biological Effects Tissue Concentrations for Chlordane

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									
Lumbriculus variegatus, Oligochaete worm	125 ng/g		28,197 ng/g					[10]	F
	1,406 ng/g		23,031 ng/g[3]						
		BDL[4]	0.03 µ/kg					[17]	F
Crassostrea virginica, Eastern oyster			0.02 mg/kg (whole body)[5]	Growth, ED18				[22]	L; exposure media 65% heptachlor (technical grade)
			2.2 mg/kg (whole body)[5]	Growth, ED28				[22]	L; exposure media 65% heptachlor (technical grade)
			0.3 mg/kg (whole body)[5]	Growth, ED28					L; exposure media 65% heptachlor (technical grade)
			0.075 mg/kg (whole body)[5]	Growth, ED30				[22]	L; exposure media 65% heptachlor (technical grade)
			0.6 mg/kg (whole body)[5]	Growth, ED30				[22]	L; exposure media 65% heptachlor (technical grade)
			0.78 mg/kg (whole body)[5]	Growth, ED33				[22]	L; exposure media 65% heptachlor (technical grade)
			6.5 mg/kg (whole body)[5]	Growth, ED33				[22]	L; exposure media 65% heptachlor (technical grade)

Summary of Biological Effects Tissue Concentrations for Chlordane

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]**
			1.9 mg/kg (whole body)[5]	Growth, ED78				[22]	L; exposure media 65% heptachlor (technical grade)
			14 mg/kg (whole body)[5]	Growth, ED78				[22]	L; exposure media 65% heptachlor (technical grade)
			5.6 mg/kg (whole body)[5]	Growth, ED95				[22]	L; exposure media 65% heptachlor (technical grade)
			47 mg/kg (whole body)[5]	Growth, ED95				[22]	L; exposure media 65% heptachlor (technical grade)
			0.022 mg/kg (whole body)[5]	Growth, NOED				[22]	L; exposure media 65% heptachlor (technical grade)
Crassostrea virginica, Eastern oyster			27 mg/kg (whole body)[5]	Growth, LOED				[23]	L; estimated LOED - no statistical summary in text
			11 mg/kg (whole body)[5]	Growth, NOED				[23]	L; estimated NOED - no statistical summary in text
Corbicula fluminea, Asian clam	21.7 µg/kg OC		2,400 µg/kg lipid				2.04	[21]	F; *trans*-chlordane; %lipid not reported; %sed OC = 2.30

Summary of Biological Effects Tissue Concentrations for Chlordane

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]**
Gonatopsis borealis, Eight-armed squid			*cis*-chlordane: 15 (11-18) µg/kg *trans*-chlordane: 8.1 (6.3-9.9) µg/kg *cis*-nonachlor: 2.4 (2.2-2.8) µg/kg *trans*-nonachlor: 18 (14-20) µg/kg oxychlordane: 1.2 (0.8-1.60) µg/kg total chlordanes: 44 (35-52) µg/kg					[5]	F; lipid samples
Diporeia sp., Amphipod	493 ng/g 430 ng/g		23,729 ng/g 40,086 ng/g					[10]	F
Euphasia superba, Krill			*cis*-chlordane: 0.58 µg/kg *trans*-chlordane: 0.51 µg/kg *cis*-nonachlor: 0.22 µg/kg *trans*-nonachlor: 0.8 µg/kg oxychlordane: 0.1 µg/kg					[6]	F
Palaemonetes pugio, Grass shrimp			4.5 mg/kg (whole body)[5]	Mortality, LOED				[23]	L; estimated LOED - no statistical summary in text

Summary of Biological Effects Tissue Concentrations for Chlordane

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]**
			4.8 mg/kg (whole body)[5]	Mortality, NOED				[23]	L; estimated NOED - no statistical summary in text
Penaeus duorarum, Pink shrimp			1.7 mg/kg (whole body)[5]	Mortality, LOED				[23]	L; estimated LOED - no statistical summary in text
			0.71 mg/kg (whole body)[5]	Mortality, NOED				[23]	L; estimated NOED - no statistical summary in text
Homarus americanus, American lobster			*cis*-chlordane: 80-100 µg/kg, hepatopancreas *trans*-chlordane: 80-100 µg/kg, hepatopancreas *cis*-nonachlor: 30 µg/kg, hepatopancreas *trans*-nonachlor: (380-440) µg/kg, hepatopancreas					[5]	F
Chironomus riparius, Midge	1,663 ng/g 1,741 ng/g		16,224 ng/g 8,417 ng/g					[10]	F

Summary of Biological Effects Tissue Concentrations for Chlordane

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									
Oncorhynchus, Salmo, Salvelinus sp., Salmonids		0.000034 µg/L	19 µg/kg			5.75		[20]	F; *trans*-chlordane, % lipid = 11
	77.8 µg/kg OC		172.7 µg/kg lipid				2.22	[20]	F; *trans*-chlordane; %lipid = 11; %sed OC = 2.70
Salmonids							2.00	[25]	F; *trans*-chlordane
							4.77	[25]	F; *cis*-chlordane
Osmerus mordax, Smelt; *Oncorhynchus velinus,* Coho salmon	2.1 ng/g	34 pg/L	3.6 ng/g 19 ng/g					[18,20]	F; median BSAFs calculated in [18] from field data in [20]
Cyprinus carpio, Carp	2.5 ng/g		18 ng/g				46.3 33.4	[18,19]	F; median BSAFs calculated in [18] from field data in [19]
Cyprinus carpio, Carp	437.5 µg/kg OC		217.9 µg/kg lipid				0.498	[24]	F; *trans*-chlordane; %lipid = 7.8; %sed OC = 0.80
	145.3 µg/kg OC		110.7 µg/kg lipid				0.762	[24]	F; *trans*-chlordane; %lipid = 8.4; %sed OC = 1.79
	112.1 µg/kg OC		161.3 µg/kg lipid				1.439	[24]	F; *trans*-chlordane; %lipid = 9.3; %sed OC = 1.16

Summary of Biological Effects Tissue Concentrations for Chlordane

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, Carp	212.5 µg/kg OC		294.9 µg/kg lipid				1.3878	[24]	F; *cis*-chlordane; %lipid = 7.8; %sed OC = 0.80
	128.5 µg/kg OC		190.5 µg/kg lipid				1.4825	[24]	F; *cis*-chlordane; %lipid = 8.4; %sed OC = 1.79
	86.21 µg/kg OC		258.1 µg/kg lipid				2.9939	[24]	F; *cis*-chlordane; %lipid = 9.3; %sed OC = 1.16
Catastomus commersoni, White sucker	437.5 µg/kg OC		132.5 µg/kg lipid				0.301	[24]	F; *trans*-chlordane; %lipid = 8.3; %sed OC = 0.8
	145.3 µg/kg OC		189.9 µg/kg lipid				1.307	[24]	F; *trans*-chlordane; %lipid = 7.9; %sed OC = 1.79
	112.1 µg/kg OC		266.7 µg/kg lipid				2.379	[24]	F; *trans*-chlordane; %lipid = 4.5; %sed OC = 1.16
Catastomus commersoni, White sucker	212.5 µg/kg OC		192.8 µg/kg lipid				0.9073	[24]	F; *cis*-chlordane; %lipid = 8.3; %sed OC = 0.8
	128.5 µg/kg OC		519 µg/kg lipid				4.0389	[24]	F; *cis*-chlordane; %lipid = 7.9; %sed OC = 1.79
	86.21 µg/kg OC		533.3 µg/kg lipid				6.1861	[24]	F; *cis*-chlordane; %lipid = 4.5; %sed OC = 1.16

Summary of Biological Effects Tissue Concentrations for Chlordane

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]**
Cyprinodon variegatus, Sheepshead minnow			909 mg/kg (whole body)	Mortality, ED35				[22]	L; exposure media 65% heptachlor (technical grade)
			1.2 mg/kg (whole body)[5]	Mortality, ED35				[22]	L; exposure media 65% heptachlor (technical grade)
			0.019 mg/kg (whole body)[5]	Mortality, ED5				[22]	L; exposure media 65% heptachlor (technical grade)
			0.01 mg/kg (whole body)[5]	Mortality, ED5				[22]	L; exposure media 65% heptachlor (technical grade)
			17.5 mg/kg (whole body)[5]	Mortality, ED50				[22]	L; exposure media 65% heptachlor (technical grade)
			2 mg/kg (whole body)[5]	Mortality, ED50				[22]	L; exposure media 65% heptachlor (technical grade)
			3.9 mg/kg (whole body)[5]	Mortality, ED60				[22]	L; exposure media 65% heptachlor (technical grade)
			32 mg/kg (whole body)[5]	Mortality, ED60				[22]	L; exposure media 65% heptachlor (technical grade)
			47 mg/kg (whole body)[5]	Mortality, ED85				[22]	L; exposure media 65% heptachlor (technical grade)
			6.1 mg/kg (whole body)[5]	Mortality, ED85				[22]	L; exposure media 65% heptachlor (technical grade)

Summary of Biological Effects Tissue Concentrations for Chlordane

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]**
Cyprinodon variegatus, Sheepshead minnow			281 mg/kg (whole body)[5]	Mortality, LOED				[23]	L; estimated LOED - no statistical summary in text
			3.18 mg/kg (whole body)[5]	Mortality, LOED				[23]	L
			3.18 mg/kg (whole body)[5]	Reproduction, LOED				[23]	L; hatching success of fry from exposed parents
			0.6 mg/kg (whole body)[5]	Mortality, NOED				[23]	L; estimated NOED - no statistical summary in text
			87 mg/kg (whole body)[5]	Mortality, NOED				[23]	L; estimated NOED - no statistical summary in text
			1.38 mg/kg (whole body)[5]	Mortality, NOED				[23]	L
			1.38 mg/kg (whole body)[5]	Reproduction, NOED				[23]	L; hatching success of fry from exposed parents
Lagodon rhomboides, Pinfish			16.6 mg/kg (whole body)[5]	Mortality, LOED				[23]	L; estimated LOED - no statistical summary in text
Leiostomus xanthurus, Spot			0.16 mg/kg (whole body)[5]	Mortality, ED25				[22]	L; exposure media 65% heptachlor (technical grade)

Summary of Biological Effects Tissue Concentrations for Chlordane

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]
			0.55 mg/kg (whole body)[5]	Mortality, ED25				[22]	L; exposure media 65% heptachlor (technical grade)
			0.89 mg/kg (whole body)[5]	Mortality, ED35				[22]	L; exposure media 65% heptachlor (technical grade)
			0.22 mg/kg (whole body)[5]	Mortality, ED35				[22]	L; exposure media 65% heptachlor (technical grade)
			3.3 mg/kg (whole body)[5]	Mortality, ED40				[22]	L; exposure media 65% heptachlor (technical grade)
			0.94 mg/kg (whole body)[5]	Mortality, ED40				[22]	L; exposure media 65% heptachlor (technical grade)
			1.6 mg/kg (whole body)[5]	Mortality, ED70				[22]	L; exposure media 65% heptachlor (technical grade)
			7.1 mg/kg (whole body)[5]	Mortality, ED70				[22]	L; exposure media 65% heptachlor (technical grade)
			0.7 mg/kg (whole body)[5]	Mortality, ED85				[22]	L; exposure media 65% heptachlor (technical grade)
			3.5 mg/kg (whole body)[5]	Mortality, ED85				[22]	L; exposure media 65% heptachlor (technical grade)
			0.01 mg/kg (whole body)[5]	Mortality, NOED				[22]	L; exposure media 65% heptachlor (technical grade)

Summary of Biological Effects Tissue Concentrations for Chlordane

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]**
			0.01 mg/kg (whole body)[5]	Mortality, NOED				[22]	L; exposure media 65% heptachlor (technical grade)
			0.01 mg/kg (whole body)[5]	Mortality, NOED				[22]	L; exposure media 65% heptachlor (technical grade)
			0.01 mg/kg (whole body)[5]	Mortality, NOED				[22]	L; exposure media 65% heptachlor (technical grade)
Cottus cognatus, Slimy sculpin	2.1 ng/g	34 g/L	30 µg/kg			5.95	2.47	[18,20]	F; *trans*-chlordane, % lipid = 8
	77.8 µg/kg OC		375 µg/kg lipid				4.821	[20]	F; *trans*-chlordane; %lipid = 8; %sed OC = 2.70
Pimelodus albicans, Oligosarcus jenynsi, Prochilodus platensis	3.4 ng/g	0.8 ng/L	2.9 µg/g				20	[18,21]	F; median BSAFs calculated in [18] from field data in [21]
Prochilodus platensis, Curimata	20 µg/kg OC		4,600 µg/kg lipid				230	[21]	F; *trans*-chlordane; %lipid not reported; %sed OC = 1
Pimelodus albicans, Mandi	20 µg/kg OC		1,000 µg/kg lipid				50	[21]	F; *trans*-chlordane; %lipid not reported; %sed OC = 1

Summary of Biological Effects Tissue Concentrations for Chlordane

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]**
Wildlife									
Ducks							0.83 19.5	[18,19]	F; median BSAFs calculated in [18] from field data in [19]

[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.
[5] 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: PESTICIDE (ORGANOPHOSPHATE)

Chemical Name (Common Synonyms): CHLORPYRIFOS **CASRN:** 2921-88-2

Chemical Characteristics

Solubility in Water: 0.7 ppm at 20°C [1]

Half-Life: No data [2]

Log K_{ow}: 5.26 [3]

Log K_{oc}: 5.17 L/kg organic carbon

Human Health

Oral RfD: 3 x 10^{-3} mg/kg/day [4]

Confidence: Medium, uncertainty factor =10[4]

Critical Effect: Decreased plasma cholinesterase activity after 9 days of 20-day human feeding study

Oral Slope Factor): No data [4]

Carcinogenic Classification: No data [4], D[5]

Wildlife

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

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

Aquatic Organisms

Partitioning Factors: The only partitioning factors for chlorpyrifos in aquatic organisms found in the literature were log BCF of 3.23 for an isopod [14].

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

Toxicity/Bioaccumulation Assessment Profile

Chlorpyrifos or Dursban is an organophosphorus insecticide which is used to control both adult and larval mosquitoes [6]. It is more toxic to nontarget organisms like cladocerans, amphipods, and other organisms than to mosquito larvae, however. The increase of chlorpyrifos concentration in water proportionally increased the bioconcentration factor in fish [7]. A low recovery (20 percent or lower) of chlorpyrifos from C-18 columns was attributed to its high binding affinity [8]. Also, acidic or basic conditions were

not effective in reducing its concentration in water [9]. Because of the binding capacity and the high K_{ow}, chlorpyrifos does not remain in aqueous solution or suspension but is bound to the organic and clay fractions of sediments. The time for sediment-associated pesticides to degrade and reach nontoxic states is much greater than for aqueous phases [10]. The responses to chlorpyrifos from single-species tests were compared to responses observed in a field mesocosm [11]. The EC50 for seven species in the mesocosms ranged from 0.1 to 3.4 µg/L and were within the same order of magnitude as the laboratory data. Toxicity to the most sensitive test species, *D. magna* , at 1 µg/L was representative of sensitive indigenous species.

The results of toxicity tests exposing *Chironomus tentans* to sediments with differing organic carbon content spiked with chlorpyrifos revealed that an organic carbon partitioning model can be reasonably used to predict the toxicity of chlorpyrifos to benthic macroinvertebrates [12]. The TOC-normalized, solid-phase concentration of chlorpyrifos was no better predictor of the toxicity of the pesticide to *C. tentans* than the sediment dry-weight concentration of chlorpyrifos. The effects based on predicted pore-water concentrations were accurate to within a factor of two of expected effects based on water-only toxicity tests with the midge.

Distinct pulses of pesticides, including chlorpyrifos, were detected in the San Joaquin River and in the Sacramento River following rainfall events [13]. The results of short-term chronic tests with *Ceriodaphnia dubia* indicated that Sacramento River water at Rio Vista was acutely toxic for three consecutive days, while San Joaquin River water at Vernalis was toxic for 12 consecutive days.

Summary of Biological Effects Tissue Concentrations for Chlorpyrifos

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									
Mytilus galloprovincialis, Mediterranean mussel			42 mg/kg (whole body)[4]	Mortality, ED50				[19]	L; estimated from table 4
			4 mg/kg (whole body)[4]	Morphology, LOED				[19]	L; presence of functional byssus
			1.9 mg/kg (whole body)[4]	Morphology, NOED				[19]	
			4 mg/kg (whole body)[4]	Mortality, NOED				[19]	L; estimated from table 4
Asellus aquaticus, Isopod		0.7 µg/L 5.0 µg/L	140,000 µg/kg 260,000 µg/kg		3.23			[14]	F
Fishes									
Pimephales promelas, Fathead minnow			2 mg/kg (whole body)[4]	Growth, LOED				[21]	L; significant reduction in growth
			4.5 mg/kg (whole body)[4]	Morphology, LOED				[21]	L; body constriction behind opercula, shortening of caudal peduncle
			4.5 mg/kg (whole body)[4]	Mortality, LOED				[21]	L; significant reduction in survival

Summary of Biological Effects Tissue Concentrations for Chlorpyrifos

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]
			0.45 mg/kg (whole body)[4]	Physiological, LOED				[21]	L; inhibition of acetylcholinesterase (ACHE) activity
			4.5 mg/kg (whole body)[4]	Growth, NA				[21]	L; significant reduction in growth
			4.5 mg/kg (whole body)[4]	Physiological, NA				[21]	L; inhibition of acetylcholinesterase (ACHE) activity
			2 mg/kg (whole body)[4]	Physiological, NA				[21]	L; inhibition of acetylcholinesterase (ACHE) activity
			1.1 mg/kg (whole body)[4]	Physiological, NA				[21]	L; inhibition of acetylcholinesterase (ACHE) activity
			1.1 mg/kg (whole body)[4]	Growth, NOED				[21]	L; no effect on growth
			0.45 mg/kg (whole body)[4]	Growth, NOED				[21]	L; no effect on growth
			0.2 mg/kg (whole body)[4]	Growth, NOED				[21]	L; no effect on growth
			2 mg/kg (whole body)[4]	Morphology, NOED				[21]	L; no effect on appearance or development
			1.1 mg/kg (whole body)[4]	Morphology, NOED				[21]	L; no effect on appearance or development

Summary of Biological Effects Tissue Concentrations for Chlorpyrifos

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]**
			0.45 mg/kg (whole body)[4]	Morphology, NOED				[21]	L; no effect on appearance or development
			0.2 mg/kg (whole body)[4]	Morphology, NOED				[21]	L; no effect on appearance or development
			2 mg/kg (whole body)[4]	Mortality, NOED				[21]	L; no effect on survival
			1.1 mg/kg (whole body)[4]	Mortality, NOED				[21]	L; no effect on survival
			0.45 mg/kg (whole body)[4]	Mortality, NOED				[21]	L; no effect on survival
			0.2 mg/kg (whole body)[4]	Mortality, NOED				[21]	L; no effect on survival
			0.2 mg/kg (whole body)[4]	Physiological, NOED				[21]	L; inhibition of acetylcholinesterase (ACHE) activity
Gambusia affinis, Mosquito fish			0.0352 mg/kg (whole body)[4]	Mortality, NOED				[22]	L; no effect on survivorship after 3 days
Poecilia reticulata, Guppy		0.9 µg/L 1.9 µg/L 3.9 µg/L 10 µg/L 19 µg/L 37 µg/L	6 µg/g lipid 33 µg/g lipid 66 µg/g lipid 350 µg/g lipid 710 µg/g lipid 2,100 µg/g lipid					[15]	L

Summary of Biological Effects Tissue Concentrations for Chlorpyrifos

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]**
Poecilia reticulata, Guppy			2,810 mg/kg (whole body)[4]	Mortality, ED100				[18]	L; lifestage: 2-3 months
Gasterosteus aculeatus, Three-spined stickleback		0.12 µg/L 0.46 µg/L 1.0 µg/L	8.1 µg/g lipid 31.2 µg/g lipid 125 µg/g lipid					[16]	L
Cyprinodon variegatus, Sheepshead minnow		series 1 0.78µg/L 1.7 µg/L 3.0 µg/L 6.8 µg/L	 0.033 µg/g 0.22 µg/g 0.45 µg/g 4.8 µg/g					[17]	L (low feeding: 20 *Artemia*/fish/ feeding)
Cyprinodon variegatus, Sheepshead minnow		series 1 0.78µg/L 1.7 µg/L 3.0 µg/L 6.8 µg/L	 0.054 µg/g 0.12 µg/g 0.78 µg/g 2.9 µg/g					[17]	L (medium feeding: 110 *Artemia*/fish/ feeding)
		series 1 0.78µg/L 1.7 µg/L 3.0 µg/L 6.8 µg/L	 0.66 µg/g 0.19 µg/g 2.9 µg/g 7.3 µg/g					[17]	L (high feeding: 550 *Artemia*/ fish/feeding)
		series 2 3.1 µg/L 7.2 µg/L 14 µg/L 26 µg/L 52 µg/L	 0.67 µg/g 1.8 µg/g 4.3 µg/g 17 µg/g 34 µg/g					[17]	L (low feeding: 20 *Artemia*/fish/ feeding)

Summary of Biological Effects Tissue Concentrations for Chlorpyrifos

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]**
		series 2 3.1 µg/L 7.2 µg/L 14 µg/L 26 µg/L 52 µg/L	 0.82 µg/g 2.9 µg/g 5.5 µg/g 15.9 µg/g 52 µg/g					[17]	L (medium feeding: 110 *Artemia*/fish/ feeding)
		series 2 3.1 µg/L 7.2 µg/L 14 µg/L 26 µg/L 52 µg/L	 2.2 µg/g 5.3 µg/g 13.9 µg/g 37 µg/g 95 µg/g					[17]	L (high feeding: 550 *Artemia*/ fish/feeding)
Leuresthes tenuis, California grunion			0.21 mg/kg (whole body)[4]	Behavior, LOED				[23]	L; reduced activity
			0.038 mg/kg (whole body)[4]	Growth, LOED				[23]	L; significant reduction in weight of fry
			0.21 mg/kg (whole body)[4]	Growth, LOED				[23]	L; significant reduction in mean fish weight
			0.21 mg/kg (whole body)[4]	Morphology, LOED				[23]	L; fish appeared darker, abnormal lateral flexure of the back
			0.58 mg/kg (whole body)[4]	Mortality, LOED				[23]	L; nearly 40% reduction in fry survival
			0.39 mg/kg (whole body)[4]	Mortality, LOED				[23]	L; 38% reduction in fry survival

Summary of Biological Effects Tissue Concentrations for Chlorpyrifos

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]**
			0.58 mg/kg (whole body)[4]	Growth, NA				[23]	L; significant reduction in weight of fry
			0.15 mg/kg (whole body)[4]	Behavior, NOED				[23]	L; no effect on behavior
			0.015 mg/kg (whole body)[4]	Growth, NOED				[23]	L; no effect on weight of fry
			0.15 mg/kg (whole body)[4]	Growth, NOED				[23]	L; no effect on growth
			0.15 mg/kg (whole body)[4]	Morphology, NOED				[23]	L; no effect on morphology
			0.015 mg/kg (whole body)[4]	Mortality, NOED				[23]	L; no effect on fry mortality
			0.15 mg/kg (whole body)[4]	Mortality, NOED				[23]	L; no effect on fry survival
			0.038 mg/kg (whole body)[4]	Mortality, NOED				[23]	L; no effect on fry mortality
			0.21 mg/kg (whole body)[4]	Mortality, NOED				[23]	L; no effect on fry survival
Opsanus beta, Gulf toadfish			770 mg/kg (whole body)[4]	Development, ED25				[20]	L; delayed development of 25% of sac fry
			12 mg/kg (whole body)[4]	Growth, ED25				[20]	L; 25% reduction in average weight of fry
			175 mg/kg (whole body)[4]	Growth, ED50				[20]	L; 50% reduction in average weight of fry

Summary of Biological Effects Tissue Concentrations for Chlorpyrifos

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]
			770 mg/kg (whole body)[4]	Behavior, LOED				[20]	L; hyperactivity, hyperventilation
			0.95 mg/kg (whole body)[4]	Growth, LOED				[20]	L; 9% reduction in fry weight
			770 mg/kg (whole body)[4]	Mortality, LOED				[20]	L; significant increase in fry mortality
			2.2 mg/kg (whole body)[4]	Growth, NA				[20]	L; 19% reduction in fry weight
			4.7 mg/kg (whole body)[4]	Growth, NA				[20]	L; 21% reduction in fry weight
			15 mg/kg (whole body)[4]	Growth, NA				[20]	L; 37% reduction in fry weight
			30 mg/kg (whole body)[4]	Growth, NA				[20]	L; 42% reduction in fry weight
			9.9 mg/kg (whole body)[4]	Growth, NA				[20]	L; 21% reduction in average weight of fry
			45 mg/kg (whole body)[4]	Growth, NA				[20]	L; 35% reduction in average weight of fry
			770 mg/kg (whole body)[4]	Growth, NA				[20]	L; 62% reduction in average weight of fry
			0.14 mg/kg (whole body)[4]	Growth, NOED				[20]	L; no effect on growth
			12 mg/kg (whole body)[4]	Mortality, NOED				[20]	L; no effect on fry mortality
			9.9 mg/kg (whole body)[4]	Mortality, NOED				[20]	L; no effect on fry mortality

Summary of Biological Effects Tissue Concentrations for Chlorpyrifos

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]
			45 mg/kg (whole body)[4]	Mortality, NOED				[20]	L; no effect on fry mortality
			175 mg/kg (whole body)[4]	Mortality, NOED				[20]	L; no effect on fry 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: METAL

Chemical Name (Common Synonyms): CHROMIUM (hexavalent) **CASRN:** 18540-29-9

Chemical Characteristics

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

Log K_{ow}: – **Log K_{oc}:** –

Human Health

Oral RfD: 5 x 10^{-3} mg/kg/day [2] **Confidence:** Low, uncertainty factor = 500

Critical Effect: No effects observed (Currently under review by RfD/RfC Work Group)

Oral Slope Factor: Not available [2] **Carcinogenic Classification:** A [2]

Wildlife

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

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

Aquatic Organisms

Partitioning Factors: In aqueous solutions, within a pH range of 6 to 8, hexavalent chromium is distributed between two species: monovalent hydrochromate anion and divalent chromate anion. Hexavalent chromium may account for 75 to 85 percent of the dissolved chromium while trivalent chromium is generally below detection limits in most oxic surface waters [3]. In some surface waters, as much as 10 to 15 percent of the dissolved chromium may be present in the colloidal/organic form. A log BCF of 2.74 was reported for *Daphia magna* [9].

Food Chain Multipliers: Little evidence exists for the bioaccumulation/biomagnification of chromium in aquatic food webs, although sediments frequently contain elevated concentrations of trivalent chromium [4].

Toxicity/Bioaccumulation Assessment Profile

Chromium appears to have limited mobility under typical conditions in most aquatic habitats because the trivalent form tends to bind to sediments. Plants can, however, bioaccumulate and reduce chromium.

Tissue residue-toxicity relationships can also be variable because organisms might sequester metal in various forms that might be analytically measurable as tissue residue but are actually stored in unavailable forms within the organism as a form of detoxification [5]. Whole body residues might also not be indicative of effects concentrations at the organ level because concentrations in target organs, such as the kidneys and liver, may be 20 times more than whole body residues [6]. The application of "clean" chemical analytical and sample preparation techniques is critical for the accurate measurement of metal tissue residues [7]. Accumulation of hexavalent chromium in the gills of rainbow trout was significantly higher at pH 6.5 than at 8.1 and is directly coupled with oxygen transfer, irrespective of exposure time or concentration [8]. The authors of that study suggested that chromium uptake might be related to the $HCr0_4$ to $Cr0_4$ ratio, whereby the monovalent hydrochromate anion is taken up more readily by the gill tissue.

Summary of Biological Effects Tissue Concentrations for Chromium

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									
Mytilus galloprovincialis, Mussel			0.73-1.04 mg/kg				0.018	[13]	F
Daphnia magna, Cladoceran			Day 21: 1.1 mmol/kg	10% mortality	2.74			[9]	F
Xantho hydrophilus, Mud crab		1 µg/L	0.2 µg/g (whole body) 0.2 µg/g (hepatopancreas) 0.4 µg/g (gill) 0.05 µg/g (muscle)					[12]	F
Fishes									
Oncorhynchus mykiss (*Salmo gairdneri)*, Rainbow trout		2.5 mg/L	Day 22: 171 µg/g (skin) 187 µg/g (muscle) 132 µg/g (gastro-intestinal) 49.8 µg/g (bone) 75.4 µg/g (kidney) 77.2 µg/g (blood) 41.4 µg/g (gill) 16.9 µg/g (fat) 27.3 µg/g (liver)					[10]	L
		10.0 µg/L 1.3 µg/L	133.6 µg/g 16.6 µg/g					[11]	L

Summary of Biological Effects Tissue Concentrations for Chromium

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 *Salmo gairdneri)*, Rainbow trout		2.0 mg/L	2.0 µg/g (whole body) 31.7 µg/g (gill) 6.2 µg/g (digestive tract) 2.0 µg/g (liver) 6.7 µg/g (kidney)	100% survival				[8]	L; pH = 6.5
		2.0 mg/L	0.9 µg/g (whole body) 5.1 µg/g (gill) 7.4 µg/g (digestive tract) 3.4 µg/g (liver) 8.5 µg/g (kidney)	100% survival				[8]	L; pH = 7.8
		5.0 mg/L	5.5 µg/g (whole body) 51.8 µg/g (gill) 9.5 µg/g (digestive tract) 3.8 µg/g (liver) 10.7 µg/g (kidney)	100% survival				[8]	L; pH = 6.5
		5.0 mg/L	2.3 µg/g (whole body) 10.6 µg/g (gill) 11.2 µg/g (digestive tract) 5.1 µg/g (liver) 12.2 µg/g (kidney)	100% survival				[8]	L; pH = 7.8
		16.5 mg/L	8.7 µg/g (whole body) 139 µg/g (gill) 23.4 µg/g (digestive tract) 24.8 µg/g (liver) 43.2 µg/g (kidney)	25% survival				[8]	L; pH = 6.5

Summary of Biological Effects Tissue Concentrations for Chromium

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 (Salmo gairdneri), Rainbow trout		16.5 mg/L	8.9 µg/g (whole body) 35.3 µg/g (gill) 22.6 µg/g (digestive tract) 25.9 µg/g (liver) 24.6 µg/g (kidney)	63% survival				[8]	L; pH = 7.8
		50 mg/L		0% survival				[8]	L; pH = 6.5
		50 mg/L	10.5 µg/g (whole body) 37.6 µg/g (gill) 45.0 µg/g (digestive tract) 84.6 µg/g (liver) 70.3 µg/g (kidney)	50% survival				[8]	L; pH = 7.8
Oncorhynchus mykiss, Rainbow trout			45 mg/kg (digestive tract)[4]	Mortality, ED50				[14]	L; pH 7.8; increased mortality relative to control
			37.6 mg/kg (gill)[4]	Mortality, ED50				[14]	L; pH 7.8; increased mortality relative to control
			70.3 mg/kg (kidney)[4]	Mortality, ED50				[14]	L; pH 7.8; increased mortality relative to control
			85.6 mg/kg (liver)[4]	Mortality, ED50				[14]	L; pH 7.8; increased mortality relative to control

Summary of Biological Effects Tissue Concentrations for Chromium

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]
			10.5 mg/kg (whole body)[4]	Mortality, ED50				[14]	L; pH 7.8; increased mortality relative to control
			23.4 mg/kg (digestive tract)[4]	Mortality, ED75				[14]	L; pH 6.5; increased mortality relative to control
			139 mg/kg (gill)[4]	Mortality, ED75				[14]	L; pH 6.5; increased mortality relative to control
			43.1 mg/kg (kidney)[4]	Mortality, ED75				[14]	L; pH 6.5; increased mortality relative to control
			24.8 mg/kg (liver)[4]	Mortality, ED75				[14]	L; pH 6.5; increased mortality relative to control
			8.7 mg/kg (whole body)[4]	Mortality, ED75				[14]	L; pH 6.5; increased mortality relative to control
			22.6 mg/kg (digestive tract)[4]	Mortality, NA				[14]	L; pH 7.8; increased mortality relative to control
			35.3 mg/kg (gill)[4]	Mortality, NA				[14]	L; pH 7.8; increased mortality relative to control

Summary of Biological Effects Tissue Concentrations for Chromium

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]
			24.6 mg/kg (kidney)[4]	Mortality, NA				[14]	L; pH 7.8; increased mortality relative to control
			25.9 mg/kg (liver)[4]	Mortality, NA				[14]	L; pH 7.8; increased mortality relative to control
			8.9 mg/kg (whole body)[4]	Mortality, NA				[14]	L; pH 7.8; increased mortality relative to control
			9.5 mg/kg (digestive tract)[4]	Mortality, NOED				[14]	L; pH 6.5; no increased mortality relative to control
			11.2 mg/kg (digestive tract)[4]	Mortality, NOED				[14]	L; pH 7.8; no increased mortality relative to control
			51.8 mg/kg (gill)[4]	Mortality, NOED				[14]	L; pH 6.5; no increased mortality relative to control
			10.6 mg/kg (gill)[4]	Mortality, NOED				[14]	L; pH 7.8; no increased mortality relative to control
			10.7 mg/kg (kidney)[4]	Mortality, NOED				[14]	L; pH 6.5; no increased mortality relative to control

Summary of Biological Effects Tissue Concentrations for Chromium

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]
			12.2 mg/kg (kidney)[4]	Mortality, NOED				[14]	L; ph 7.8; no increased mortality relative to control
			3.8 mg/kg (liver)[4]	Mortality, NOED				[14]	L; pH 6.5; no increased mortality relative to control
			5.1 mg/kg (liver)[4]	Mortality, NOED				[14]	L; pH 7.8; no increased mortality relative to control
			5.5 mg/kg (whole body)[4]	Mortality, NOED				[14]	L; pH 6.5; no increased mortality relative to control
			2.3 mg/kg (whole body)[4]	Mortality, NOED				[14]	L; pH 7.8; no increased mortality relative to control

[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): CHRYSENE **CASRN:** 218-01-9

Chemical Characteristics

Solubility in Water: 0.0020 mg/L at 25°C [1]

Half-Life: 1.02 yrs - 2.72 yrs based on aerobic soil die-away test 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): Not available [4]

Carcinogenic Classification: B2 [4]

Wildlife

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

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

Aquatic Organisms

Partitioning Factors: Partitioning factors for chrysene in aquatic organisms were not found in the literature.

Food Chain Multipliers: Food chain multipliers for chrysene in aquatic organisms were not found in the literature. Log BAF values found in the literature ranged from -0.68 for the clam *Macoma nasuta* [7] to 4.31 for the amphipod *Pontoporeia hoyi* [9].

Toxicity/Bioaccumulation Assessment Profile

The results from the laboratory experiments performed by Harkey [5] indicated that accumulation of chrysene from elutriates was significantly lower than that from whole sediment, and the elutriate-sediment accumulations followed a downward curve over time. A similar curve was observed for pore water-to-sediment accumulation ratios. The concentrations of chrysene in whole sediment and pore water were 34.2 ng/g and 0.305 mg/mL, respectively [5]. Uptake rate coefficients for *Diporeia* spp. were highest in pore water (244.3 $\mu g/g_{oc}/h$) and lowest in elutriate (55.2 $\mu g/g_{oc}/h$). The authors concluded that aqueous

extracts of whole sediment did not accurately represent the exposure observed in whole sediment [5]. The aqueous extracts of whole sediment underexposed organisms, compared to whole sediment, even after adjusting accumulation to the fraction of organic carbon contained in the test media. While the total chrysene concentration in the sediment stayed constant, total concentration decreased appreciably in pore water and elutriate over the course of the exposure, and it is likely that the bioavailability concentrations in these media also decreased. Benthic amphipods, *Gammarus pulex,* exposed to sediments containing polynuclear aromatic hydrocarbons (PAHs) and water spiked with sediment extract from PAH-contaminated sediment, accumulated chrysene in direct proportion to exposure concentrations [6].

Summary of Biological Effects Tissue Concentrations for Chrysene

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									
Macoma nasuta, Clam	7.4 ng/g		29 ng/g			-0.21		[7]	F
	5.9 ng/g		8.1 ng/g			-0.68		[7]	F
	50 ng/g		29.8 ng/g			-0.40		[7]	F
	41 ng/g		30 ng/g			-0.28		[7]	F
	174 ng/g		88 ng/g			-0.33		[7]	F
	249 ng/g		72 ng/g			-0.41		[7]	F
Diporeia spp., Amphipod	15 nmol/g		213 nmol/g					[8]	L
Diporeia spp., Amphipod			2.6 mg/kg (whole body)[4]	Mortality, NOED				[5]	L; no increase in mortality in 96 hours
Pontoporeia hoyi, Amphipod	50 ng/g	7 ng/mL	600 ng/g			4.31		[9]	L
	30 ng/g	1.5 ng/mL	180 ng/g					[9]	L

Summary of Biological Effects Tissue Concentrations for Chrysene

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									
Oncorhynchus mykiss, Rainbow trout			30 mg/kg (whole body)[4]	Physiological, LOED				[11]	L; induction of hepatic mixed function oxidases
Cyprinus carpio, Common carp			109 mg/kg (liver)[4]	Physiological, NA				[10]	L; significant increase in EROD enzyme and P450 1a protein content

[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: METAL

Chemical Name (Common Synonyms): COPPER **CASRN:** 7440-50-8

Chemical Characteristics

Solubility in Water: Insoluble [1]

Half-Life: Not applicable, stable [1]

Log K_{ow}: —

Log K_{oc}: —

Human Health

Oral RfD: Not available [2]

Confidence: —

Critical Effect: —

Oral Slope Factor: No data [2]

Carcinogenic Classification: D [2]

Wildlife

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

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

Aquatic Organisms

Partitioning Factors: Up to 29 different species of copper can be present in aqueous solution in the pH range from 6 to 9. Aqueous copper speciation and toxicity depend on the ionic strength of the water. The hydroxide species and free copper ions are mostly responsible for toxicity, while copper complexes consisting of carbonates, phosphates, nitrates, ammonia, and sulfates are weakly toxic or nontoxic. Copper in the aquatic environment can partition to dissolved and particulate organic carbon. The bioavailability of copper also can be influenced to some extent by total water hardness. Bioavailability of copper in sediments is controlled by the acid-volatile sulfide (AVS) concentration [12]. A log BCF of 3.77 was reported for the midge [4].

Food Chain Multipliers: Little evidence exists to support the general occurrence of biomagnification of copper in the aquatic environment [3]. Copper is taken up by aquatic organisms primarily through dietary exposure.

Toxicity/Bioaccumulation Assessment Profile

The free copper ions are the most bioavailable inorganic forms, although they might account for only a minor proportion of the total dissolved metal. The concentration of copper found in interstitial water is usually much lower than that in surface water. The amount of bioavailable copper in sediment is controlled in large part by the concentration of AVS and organic matter. A considerable number of aquatic species are sensitive to dissolved concentrations of copper in the range of 1-10 µg/L. Metal metabolism by aquatic biota has significant affects on metal accumulation, distribution in tissues, and toxic effects. Concentration of copper in benthic organisms from contaminated areas can be one to two orders of magnitude higher than normal. Copper is accumulated by aquatic organisms primarily through dietary exposure [3]. However, most organisms retain only a small proportion of the heavy metals ingested with their diet.

Rule and Alden [13] studied the relationship between uptake of cadmium and copper from the sediment by blue mussel (*Mytilus edulis*), grass shrimp (*Palaemonetes pugio*), and hard clam (*Mercenaria mercenaria*). The uptake of copper by all organisms was related only to copper concentration in sediment.

Summary of Biological Effects Tissue Concentrations for Copper

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]**
Plants									
Eichhornia crassipes, Water Hyacinth			11.4 mg/kg (leaf)	Growth, LOED				[22]	L; reduced growth rate, chlorosis
			549 mg/kg (root)	Growth, LOED				[22]	L; reduced growth rate, chlorosis
			37.8 mg/kg (stem)	Growth, LOED				[22]	L; reduced growth rate, chlorosis
			11.4 mg/kg (leaf)	Morphology, LOED				[22]	L; chlorosis, browning, necrosis, waterlogging of tissues
			549 mg/kg (root)	Morphology, LOED				[22]	L; chlorosis, browning, necrosis, waterlogging of tissues
			37.8 mg/kg (stem)	Morphology, LOED				[22]	L; chlorosis, browning, necrosis, waterlogging of tissues
			13.8 mg/kg (leaf)	Growth, NA				[22]	L; reduced growth rate, chlorosis
			1,750 mg/kg (root)	Growth, NA				[22]	L; reduced growth rate, chlorosis
			74.4 mg/kg (stem)	Growth, NA				[22]	L; reduced growth rate, chlorosis

Summary of Biological Effects Tissue Concentrations for Copper

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]**
			13.8 mg/kg (leaf)	Morphology, NA				[22]	L; chlorosis, browning, necrosis, waterlogging of tissues
			1,750 mg/kg (root)	Morphology, NA				[22]	L; chlorosis, browning, necrosis, waterlogging of tissues
			74.4 mg/kg (stem)	Morphology, NA				[22]	L; chlorosis, browning, necrosis, waterlogging of tissues
			4.6 mg/kg (leaf)	Growth, NOED				[22]	L; no effect on growth
			7.8 mg/kg (leaf)	Growth, NOED				[22]	L; no effect on growth
			20.8 mg/kg (root)	Growth, NOED				[22]	L; no effect on growth
			82.8 mg/kg (root)	Growth, NOED				[22]	L; no effect on growth
			10 mg/kg (stem)	Growth, NOED				[22]	L; no effect on growth
			15.2 mg/kg (stem)	Growth, NOED				[22]	L; no effect on growth
			4.6 mg/kg (leaf)	Morphology, NOED				[22]	L; no effect on plant appearance

Summary of Biological Effects Tissue Concentrations for Copper

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]**
					7.8 mg/kg (leaf)	Morphology, NOED				[22]	L; no effect on plant appearance
					20.8 mg/kg (root)	Morphology, NOED				[22]	L; no effect on plant appearance
					82.8 mg/kg (root)	Morphology, NOED				[22]	L; no effect on plant appearance
					10 mg/kg (stem)	Morphology, NOED				[22]	L; no effect on plant appearance
					15.2 mg/kg (stem)	Morphology, NOED				[22]	L; no effect on plant appearance
Invertebrates											
Invertebrates field-collected	Total µg/g	SEM µg/g	Filt µg/L	Nonfilt µg/L	Body					[10]	F
	7,820	6,971	79	11,080	1,382 µg/g						
	583	325	36	698	122 µg/g						
	480	287	16	274	181 µg/g						
	478	251	9	184	266 µg/g						
	128	77	9	58	48 µg/g						
	16	<12	2	35	26 µg/g						
Tubificidae	172 µg/g				17.14 mg/g					[9]	F
	185 µg/g				10.23 mg/g						
	175 µg/g				16.11 mg/g						
	125 µg/g				20.12 mg/g						
	130 µg/g				14.73 mg/g						

Summary of Biological Effects Tissue Concentrations for Copper

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]**
Nereis diversicolor, Polychaete worm	41 µg/g 44 µg/g 52 µg/g 73 µg/g 436 µg/g 591 µg/g 3,020 µg/g		28 µg/g 22 µg/g 33 µg/g 31 µg/g 106 µg/g 257 µg/g 1,142 µg/g					[5]	L
Meretrix casta, Marine clam			201 mg/kg (whole body)[4]	Mortality, ED50				[25]	L; lethal body burden
Mytilus edulis, Mussel			67.4 mg/kg (whole body)[4]	Mortality, ED50				[21]	L; lethal body burden after 7 - 8 days
			67.4 mg/kg (whole body)[4]	Behavior, LOED				[21]	L; total valve closure, increased mucus production, reduced byssus production
			80 mg/kg (whole body)[4]	Mortality, ED100				[26]	L; lethal body burden
			36 mg/kg (whole body)[4]	Mortality, ED100				[26]	L; lethal body burden
			23 mg/kg (whole body)[4]	Mortality, ED100				[26]	L; lethal body burden
			15 mg/kg (whole body)[4]	Mortality, ED100				[26]	L; lethal body burden

Summary of Biological Effects Tissue Concentrations for Copper

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]**
			12 mg/kg (whole body)[4]	Mortality, ED100				[26]	L; lethal body burden
			12 mg/kg (whole body)[4]	Mortality, ED100				[26]	L; lethal body burden
			12 mg/kg (whole body)[4]	Mortality, ED100				[26]	L; lethal body burden
			56 mg/kg (whole body)[4]	Mortality, ED100				[26]	L; lethal body burden
Mytilus galloprovincialis, Mussel			1.9-3.1 mg/kg				0.04	[14]	F
Dreissena polymorpha, Zebra mussel			8.1 mg/kg (whole body)[4]	Physiological; LOED				[24]	L; indicative of breakdown of internal Cu regulatory process
			2.7 mg/kg (whole body)[4]	Physiological, NOED				[24]	L; no effect on internal Cu regulatory process

Summary of Biological Effects Tissue Concentrations for Copper

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]**
Elliptio complanata, Freshwater mussel	0.1-23.7 µg/g		5.4 µg/g (foot) 2.4 µg/g (muscle) 8.5 µg/g (visceral) 29.0 µg/g (hepatopancreas) 29.5 µg/g (gill) 17.6 µg/g (mantle)					[11]	F
	0.1-40.7 µg/g		5.4 µg/g (foot) 2.7 µg/g (muscle) 10.5 µg/g (visceral) 28.8 µg/g (hepatopancreas) 27.8 µg/g (gill) 11.8 µg/g (mantle)						
	0.2-106 µg/g		12.7 µg/g (foot) 11.7 µg/g (muscle) 16.5 µg/g (visceral) 44.5 µg/g (hepatopancreas) 214 µg/g (gill) 94 µg/g (mantle)						

Summary of Biological Effects Tissue Concentrations for Copper

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]**
Elliptio complanata, Freshwater mussel	0.3-142 µg/g		13.1 µg/g (foot) 10.7 µg/g (muscle) 16.1 µg/g (visceral) 72.9 µg/g (hepatopancreas) 132 µg/g (gill) 81.7 µg/g (mantle)						
Unio pictorum, Freshwater mussel			6.5 mg/kg (digestive gland)[4]	Physiological; LOED				[24]	L; indicative of breakdown of internal Cu regulatory process
			10 mg/kg (gill)[4]	Physiological; LOED				[24]	L; indicative of breakdown of internal Cu regulatory process
			4.6 mg/kg (mantle)[4]	Physiological; LOED				[24]	L; indicative of breakdown of internal Cu regulatory process
			2.7 mg/kg (digestive gland)[4]	Physiological; NOED				[24]	L; no effect on internal Cu regulatory process

Summary of Biological Effects Tissue Concentrations for Copper

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]**
			1.9 mg/kg (gill)[4]	Physiological; NOED				[24]	L; no effect on internal Cu regulatory process
			1.7 mg/kg (gonad)[4]	Physiological; NOED				[24]	L; no effect on internal Cu regulatory process
			4 mg/kg (gonad)[4]	Physiological; NOED				[24]	L; no effect on internal Cu regulatory process
			2 mg/kg (kidney)[4]	Physiological; NOED				[24]	L; no effect on internal Cu regulatory process
			3.7 mg/kg (kidney)[4]	Physiological; NOED				[24]	L; no effect on internal Cu regulatory process
			1.1 mg/kg (mantle)[4]	Physiological; NOED				[24]	L; no effect on internal Cu regulatory process

Summary of Biological Effects Tissue Concentrations for Copper

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					5.8 mg/kg (whole body)[4]	Reproduction, ED10				[7]	L; 10% reduction in number of offspring
					68 mg/kg (whole body)[4]	Mortality, ED50				[7]	L; lethal body burden after 21 day exposure
Hyalella azteca, Amphipod		7.7 µg/L			91 µg/g	54% survival				[6]	L
		10.7 µg/L			92 µg/g	50% survival					
		16.7 µg/L			95 µg/g	40% survival					
		25.4 µg/L			88 µg/g	29% survival					
		43.8 µg/L			80 µg/g	6% survival					
		81.3 µg/L			—	0% survival					
Hyalella azteca, Amphipod	Total µg/g	SEM µg/g	Filt µg/L	Nonfilt µg/L	Body;					[10]	F
	7,820	6,971	79	11,080	249 µg/g						
	583	325	36	698	87 µg/g						
	480	287	16	274	124 µg/g						
	478	251	9	184	127 µg/g						
	128	77	9	58	124 µg/g						
	16	<12	2	35	84 µg/g						

Summary of Biological Effects Tissue Concentrations for Copper

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]**
Corophium volutator, Amphipod			16.9 mg/kg (whole body)[4]	NA, LOED				[18]	L; 100% dissolved oxygen saturation during test
Balanus crenatus, Barnacle			80 mg/kg (whole body)[4]	Behavior, NOED				[29]	L; regulation of metals endpoint-summer experiment
Orconectes rusticus, Crayfish			24 mg/kg (abdomen)[4]	Mortality, NOED				[19]	L; no effect on survivorship
			26 mg/kg (abdomen)[4]	Mortality, NOED				[19]	L; no effect on survivorship
			32 mg/kg (abdomen)[4]	Mortality, NOED				[19]	L; no effect on survivorship
			42 mg/kg (abdomen)[4]	Mortality, NOED				[19]	L; no effect on survivorship
			52 mg/kg (abdomen)[4]	Mortality, NOED				[19]	L; no effect on survivorship
			17.8 mg/kg (claw)[4]	Mortality, NOED				[19]	L; no effect on survivorship
			24 mg/kg (claw)[4]	Mortality, NOED				[19]	L; no effect on survivorship
			24 mg/kg (claw)[4]	Mortality, NOED				[19]	L; no effect on survivorship
			30 mg/kg (claw)[4]	Mortality, NOED				[19]	L; no effect on survivorship

Summary of Biological Effects Tissue Concentrations for Copper

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]
			34 mg/kg (claw)[4]	Mortality, NOED				[19]	L; no effect on survivorship
			42 mg/kg (thorax)[4]	Mortality, NOED				[19]	L; no effect on survivorship
			50 mg/kg (thorax)[4]	Mortality, NOED				[19]	L; no effect on survivorship
			56 mg/kg (thorax)[4]	Mortality, NOED				[19]	L; no effect on survivorship
			60 mg/kg (thorax)[4]	Mortality, NOED				[19]	L; no effect on survivorship
			70 mg/kg (thorax)[4]	Mortality, NOED				[19]	L; no effect on survivorship
			2 mg/kg (whole body)[4]	Mortality, NOED				[19]	L; no effect on survivorship
			9 mg/kg (whole body)[4]	Mortality, NOED				[19]	L; no effect on survivorship
			11.2 mg/kg (whole body)[4]	Mortality, NOED				[19]	L; no effect on survivorship
			19.2 mg/kg (whole body)[4]	Mortality, NOED				[19]	L; no effect on survivorship
			26 mg/kg (whole body)[4]	Mortality, NOED				[19]	L; no effect on survivorship

Summary of Biological Effects Tissue Concentrations for Copper

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]**
Chironomus riparius, Midge		0.087 mg/L	500 µg/g		3.77			[4]	F
Chironomus thummi, Midge	12.55 mg/kg		35.7 mg/kg 39.7 mg/kg	Normal larvae Deformed larvae				[8]	F
Chironomus decorus, Midge			1,000 mg/kg (whole body)[4]	Mortality, ED100				[23]	L; 100% mortality
			142 mg/kg (whole body)[4]	Mortality, ED50				[23]	L; ED50
			107 mg/kg (whole body)[4]	Mortality, LOED				[23]	L; significant mortality
			126 mg/kg (whole body)[4]	Mortality, LOED				[23]	L; significant mortality
			86.2 mg/kg (whole body)[4]	Mortality, NOED				[23]	L; no effect on mortality
			130 mg/kg (pupal exuviae)[4]	Development, LOED				[23]	L; increased time to adult emergence by 10 days
			18 mg/kg (whole body)[4]	Development, LOED				[23]	L; increased time to adult emergence by 10 days
			14.8 mg/kg (pupal exuviae)[4]	Development, NOED				[23]	L; no effect on time to adult emergence

Summary of Biological Effects Tissue Concentrations for Copper

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]**
			75.6 mg/kg (pupal exuviae)[4]	Development, NOED				[23]	L; no effect on time to adult emergence
			2.28 mg/kg (whole body)[4]	Development, NOED				[23]	L; no effect on time to adult emergence
			7.2 mg/kg (whole body)[4]	Development, NOED				[23]	L; no effect on time to adult emergence
			13 mg/kg (whole body)[4]	Development, NOED				[23]	L; no effect on time to adult emergence
			7.14 mg/kg (whole body)[4]	Morphology, NOED				[8]	L; 4th instar larvae
Fishes									
Oncorhynchus mykiss, Rainbow trout			40 mg/kg (whole body)[4]	Physiological; LOED				[16]	L; induction of metallothionein
			1.6 mg/kg (whole body)[4]	Mortality, ED100				[17]	L; 100% mortality in non-metallo-thionein-induced fish
			6.8 mg/kg (whole body)[4]	Physiological, LOED				[17]	L; induction of metallothionein
			2.22 mg/kg (whole body)[4]	Mortality, LOED				[20]	L; 50% mortality in 7 hours

Summary of Biological Effects Tissue Concentrations for Copper

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]**
			4.48 mg/kg (whole body)[4]	Survival, LOED				[27]	L
			3.92 mg/kg (whole body)[4]	Not applicable, NOED				[27]	L
Pimephales promelas, Fathead minnow	78.9 µg/g		10.28 mg/g					[9]	F
	110 µg/g		9.32 mg/g						
	125 µg/g		9.13 mg/g						
	130 µg/g		9.70 mg/g						
	130 µg/g		9.86 mg/g						
	172 µg/g		6.92 mg/g						
	175 µg/g		7.28 mg/g						
	175 µg/g		10.96 mg/g						
	185 µg/g		9.37 mg/g						
Cyprinus carpio, Common carp			12.1 mg/kg (whole body)[4]	Morphology, LOED				[29]	L; larval deformation, pH 6.3, body burden from graph
			12.1 mg/kg (whole body)[4]	Morphology, LOED				[29]	L; larval deformation, pH 7.6, body burden from graph
			12.1 mg/kg (whole body)[4]	Mortality, LOED				[29]	L; larval mortality, pH 6.3, body burden from graph

Summary of Biological Effects Tissue Concentrations for Copper

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]**
			12.1 mg/kg (whole body)[4]	Mortality, LOED				[29]	L; larval mortality, pH 7.6, body burden from graph
			24.1 mg/kg (whole body)[4]	Reproduction, LOED				[29]	L; egg mortality, pH 6.3, body burden from graph
			7.62 mg/kg (whole body)[4]	Morphology, NOED				[29]	L; larval deformation, pH 7.6, body burden from graph
			7.62 mg/kg (whole body)[4]	Mortality, NOED				[29]	L; larval mortality, pH 7.6, body burden from graph
			12.1 mg/kg (whole body)[4]	Reproduction, NOED				[29]	L; egg mortality, pH 7.6, body burden from graph
			12.1 mg/kg (whole body)[4]	Reproduction, NOED				[29]	L; egg mortality, pH 6.3, body burden from graph
Lepomis macrochirus, Bluegill			13 mg/kg (gill)[4]	Growth, LOED				[15]	L; duration = 22 months or 660 days

Summary of Biological Effects Tissue Concentrations for Copper

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]**
			44 mg/kg (kidney)[4]	Growth, LOED				[15]	L; duration = 22 months or 660 days
			480 mg/kg (liver)[4]	Growth, LOED				[15]	L; duration = 22 months or 660 days
			13 mg/kg (gill)[4]	Mortality, LOED				[15]	L; duration = 22 months or 660 days
			44 mg/kg (kidney)[4]	Mortality, LOED				[15]	L; duration = 22 months or 660 days
			480 mg/kg (liver)[4]	Mortality, LOED				[15]	L; duration = 22 months or 660 days
			13 mg/kg (gill)[4]	Reproduction, LOED				[15]	L; duration = 22 months or 660 days
			44 mg/kg (kidney)[4]	Reproduction, LOED				[15]	L; duration = 22 months or 660 days
			480 mg/kg (liver)[4]	Reproduction, LOED				[15]	L; duration = 22 months or 660 days
			6 mg/kg (gill)[4]	Growth, NOED				[15]	L; duration = 22 months or 660 days
			12 mg/kg (kidney)[4]	Growth, NOED				[15]	L; duration = 22 months or 660 days

Summary of Biological Effects Tissue Concentrations for Copper

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]
			57 mg/kg (liver)[4]	Growth, NOED				[15]	L; duration = 22 months or 660 days
			6 mg/kg (gill)[4]	Mortality, NOED				[15]	L; duration = 22 months or 660 days
			12 mg/kg (kidney)[4]	Mortality, NOED				[15]	L; duration = 22 months or 660 days
			57 mg/kg (liver)[4]	Mortality, NOED				[15]	L; duration = 22 months or 660 days
			6 mg/kg (gill)[4]	Reproduction, NOED				[15]	L; duration = 22 months or 660 days
			12 mg/kg (kidney)[4]	Reproduction, NOED				[15]	L; duration = 22 months or 660 days
			57 mg/kg (liver)[4]	Reproduction, NOED				[15]	L; duration = 22 months or 660 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: POLYCHLORINATED DIBENZO-*p*-DIOXINS

Chemical Name (Common Synonyms): **CASRN:** 35822-46-9
1,2,3,4,6,7,8-HEPTACHLORO DIBENZO-*p*-DIOXIN

Chemical Characteristics

Solubility in Water: No data [1], 2.4 mg/L [2]

Half-Life: No data [2,3]

Log K_{ow}: No data [4], 8.00 [2]

Log K_{oc}: 7.86 L/kg organic carbon

Human Health

Oral RfD: No data [5]

Confidence: —

Critical Effect: —

Oral Slope Factor: No data [5]

Carcinogenic Classification: —

Wildlife

Partitioning Factors: Partitioning factors for 1,2,3,4,6,7,8-heptaCDD in wildlife were not found in the studies reviewed.

Food Chain Multipliers: Limited information was found on specific biomagnification factors of PCDDs and PCDFs through terrestrial wildlife. Due to the toxicity, high K_{ow} values, and highly persistent nature of the PCDDs and PCDFs, they possess a high potential to bioaccumulate and biomagnify through the food web. PCDDs and PCDFs have been identified in fish and wildlife throughout the global aquatic and marine environments [6]. Studies conducted in Lake Ontario indicated that biomagnification of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD) appears to be significant between fish and fish-eating birds but not between fish and their food. When calculated for older predaceous fish such as lake-trout-eating young smelt, the biomagnification factor (BMF) can equal 3. The log BMF from alewife to herring gulls in Lake Ontario was 1.51 for 2,3,7,8-TCDD [7]. Log BMFs of 1.18 to 1.70 were determined for mink fed 1,2,3,4,6,7,8-Hepta CDD in the diet [18].

EPA has developed risk-based concentrations of 2,3,7,8-TCDD in different media that present low and high risk to fish, mammalian, and avian wildlife. These concentrations were developed based on toxic effects of 2,3,7,8-TCDD and its propensity to bioaccumulate in fish, mammals, and birds.

Environmental Concentrations Associated With 2,3,7,8-TCDD Risk to Aquatic Life and Associated Wildlife [8]

Organism	Fish Concentration (pg/g)	Sediment Concentration (pg/g dry wt.)	Water Concentration (pg/L)	
			POC=0.2	POC=1.0
Low Risk				
Fish	50	60	0.6	3.1
Mammalian Wildlife	0.7	2.5	0.008	0.04
Avian Wildlife	6	21	0.07	0.35
High Risk to Sensitive Species				
Fish	80	100	1.0	5
Mammalian Wildlife	7	25	0.08	0.4
Avian Wildlife	60	210	0.7	3.5

Note: POC - Particulate organic carbon
Fish lipid of 8% and sediment organic carbon of 3% assumed where needed.
For risk to fish, BSAF of 0.3 used; for risk to wildlife, BSAF of 0.1 used.
Low risk concentrations are derived from no-effects thresholds for reproductive effects (mortality in embryos and young) in sensitive species.
High risk concentrations are derived from TCDD doses expected to cause 50 to 100% mortality in embryos and young of sensitive species.

Aquatic Organisms

Partitioning Factors: In one study, the BSAF for carp collected from a reservoir in central Wisconsin was 0.0048. In a laboratory study, log BCFs for fathead minnow, rainbow trout, and goldfish exposed to 1,2,3,4,6,7,8-HeptaCDD were 2.71, 3.15, and 4.28, respectively.

Food Chain Multipliers: No specific food chain multipliers were identified for 1,2,3,4,6,7,8-heptaCDD. Food chain multiplier information was only available for 2,3,7,8-TCDD. Biomagnification of 2,3,7,8-TCDD does not appear to be significant between fish and their prey. Limited data for the base of the Lake Ontario lake trout food chain indicated little or no biomagnification between zooplankton and forage fish. BMFs based on fish consuming invertebrate species are probably close to 1.0 because of the 2,3,7,8-TCDD biotransformation by forage fish. BMFs greater than 1.0 may exist between some zooplankton species and their prey due to the lack of 2,3,7,8-TCDD biotransformation in invertebrates [8].

Toxicity/Bioaccumulation Assessment Profile

The polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) each consist of 75 isomers that differ in the number and position of attached chlorine atoms. The PCDDs and PCDFs are polyhalogenated aromatic compounds and exhibit several properties common to this group of compounds. These compounds tend to be highly lipophilic and the degree of lipophilicity is increased with increasing ring chlorination [6]. In general, the PCDDs and PCDFs exhibit relative inertness to acids, bases, oxidation, reduction, and heat, increasing in environmental persistence and chemical

stability with increasing chlorination [9,6]. Because of their lipophilic nature, the PCDDs and PCDFs have been detected in fish, wildlife, and human adipose tissue, milk, and serum [6].

Each isomer has its own unique chemical and toxicological properties. The most toxic of the PCDD and PCDF isomers is 2,3,7,8-TCDD, one of the 22 possible congeners of tetrachlorodibenzo-*p*-dioxin [9]. Toxicity equivalency factors (TEFs) have been developed by EPA relating the toxicities of other PCDD and PCDF isomers to that of 2,3,7,8-TCDD [10]. The biochemical mechanisms leading to the toxic response resulting from exposure to PCDDs and PCDFs are not known in detail, but experimental data suggest that an important role in the development of systemic toxicity resulting from exposure to these chemicals is played by an intracellular protein, the Ah receptor. This receptor binds halogenated polycyclic aromatic molecules, including PCDDs and PCDFs. In several mouse strains, the expression of toxicity of 2,3,7,8-TCDD-related compounds, including cleft palate formation, liver damage, effects on body weight gain, thymic involution, and chloracnegenic response, has been correlated with their binding affinity for the Ah receptor, and with their ability to induce several enzyme systems [10].

Toxicity Equivalency Factors (TEF) for PCDD and PCDF Isomers [10]

Isomer	TEF
Total TetraCDD	1
2,3,7,8-TCDD	1
Other TCDDs	0.01
Total PentaCDDs	0.5
2,3,7,8-PentaCDDs	0.5
Other PentaCDDs	0.005
Total HexaCDDs	0.04
2,3,7,8-HexaCDDs	0.04
Other HexaCDDs	0.0004
Total HeptaCDDs	0.001
2,3,7,8-HeptaCDDs	0.001
Other HeptaCDDs	0.00001
Total TetraCDFs	0.1
2,3,7,8-TetraCDF	0.1
Other TetraCDFs	0.001
Total PentaCDFs	0.1
2,3,7,8-PentaCDFs	0.1
Other PentaCDFs	0.001
Total HexaCDFs	0.01
2,3,7,8-HexaCDFs	0.01
Other HexaCDFs	0.0001
Total HeptaCDFs	0.001
2,3,7,8-HeptaCDFs	0.001
Other HeptaCDFs	0.00001

In natural systems, PCDDs and PCDFs are typically associated with sediments, biota, and the organic carbon fraction of ambient waters [8]. Congener-specific analyses have shown that the 2,3,7,8-substituted PCDDs and PCDFs were the major compounds present in most sample extracts [6]. Results from limited epidemiology studies are consistent with laboratory-derived threshold levels to 2,3,7,8-TCDD impairment of reproduction in avian wildlife. Population declines in herring gulls (*Larus argentatus*) on Lake Ontario during the early 1970s coincided with egg concentrations of 2,3,7,8-TCDD and related chemicals expected to cause reproductive failure based on laboratory experiments (2,3,7,8-TCDD concentrations in excess of 1,000 pg/g). Improvements in herring gull reproduction through the mid-1980s were correlated with declining 2,3,7,8-TCDD concentrations in eggs and lake sediments [8]. Based on limited information on isomer-specific analysis from animals at different trophic levels, it appears that at higher trophic levels, i.e., fish-eating birds and fish, there is a selection of the planar congeners with the 2,3,7,8-substituted positions [11].

PCDDs and PCDFs are accumulated by aquatic organisms through exposure routes that are determined by the habitat and physiology of each species. With log K_{ow}>5, exposure through ingestion of contaminated food becomes an important route for uptake in comparison to respiration of water [8]. The relative contributions of water, sediment, and food to uptake of 2,3,7,8-TCDD by lake trout in Lake Ontario were examined by exposing yearling lake trout to Lake Ontario smelt and sediment from Lake Ontario along with water at a 2,3,7,8-TCDD concentration simulated to be at equilibrium with the sediments. Food ingestion was found to contribute approximately 75 percent of total 2,3,7,8-TCDD [8]. There have been a number of bioconcentration studies of 2,3,7,8-TCDD using model ecosystem and single species exposure. Although there is variation in the actual BCF values, in general, the algae and plants have the lowest BCF values, on the order of a few thousand. A log BCF value of 4.38 has been reported for the snail *Physa* sp. Crustacea and insect larvae appear to have the next highest BCF values, followed by several species of fish, with the highest log BCF value of 4.79 [11].

Exposure of juvenile rainbow trout to 2,3,7,8-TCDD and -TCDF in water for 28 days resulted in adverse effects on survival, growth, and behavior at extremely low concentrations. A no-observed-effects concentration (NOEC) for 2,3,7,8-TCDD could not be determined because the exposure to the lowest dose of 0.038 ng/l resulted in significant mortality [12]. A number of biological effects have been reported in fish following exposure to 2,3,7,8-TCDD including enzyme induction, immunological effects, wasting syndrome, dermatological effects, hepatic effects, hematological effects, developmental effects, and cardiovascular effects [11].

Summary of Biological Effects Tissue Concentrations for 1,2,3,4,6,7,8-HeptaCDD

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									
Salmonids							0.0031	[20]	F
Oncorhynchus mukiss (Salmo gairderni), Rainbow trout		Exposure water 11-55 ng/L			3.15 ± 2.35			[16]	L
Oncorhynchus mukiss (Salmo gairderni), Rainbow trout			0.000035 mg/kg (liver)[4]	Biochemical, LOED				[19]	L; significant increase in liver ethoxyresorufin O-deethylase (EROD)
Cyprinus carpio, Carp	2,190 pg/g[5]		27 pg/g[5]				0.0048	[13]	F; Petenwell Reservoir, central Wisconsin; BSAF based on 8% tissue lipid content and 3.1% sediment organic carbon

Summary of Biological Effects Tissue Concentrations for 1,2,3,4,6,7,8-HeptaCDD

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]**
Carassius auratus, Goldfish			1.91/2.2 ng/g[5] (whole body)		4.28			[15]	L; fish were exposed for 120 hr; exposure water contained fly ash extract; concentrations were measured in water, but data were not presented
Pimephales promelas, Fathead minnow		Exposure water 8-39 ng/L			2.71 ± 2.03			[16]	L
Platycephalus caerulopunctatus and *Platycephalus bassensis,* Flathead	0.356 pg/g, dw		558 pg/kg					[14]	F; unimpacted coastal site; surface sediment composite; most other dioxin congeners were below detection.
Sillago bassensis, School whiting	0.356 pg/g, dw		375 pg/kg						
Wildlife									
Falco peregrinus, Peregrine falcon			0.7 ng/g (eggs) (n = 6)	11.4% eggshell thinning				[17]	F; Kola Peninsula, Russia

Summary of Biological Effects Tissue Concentrations for 1,2,3,4,6,7,8-HeptaCDD

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]**
Mustela vison, Mink	Diet: 5 pg/g[5]		115 pg/g[5] (liver)	NOAEL		log BMF = 1.18		[18]	L; BMF = lipid-normalized concentration in the liver divided by the lipid-normalized dietary concentration
	7 pg/g[5]		330 pg/g[5] (liver)	LOAEL; reduced kit body weights followed by reduced survival		log BMF = 1.70			
	6 pg/g[5]		290 pg/g[5] (liver)	Reduced kit body weights followed by reduced survival		log BMF = 1.69			
	13 pg/g[5]		380 pg/g[5] (liver)	Significant decrease in number of live kits whelped per female		log BMF = 1.66			

[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.
[5] Not clear from reference if concentration is based on wet or dry weight.

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Chemical Category: POLYCHLORINATED DIBENZO-*p*-DIOXINS

Chemical Name (Common Synonyms): **CASRN:** 39227-28-6
1,2,3,4,7,8-HEXACHLORODIBENZO-*p*-DIOXIN

Chemical Characteristics

Solubility in Water: No data [1], 8.25×10^{-6} mg/L [1,2]

Half-Life: No data [2,3]

Log K_{ow}: No data [4], 7.70 [2]

Log K_{oc}: 7.57 L/kg organic carbon

Human Health

Oral RfD: No data [5]

Confidence: —

Critical Effect: —

Oral Slope Factor (Reference): No data [5]

Carcinogenic Classification: —

Wildlife

Partitioning Factors: Partitioning factors for 1,2,3,4,7,8-hexaCDD in wildlife were not found in the studies reviewed.

Food Chain Multipliers: Limited information was found reporting on specific biomagnification factors of PCDDs and PCDFs through terrestrial wildlife. Due to the toxicity, high K_{ow} values, and highly persistent nature of the PCDDs and PCDFs, they possess a high potential to bioaccumulate and biomagnify through the food web. PCDDs and PCDFs have been identified in fish and wildlife throughout the global aquatic and marine environments [6]. Studies conducted in Lake Ontario indicated that biomagnification of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD) appears to be significant between fish and fish-eating birds but not between fish and their food. When calculated for older predaceous fish such as lake-trout-eating young smelt, the biomagnification factor (BMF) can equal 3. The BMF from alewife to herring gulls in Lake Ontario was 32 for 2,3,7,8-TCDD [7]. A log BMF of 0.97 was reported for mink exposed to 1,2,3,4,7,8-hexaCDD in the diet. [14].

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Environmental Concentrations Associated With 2,3,7,8-TCDD Risk to Aquatic Life and Associated Wildlife [8]

Organism	Fish Concentration (pg/g)	Sediment Concentration (pg/g dry wt.)	Water Concentration (pg/L)	
			POC=0.2	POC=1.0
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High risk concentrations are derived from TCDD doses expected to cause 50 to 100% mortality in embryos and young of sensitive species.

Aquatic Organisms

Partitioning Factors: In a laboratory study, log BCFs for rainbow trout and fathead minnow exposed to 1,2,3,4,7,8-HexaCDD were 3.73 and 4.00, respectively.

Food Chain Multipliers: No specific food chain multipliers were identified for 1,2,3,4,7,8-hexaCDD. Food chain multiplier information was only available for 2,3,7,8-TCDD. Biomagnification of 2,3,7,8-TCDD does not appear to be significant between fish and their prey. Limited data for the base of the Lake Ontario lake trout food chain indicated little or no biomagnification between zooplankton and forage fish. BMFs based on fish consuming invertebrate species are probably close to 1.0 because of the 2,3,7,8-TCDD biotransformation by forage fish. BMFs greater than 1.0 may exist between some zooplankton species and their prey due to the lack of 2,3,7,8-TCDD biotransformation in invertebrates [8].

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Summary of Biological Effects Tissue Concentrations for 1,2,3,4,7,8-HexaCDD

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									
Oncorhynchus mukiss (Salmo gairderni), Rainbow trout		Exposure water 10-47 ng/L			3.73			[11]	L
Oncorhynchus mukiss, Rainbow trout			0.0000395 mg/kg (liver)[4]	Biochemical, LOED				[15]	L; significant increase in liver ethoxyresorufin O-deethylase (EROD)
Pimephales promelas, Fathead minnow		Exposure water 10-47 ng/L			4.00			[11]	L
Wildlife									
Falco peregrinus, Peregrine falcon			3.3 ng/g (eggs) (n = 6)	11.4% eggshell thinning				[13]	F; Kola Peninsula, Russia

Summary of Biological Effects Tissue Concentrations for 1,2,3,4,7,8-HexaCDD

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]**
Mustela vison, Mink	Diet: 2 pg/g[5]		6 pg/g[5] (liver)	LOAEL; reduced kit body weights followed by reduced survival		No BMF reported		[14]	L; BMF = biomagnification factor = v_l/v_d v_l = lipid-normalized tissue concentration, v_d = lipid-normalized dietary concentration.
	1 pg/g[5]		77 pg/g[5] (liver)	Reduced kit body weights followed by reduced survival		No BMF reported			
	3 pg/g[5]		15 pg/g[5] (liver)	Significant decrease in number of live kits whelped per female		Log BMF = 0.97			

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.
5 Not clear from reference if concentration is based on wet or dry weight.

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Chemical Category: POLYCHLORINATED DIBENZO-*p*-DIOXINS

Chemical Name (Common Synonyms): **CASRN:** 57653-85-7
1,2,3,6,7,8-HEXACHLORODIBENZO-*p*-DIOXIN

Chemical Characteristics

Solubility in Water: No data [1,2]

Half-Life: No data [2,3]

Log K_{ow}: No data [2,4]

Log K_{oc}: —

Human Health

Oral RfD: No data [5]

Confidence: —

Critical Effect: Hepatic tumors in mice and rats

Oral Slope Factor: $6.2 \times 10^{+3}$ per (mg/kg)/day [5]

Carcinogenic Classification: B2 [5]

Wildlife

Partitioning Factors: Partitioning factors for 1,2,3,6,7,8-hexaCDD in wildlife were not found in the studies reviewed.

Food Chain Multipliers: Limited information was found reporting on specific biomagnification factors of PCDDs and PCDFs through terrestrial wildlife. Due to the toxicity, high K_{ow} values, and highly persistent nature of the PCDDs and PCDFs, they possess a high potential to bioaccumulate and biomagnify through the food web. PCDDs and PCDFs have been identified in fish and wildlife throughout the global aquatic and marine environments [6]. Studies conducted in Lake Ontario indicated that biomagnification of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD) appears to be significant between fish and fish-eating birds but not between fish and their food. When calculated for older predaceous fish such as lake-trout-eating young smelt, the biomagnification factor (BMF) can equal 3. The BMF from alewife to herring gulls in Lake Ontario was 32 for 2,3,7,8-TCDD [7]. Log BMFs of 1.42 and 1.43 were reported for mink exposed to 1,2,3,6,7,8-hexaCDD in the diet [18].

EPA has developed risk-based concentrations of 2,3,7,8-TCDD in different media that present low and high risk to fish, mammalian, and avian wildlife. These concentrations were developed based on toxic effects of 2,3,7,8-TCDD and its propensity to bioaccumulate in fish, mammals, and birds.

Environmental Concentrations Associated With 2,3,7,8-TCDD Risk to Aquatic Life and Associated Wildlife [8]

Organism	Fish Concentration (pg/g)	Sediment Concentration (pg/g dry wt.)	Water Concentration (pg/L)	
			POC=0.2	POC=1.0
Low Risk				
Fish	50	60	0.6	3.1
Mammalian Wildlife	0.7	2.5	0.008	0.04
Avian Wildlife	6	21	0.07	0.35
High Risk to Sensitive Species				
Fish	80	100	1.0	5
Mammalian Wildlife	7	25	0.08	0.4
Avian Wildlife	60	210	0.7	3.5

Note: POC - Particulate organic carbon
Fish lipid of 8% and sediment organic carbon of 3% assumed where needed.
For risk to fish, BSAF of 0.3 used; for risk to wildlife, BSAF of 0.1 used.
Low risk concentrations are derived from no-effects thresholds for reproductive effects (mortality in embryos and young) in sensitive species.
High risk concentrations are derived from TCDD doses expected to cause 50 to 100% mortality in embryos and young of sensitive species.

Aquatic Organisms

Partitioning Factors: In one study, the BSAF for carp collected from a reservoir in central Wisconsin was 0.035. The log BCF for goldfish during a laboratory exposure for 120 hours was 4.61.

Food Chain Multipliers: No specific food chain multipliers were identified for 1,2,3,6,7,8-hexaCDD. Food chain multiplier information was only available for 2,3,7,8-TCDD. Biomagnification of 2,3,7,8-TCDD does not appear to be significant between fish and their prey. Limited data for the base of the Lake Ontario lake trout food chain indicated little or no biomagnification between zooplankton and forage fish. BMFs based on fish consuming invertebrate species are probably close to 1.0 because of the 2,3,7,8-TCDD biotansformation by forage fish. BMFs greater than 1.0 may exist between some zooplankton species and their prey due to the lack of 2,3,7,8-TCDD biotransformation in invertebrates[8].

Toxicity/Bioaccumulation Assessment Profile

The polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) each consist of 75 isomers that differ in the number and position of attached chlorine atoms. The PCDDs and PCDFs are polyhalogenated aromatic compounds and exhibit several properties common to this group of compounds. These compounds tend to be highly lipophilic and the degree of lipophilicity is increased with increasing ring chlorination [6]. In general, the PCDDs and PCDFs exhibit relative inertness to acids, bases, oxidation, reduction, and heat, increasing in environmental persistence and chemical

stability with increasing chlorination [9,6]. Because of their lipophilic nature, the PCDDs and PCDFs have been detected in fish, wildlife, and human adipose tissue, milk, and serum [6].

Each isomer has its own unique chemical and toxicological properties. The most toxic of the PCDD and PCDF isomers is 2,3,7,8 TCDD, one of the 22 possible congeners of tetrachlorodibenzo-*p*-dioxin [9]. Toxicity equivalency factors (TEFs) have been developed by EPA relating the toxicities of other PCDD and PCDF isomers to that of 2,3,7,8-TCDD [10]. The biochemical mechanisms leading to the toxic response resulting from exposure to PCDDs and PCDFs are not known in detail, but experimental data suggest that an important role in the development of systemic toxicity resulting from exposure to these chemicals is played by an intracellular protein, the Ah receptor. This receptor binds halogenated polycyclic aromatic molecules, including PCDDs and PCDFs. In several mouse strains, the expression of toxicity of 2,3,7,8-TCDD-related compounds, including cleft palate formation, liver damage, effects on body weight gain, thymic involution, and chloracnegenic response, has been correlated with their binding affinity for the Ah receptor, and with their ability to induce several enzyme systems [10].

Toxicity Equivalency Factors (TEF) for PCDD and PCDF Isomers [10]

Isomer	TEF
Total TetraCDD	1
2,3,7,8-TCDD	1
Other TCDDs	0.01
Total PentaCDDs	0.5
2,3,7,8-PentaCDDs	0.5
Other PentaCDDs	0.005
Total HexaCDDs	0.04
2,3,7,8-HexaCDDs	0.04
Other HexaCDDs	0.0004
Total HeptaCDDs	0.001
2,3,7,8-HeptaCDDs	0.001
Other HeptaCDDs	0.00001
Total TetraCDFs	0.1
2,3,7,8-TetraCDF	0.1
Other TetraCDFs	0.001
Total PentaCDFs	0.1
2,3,7,8-PentaCDFs	0.1
Other PentaCDFs	0.001
Total HexaCDFs	0.01
2,3,7,8-HexaCDFs	0.01
Other HexaCDFs	0.0001
Total HeptaCDFs	0.001
2,3,7,8-HeptaCDFs	0.001
Other HeptaCDFs	0.00001

In natural systems, PCDDs and PCDFs are typically associated with sediments, biota, and the organic carbon fraction of ambient waters [7]. Congener-specific analyses have shown that the 2,3,7,8-substituted PCDDs and PCDFs were the major compounds present in most sample extracts [6]. Results from limited epidemiology studies are consistent with laboratory-derived threshold levels to 2,3,7,8-TCDD impairment of reproduction in avian wildlife. Population declines in herring gulls (*Larus argentatus*) on Lake Ontario during the early 1970s coincided with egg concentrations of 2,3,7,8-TCDD and related chemicals expected to cause reproductive failure based on laboratory experiments (2,3,7,8-TCDD concentrations in excess of 1,000 pg/g). Improvements in herring gull reproduction through the mid-1980s were correlated with declining 2,3,7,8-TCDD concentrations in eggs and lake sediments [8]. Based on limited information on isomer-specific analysis from animals at different trophic levels, it appears that at higher trophic levels, i.e., fish-eating birds and fish, there is a selection of the planar congeners with the 2,3,7,8-substituted positions [11].

PCDDs and PCDFs are accumulated by aquatic organisms through exposure routes that are determined by the habitat and physiology of each species. With log K_{ow}>5, exposure through ingestion of contaminated food becomes an important route for uptake in comparison to respiration of water [8]. The relative contributions of water, sediment, and food to uptake of 2,3,7,8-TCDD by lake trout in Lake Ontario were examined by exposing yearling lake trout to Lake Ontario smelt and sediment from Lake Ontario along with water at a 2,3,7,8-TCDD concentration simulated to be at equilibrium with the sediments. Food ingestion was found to contribute approximately 75 percent of total 2,3,7,8-TCDD [8]. There have been a number of bioconcentration studies of 2,3,7,8-TCDD using model ecosystem and single species exposure. Although there is variation in the actual BCF values, in general, the algae and plants have the lowest BCF values, on the order of a few thousand. A log value of 4.38 has been reported for the snail *Physa* sp. Crustacea and insect larvae appear to have the next highest BCF values, followed by several species of fish, with the highest log BCF value of 4.79 [11].

Exposure of juvenile rainbow trout to 2,3,7,8-TCDD and -TCDF in water for 28 days resulted in adverse effects on survival, growth, and behavior at extremely low concentrations. A no-observed-effects concentration (NOEC) for 2,3,7,8-TCDD could not be determined because the exposure to the lowest dose of 0.038 ng/l resulted in significant mortality [12]. A number of biological effects have been reported in fish following exposure to 2,3,7,8-TCDD including enzyme induction, immunological effects, wasting syndrome, dermatological effects, hepatic effects, hematological effects, developmental effects, and cardiovascular effects [11].

Summary of Biological Effects Tissue Concentrations for 1,2,3,6,7,8-HexaCDD

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									
Carassius auratus, Goldfish			0.79 ng/g[4] (whole body)		4.61			[14]	L; fish were exposed for 120 hr; exposure water contained fly ash extract; concen-trations were measured in water, but data were not presented
Cyprinus carpio, Carp	180 pg/g[4]		16 pg/g[4]				0.035	[13]	F; Petenwell Reservoir, central Wisconsin; BSAF based on 8% tissue lipid content and 3.1% sediment organic carbon
Salmonids							0.0073	[19]	F

Summary of Biological Effects Tissue Concentrations for 1,2,3,6,7,8-HexaCDD

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]**
Wildlife									
Falco peregrinus, Peregrine falcon			7.2 ng/g (eggs) (n = 6)	11.4% eggshell thinning				[17]	F; Kola Peninsula, Russia
Haliaeetus leucocephalus, Bald eagle chicks			Powell River site: ~9,000 ng/kg lipid weight basis (yolk sac) Reference site: ~500 ng/kg lipid weight basis (yolk sac)	A hepatic cytochrome P4501A cross-reactive protein (CYP1A) was induced nearly 6-fold in chicks from Powell River site compared to the reference ($p < 0.05$). No significant concentration-related effects were found for morphological, physiological, or histological parameters.				[15]	F; southern coast of British Columbia; eggs were collected from nests and hatched in the lab; ~ indicates value was taken from a figure

Summary of Biological Effects Tissue Concentrations for 1,2,3,6,7,8-HexaCDD

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]**
Ardea herodias, Great blue heron chicks			Nicomekl site: 10±3.4 ng/kg (egg) (n=11)					[16]	L; eggs were collected from three British Columbia colonies with different levels of contamination and incubated in the laboratory
			Vancouver site: 89±45.4 ng/kg (egg) (n=12)	Depression of growth compared to Nicomekl site. Presence of edema.					
			Crofton site: 430±105.9 ng/kg (egg) (n=6)	Depression of growth compared to Nicomekl site. Presence of edema.					

Summary of Biological Effects Tissue Concentrations for 1,2,3,6,7,8-HexaCDD

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]**
Mustela vison, Mink	Diet: 1 pg/g[4]		54 pg/g[4] (liver)	LOAEL; reduced kit body weights followed by reduced survival		No BMF reported		[18]	L; BMF = biomagnification factor = v_l/v_d, v_l = lipid-normalized tissue concentration, v_d = lipid-normalized dietary concentration.
	3 pg/g[4]		77 pg/g[4] (liver)	Reduced kit body weights followed by reduced survival		log BMF = 1.42			
	6 pg/g[4]		130 pg/g[4] (liver)	Significant decrease in number of live kits whelped per female		log BMF = 1.53			

[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] Not clear from reference if concentration is based on wet or dry weight.

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Chemical Category: POLYCHLORINATED DIBENZO-*p*-DIOXINS

Chemical Name (Common Synonyms): **CASRN:** 40321-76-4
1,2,3,7,8-PENTACHLORODIBENOZ-*p*-DIOXIN

Chemical Characteristics

Solubility in Water: No data [1,3]

Half-Life: No data [2,3]

Log K_{ow}: No data [3,4]

Log K_{oc}: —

Human Health

Oral RfD: No data [5]

Confidence: —

Critical Effect: —

Oral Slope Factor: No data [5]

Carcinogenic Classification: —

Wildlife

Partitioning Factors: Partitioning factors for 1,2,3,7,8-pentaCDD in wildlife were not found in the studies reviewed.

Food Chain Multipliers: Limited information was found reporting on specific biomagnification factors of PCDDs and PCDFs through terrestrial wildlife; no information was available for 1,2,3,7,8-pentaCDD, specifically. Due to the toxicity, high K_{ow} values, and highly persistent nature of the PCDDs and PCDFs, they possess a high potential to bioaccumulate and biomagnify through the food web. PCDDs and PCDFs have been identified in fish and wildlife throughout the global aquatic and marine environments [6]. Studies conducted in Lake Ontario indicated that biomagnification of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD) appears to be significant between fish and fish-eating birds but not between fish and their food. When calculated for older predaceous fish such as lake-trout-eating young smelt, the log biomagnification factor (BMF) can equal 0.48. The log BMF from alewife to herring gulls in Lake Ontario was 1.51 for 2,3,7,8-TCDD [7].

EPA has developed risk-based concentrations of 2,3,7,8-TCDD in different media that present low and high risk to fish, mammalian, and avian wildlife. These concentrations were developed based on toxic effects of 2,3,7,8-TCDD and its propensity to bioaccumulate in fish, mammals, and birds.

Environmental Concentrations Associated With 2,3,7,8-TCDD Risk to Aquatic Life and Associated Wildlife [8]

Organism	Fish Concentration (pg/g)	Sediment Concentration (pg/g dry wt.)	Water Concentration (pg/L)	
			POC=0.2	POC=1.0
Low Risk				
Fish	50	60	0.6	3.1
Mammalian Wildlife	0.7	2.5	0.008	0.04
Avian Wildlife	6	21	0.07	0.35
High Risk to Sensitive Species				
Fish	80	100	1.0	5
Mammalian Wildlife	7	25	0.08	0.4
Avian Wildlife	60	210	0.7	3.5

Note: POC - Particulate organic carbon
Fish lipid of 8% and sediment organic carbon of 3% assumed where needed.
For risk to fish, BSAF of 0.3 used; for risk to wildlife, BSAF of 0.1 used.
Low risk concentrations are derived from no-effects thresholds for reproductive effects (mortality in embryos and young) in sensitive species.
High risk concentrations are derived from TCDD doses expected to cause 50 to 100% mortality in embryos and young of sensitive species.

Aquatic Organisms

Partitioning Factors: Partitioning factors for 1,2,3,7,8-pentaCDF in aquatic organisms were not found in the studies reviewed.
Food Chain Multipliers: No specific food chain multipliers were identified for 1,2,3,7,8-pentaCDD. Food chain multiplier information was only available for 2,3,7,8-TCDD. Biomagnification of 2,3,7,8-TCDD does not appear to be significant between fish and their prey. Limited data for the base of the Lake Ontario lake trout food chain indicated little or no biomagnification between zooplankton and forage fish. BMFs based on fish consuming invertebrate species are probably close to 1.0 because of the 2,3,7,8-TCDD biotransformation by forage fish. BMFs greater than 1.0 might exist between some zooplankton species and their prey due to the lack of 2,3,7,8-TCDD biotransformation in invertebrates[8].

Toxicity/Bioaccumulation Assessment Profile

The polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) each consist of 75 isomers that differ in the number and position of attached chlorine atoms. The PCDDs and PCDFs are polyhalogenated aromatic compounds and exhibit several properties common to this group of compounds. These compounds tend to be highly lipophilic and the degree of lipophilicity is increased with increasing ring chlorination [6]. In general, the PCDDs and PCDFs exhibit relative inertness to acids, bases, oxidation, reduction, and heat, increasing in environmental persistence and chemical stability with increasing chlorination [6,9]. Because of their lipophilic nature, the PCDDs and PCDFs have been detected in fish, wildlife, and human adipose tissue, milk, and serum [6].

Each isomer has its own unique chemical and toxicological properties. The most toxic of the PCDD and PCDF isomers is 2,3,7,8-TCDD, one of the 22 possible congeners of tetrachlorodibenzo-*p*-dioxin [9]. Toxicity equivalency factors (TEFs) have been developed by the U.S. EPA relating the toxicities of other PCDD and PCDF isomers to that of 2,3,7,8-TCDD [10]. The biochemical mechanisms leading to the toxic response resulting from exposure to PCDDs and PCDFs are not known in detail, but experimental data suggest that an important role in the development of systemic toxicity resulting from exposure to these chemicals is played by an intracellular protein, the Ah receptor. This receptor binds halogenated polycyclic aromatic molecules, including PCDDs and PCDFs. In several mouse strains, the expression of toxicity of 2,3,7,8-TCDD-related compounds, including cleft palate formation, liver damage, effects on body weight gain, thymic involution, and chloracnegenic response, has been correlated with their binding affinity for the Ah receptor, and with their ability to induce several enzyme systems [10].

Toxicity Equivalency Factors (TEF) for PCDD and PCDF Isomers [10]

Isomer	TEF
Total TetraCDD	1
2,3,7,8-TCDD	1
Other TCDDs	0.01
Total PentaCDDs	0.5
2,3,7,8-PentaCDDs	0.5
Other PentaCDDs	0.005
Total HexaCDDs	0.04
2,3,7,8-HexaCDDs	0.04
Other HexaCDDs	0.0004
Total HeptaCDDs	0.001
2,3,7,8-HeptaCDDs	0.001
Other HeptaCDDs	0.00001
Total TetraCDFs	0.1
2,3,7,8-TetraCDF	0.1
Other TetraCDFs	0.001
Total PentaCDFs	0.1
2,3,7,8-PentaCDFs	0.1
Other PentaCDFs	0.001
Total HexaCDFs	0.01
2,3,7,8-HexaCDFs	0.01
Other HexaCDFs	0.0001
Total HeptaCDFs	0.001
2,3,7,8-HeptaCDFs	0.001
Other HeptaCDFs	0.00001

In natural systems, PCDDs and PCDFs are typically associated with sediments, biota, and the organic carbon fraction of ambient waters [8]. Congener-specific analyses have shown that the 2,3,7,8-substituted PCDDs and PCDFs were the major compounds present in most sample extracts [6]. Results

from limited epidemiology studies are consistent with laboratory-derived threshold levels to 2,3,7,8-TCDD impairment of reproduction in avian wildlife. Population declines in herring gulls (*Larus argentatus*) on Lake Ontario during the early 1970s coincided with egg concentrations of 2,3,7,8-TCDD and related chemicals expected to cause reproductive failure based on laboratory experiments (2,3,7,8-TCDD concentrations in excess of 1,000 pg/g). Improvements in herring gull reproduction through the mid-1980s were correlated with declining 2,3,7,8-TCDD concentrations in eggs and lake sediments [8]. Based on limited information on isomer-specific analysis from animals at different trophic levels, it appears that at higher trophic levels, i.e., fish-eating birds and fish, there is a selection of the planar congeners with the 2,3,7,8-substituted positions [11].

PCDDs and PCDFs are accumulated by aquatic organisms through exposure routes that are determined by the habitat and physiology of each species. With log K_{ow}>5, exposure through ingestion of contaminated food becomes an important route for uptake in comparison to respiration of water [8]. The relative contributions of water, sediment, and food to uptake of 2,3,7,8-TCDD by lake trout in Lake Ontario were examined by exposing yearling lake trout to Lake Ontario smelt and sediment from Lake Ontario along with water at a 2,3,7,8-TCDD concentration simulated to be at equilibrium with the sediments. Food ingestion was found to contribute approximately 75 percent of total 2,3,7,8-TCDD [8]. There have been a number of bioconcentration studies of 2,3,7,8-TCDD using model ecosystem and single species exposure. Although there is variation in the actual log BCF values, in general, the algae and plants have the lowest BCF values, on the order of a few thousand. A log BCF value of 4.38 has been reported for the snail *Physa* sp. Crustacea and insect larvae appear to have the next highest BCF values, followed by several species of fish, with the highest log BCF value of 4.78 [11].

Exposure of juvenile rainbow trout to 2,3,7,8-TCDD and -TCDF in water for 28 days resulted in adverse effects on survival, growth, and behavior at extremely low concentrations. A no-observed-effects concentration (NOEC) for 2,3,7,8-TCDD could not be determined because the exposure to the lowest dose of 0.038 ng/l resulted in significant mortality [12]. A number of biological effects have been reported in fish following exposure to 2,3,7,8-TCDD including enzyme induction, immunological effects, wasting syndrome, dermatological effects, hepatic effects, hematological effects, developmental effects, and cardiovascular effects [11].

Summary of Biological Effects Tissue Concentrations for 1,2,3,7,8-PentaCDD

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									
Carassius auratus, Goldfish			1.59/2.61 ng/g[4] (whole body)		16,982			[14]	L; fish were exposed for 120 hr; exposure water contained fly ash extract; concentrations were measured in water, but data were not presented
Cyprinus carpio, Carp	31 pg/g[4]		4.8 pg/g[4]		0.06			[13]	F; Petenwell Reservoir, central Wisconsin; BSAF based on 8% tissue lipid content and 3.1% sediment organic carbon
Salmonids							0.054	[18]	F
Wildlife									
Falco peregrinus, Peregrine falcon			11 ng/g (eggs) (n = 6)	11.4% eggshell thinning				[17]	F; Kola Peninsula, Russia

Summary of Biological Effects Tissue Concentrations for 1,2,3,7,8-PentaCDD

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]**
Haliaeetus leucocephalus, Bald eagle chicks			Powell River site: ~2,800 ng/kg lipid weight basis (yolk sac) Reference site: ~500 ng/kg lipid weight basis (yolk sac)	A hepatic cytochrome P4501A cross-reactive protein (CYP1A) was induced nearly six-fold in chicks from Powell River site compared to the reference ($p < 0.05$). No significant concentration-related effects were found for morphological, physiological, or histological parameters.				[15]	F; southern coast of British Columbia; eggs were collected from nests and hatched in the lab; ~ indicates value was taken from a figure.

Summary of Biological Effects Tissue Concentrations for 1,2,3,7,8-PentaCDD

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]**
Ardea herodias, Great blue heron chicks			Nicomekl site: 6±2.2 ng/kg (egg) (n = 11)					[16]	L; eggs were collected from three British Columbia colonies with different levels of contamination and incubated in the laboratory
			Vancouver site: 57±25.8 ng/kg (egg) (n = 12)	Depression of growth compared to Nicomekl site. Presence of edema.					
			Crofton site: 263±69.9 ng/kg (egg) (n = 6)	Depression of growth compared to Nicomekl site. Presence of edema.					

[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] Not clear from reference if concentration is based on wet or dry weight.

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Chemical Category: POLYCHLORINATED DIBENZO-*p*-DIOXINS

Chemical Name (Common Synonyms): **CASRN:** 1746-01-6
2,3,7,8-TETRACHLORODIBENZO-*p*-DIOXIN

Chemical Characteristics

Solubility in Water: 19.3 ng/L [1]

Half-Life: 1.1.15 - 1.62 years based on soil die-away test and lake water and sediment die-away test [2]

Log K_{ow}: 6.53 [3]

Log K_{oc}: 6.42 L/kg organic carbon

Human Health

Oral RfD: No data [4]

Confidence: —

Critical Effect: —

Oral Slope Factor: 1.5 x 10^{+5} per (mg/kg)/day [4]

Carcinogenic Classification: B2 [4]

Wildlife

Partitioning Factors: Partitioning factors for 2,3,7,8-TCDD in wildlife were not found in the literature.

Food Chain Multipliers: Limited information was found reporting on specific biomagnification factors of PCDDs and PCDFs through terrestrial wildlife. Due to the toxicity, high K_{ow} values, and highly persistent nature of the PCDDs and PCDFs, they possess a high potential to bioaccumulate and biomagnify through the food web. PCDDs and PCDFs have been identified in fish and wildlife throughout the global aquatic and marine environments [5]. Studies conducted in Lake Ontario indicated that biomagnification of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD) appears to be significant between fish and fish-eating birds but not between fish and their food. When calculated for older predaceous fish such as lake-trout-eating young smelt, the biomagnification factor (BMF) can equal 3. The BMF from alewife to herring gulls in Lake Ontario was 32 for 2,3,7,8-TCDD [6].

EPA has developed risk-based concentrations of 2,3,7,8-TCDD in different media that present low and high risk to fish, mammalian, and avian wildlife. These concentrations were developed based on toxic effects of 2,3,7,8-TCDD and its propensity to bioaccumulate in fish, mammals, and birds.

Environmental Concentrations Associated With 2,3,7,8-TCDD Risk to Aquatic Life and Associated Wildlife [7]

Organism	Fish Concentration (pg/g)	Sediment Concentration (pg/g dry wt.)	Water Concentration (pg/L)	
			POC=0.2	POC=1.0
Low Risk				
Fish	50	60	0.6	3.1
Mammalian Wildlife	0.7	2.5	0.008	0.04
Avian Wildlife	6	21	0.07	0.35
High Risk to Sensitive Species				
Fish	80	100	1.0	5
Mammalian Wildlife	7	25	0.08	0.4
Avian Wildlife	60	210	0.7	3.5

Note: POC - Particulate organic carbon
Fish lipid of 8% and sediment organic carbon of 3% assumed where needed.
For risk to fish, BSAF of 0.3 used; for risk to wildlife, BSAF of 0.1 used.
Low risk concentrations are derived from no-effects thresholds for reproductive effects (mortality in embryos and young) in sensitive species.
High risk concentrations are derived from TCDD doses expected to cause 50 to 100% mortality in embryos and young of sensitive species.

Aquatic Organisms

Partitioning Factors: Steady-state BSAFs for invertebrates exposed to 2,3,7,8-TCDD in the laboratory ranged from about 0.5 to 0.9 [8]. The BSAF for carp collected from a reservoir in central Wisconsin was 0.27 [9].

Food Chain Multipliers: Biomagnification of 2,3,7,8-TCDD does not appear to be significant between fish and their prey. Limited data for the base of the Lake Ontario lake trout food chain indicated little or no biomagnification between zooplankton and forage fish. BMFs based on fish consuming invertebrate species are probably close to 1.0 because of the 2,3,7,8-TCDD biotansformation by forage fish. BMFs greater than 1.0 may exist between some zooplankton species and their prey due to the lack of 2,3,7,8-TCDD biotransformation in invertebrates [7].

Toxicity/Bioaccumulation Assessment Profile

The polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) each consist of 75 isomers that differ in the number and position of attached chlorine atoms. The PCDDs and PCDFs are polyhalogenated aromatic compounds and exhibit several properties common to this group of compounds. These compounds tend to be highly lipophilic and the degree of lipophilicity is increased with increasing ring chlorination [5]. In general, the PCDDs and PCDFs exhibit relative inertness to acids, bases, oxidation, reduction, and heat, increasing in environmental persistence and chemical stability with increasing chlorination [10,5]. Because of their lipophilic nature, the PCDDs and PCDFs have been detected in fish, wildlife, and human adipose tissue, milk, and serum [5].

Each isomer has its own unique chemical and toxicological properties. The most toxic of the PCDD and PCDF isomers is 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD), one of the 22 possible congeners of tetrachlorodibenzo-*p*-dioxin [10]. Toxicity equivalency factors (TEFs) have been developed by the EPA relating the toxicities of other PCDD and PCDF isomers to that of 2,3,7,8-TCDD [11]. The biochemical mechanisms leading to the toxic response resulting from exposure to PCDDs and PCDFs are not known in detail, but experimental data suggest that an important role in the development of systemic toxicity resulting from exposure to these chemicals is played by an intracellular protein, the Ah receptor. This receptor binds halogenated polycyclic aromatic molecules, including PCDDs and PCDFs. In several mouse strains, the expression of toxicity of 2,3,7,8-TCDD-related compounds, including cleft palate formation, liver damage, effects on body weight gain, thymic involution, and chloracnegenic response, has been correlated with their binding affinity for the Ah receptor, and with their ability to induce several enzyme systems [11].

Toxicity Equivalency Factors (TEF) for PCDD and PCDF Isomers [11]

Isomer	TEF
Total TetraCDD	1
2,3,7,8-TCDD	1
Other TCDDs	0.01
Total PentaCDDs	0.5
2,3,7,8-PentaCDDs	0.5
Other PentaCDDs	0.005
Total HexaCDDs	0.04
2,3,7,8-HexaCDDs	0.04
Other HexaCDDs	0.0004
Total HeptaCDDs	0.001
2,3,7,8-HeptaCDDs	0.001
Other HeptaCDDs	0.00001
Total TetraCDFs	0.1
2,3,7,8-TetraCDF	0.1
Other TetraCDFs	0.001
Total PentaCDFs	0.1
2,3,7,8-PentaCDFs	0.1
Other PentaCDFs	0.001
Total HexaCDFs	0.01
2,3,7,8-HexaCDFs	0.01
Other HexaCDFs	0.0001
Total HeptaCDFs	0.001
2,3,7,8-HeptaCDFs	0.001
Other HeptaCDFs	0.00001

In natural systems, PCDDs and PCDFs are typically associated with sediments, biota, and the organic carbon fraction of ambient waters [7]. Congener-specific analyses have shown that the 2,3,7,8-

substituted PCDDs and PCDFs were the major compounds present in most sample extracts [5]. Results from limited epidemiology studies are consistent with laboratory-derived threshold levels to 2,3,7,8-TCDD impairment of reproduction in avian wildlife. Population declines in herring gulls (*Larus argentatus*) on Lake Ontario during the early 1970s coincided with egg concentrations of 2,3,7,8-TCDD and related chemicals expected to cause reproductive failure based on laboratory experiments (2,3,7,8-TCDD concentrations in excess of 1,000 pg/g). Improvements in herring gull reproduction through the mid-1980s were correlated with declining 2,3,7,8-TCDD concentrations in eggs and lake sediments [7]. Based on limited information on isomer-specific analysis from animals at different trophic levels, it appears that at higher trophic levels, i.e., fish-eating birds and fish, there is a selection of the planar congeners with the 2,3,7,8-substituted positions [12].

PCDDs and PCDFs are accumulated by aquatic organisms through exposure routes that are determined by the habitat and physiology of each species. With log K_{ow}>5, exposure through ingestion of contaminated food becomes an important route for uptake in comparison to respiration of water [7]. The relative contributions of water, sediment, and food to uptake of 2,3,7,8-TCDD by lake trout in Lake Ontario were examined by exposing yearling lake trout to Lake Ontario smelt and sediment from Lake Ontario along with water at a 2,3,7,8-TCDD concentration simulated to be at equilibrium with the sediments. Food ingestion was found to contribute approximately 75 percent of total 2,3,7,8-TCDD [7]. There have been a number of bioconcentration studies of 2,3,7,8-TCDD using model ecosystem and single species exposure. Although there is variation in the actual BCF values, in general, the algae and plants have the lowest BCF values, on the order of a few thousand. A log BCF value of 4.38 has been reported for the snail *Physa* sp. Crustacea and insect larvae appear to have the next highest BCF values, followed by several species of fish, with the highest log BCF value of 4.79 [12].

Exposure of juvenile rainbow trout to 2,3,7,8-TCDD and -TCDF in water for 28 days resulted in adverse effects on survival, growth, and behavior at extremely low concentrations. A no-observed-effects concentration (NOEC) for 2,3,7,8-TCDD could not be determined because the exposure to the lowest dose of 0.038 ng/l resulted in significant mortality [13]. A number of biological effects have been reported in fish following exposure to 2,3,7,8-TCDD including enzyme induction, immunological effects, wasting syndrome, dermatological effects, hepatic effects, hematological effects, developmental effects, and cardiovascular effects [12].

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDD

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]**
Plants									
Oedogonium cardiacum, Green algae			1.34 mg/kg (whole body)[4]	Growth, NOED				[35]	L; no effect on growth
Lemna minor, Duckweed			0.00614 mg/kg (whole body)[4]	Growth, NOED				[35]	L; no observed effect
Invertebrates									
Nereis virens, Sandworm	656±97 pg/g dw; (n = 6)		422±103 pg/g dw (whole body)				~0.5	[8,14]	L; 180-day exposure; sediment TOC was 57 mg/kg; ~ indicates approximate value, as numbers were estimated from bar graphs.
Physa sp., Snail			0.364 mg/kg (whole body)[4]	Mortality, NOED				[35]	L; no effect on survival

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDD

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]**
Macoma nasuta, Clam	656±97 pg/g dw; (n = 6)		142 ± 20 pg/g dw				~0.9	[8,14]	L; 120-day exposure; sediment TOC was 57 mg/kg; ~ indicates approximate value, as numbers were estimated from bar graphs.
Daphnia magna, Cladaceran			2.08 mg/kg (whole body)[4]	Mortality, NOED				[35]	L; no effect on survival
Palaemonetes pugio, Grass shrimp	656±97 pg/g dw; (n = 6)		138 ± 20 pg/g dw				~0.7	[8,14]	L; 28-day exposure; sediment TOC was 57 mg/kg ~ indicates approximate value, as numbers were estimated from bar graphs.
Pacifastacus leniusculus, Crayfish			0.003 mg/kg (whole body)[4]	Mortality, ED25				[31]	L; 25% mortality after 40 days
			0.03 mg/kg (whole body)[4]	Mortality, ED50				[31]	L; lethargy, 50% to 66% increase in mortality

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDD

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]**
			0.003 mg/kg (whole body)[4]	Behavior, LOED				[31]	L; lack of avoidance response
			0.003 mg/kg (whole body)[4]	Physiological, LOED				[31]	L; significant induction of cytochrome P450
			0.003 mg/kg (whole body)[4]	Physiological, LOED				[31]	L; significant induction of liver enzymes (cytochrome P450)
			0.1 mg/kg (whole body)[4]	Morphology, NOED				[31]	L; no significant pathology at highest dose
			0.0003 mg/kg (whole body)[4]	Mortality, NOED				[31]	L; no effect on mortality
			0.0003 mg/kg (whole body)[4]	Physiological, NOED				[31]	L; no significant induction of liver enzymes (cytochrome P450)
Callinectes sapidus, Blue crab	32.2 ppt[5] (TOC = 3.2%) 52.8 ppt[5] (TOC = 3.9%)		8.2 ppt[5] (hepatopancreas) (% lipid = 7.6)			-0.72	0.089	[15]	F; northeastern Florida; bleach-kraft paper mill receiving stream; BAF and BSAF calculated using mean of two sediment concentrations.

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDD

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]**
Chironomus tentans, Midge			0.47 mg/kg (whole body)	Growth, NOED				[44]	L; concentrations are lipid
Fishes									
Oncorhynchus mykiss (Salmo gairdneri), Rainbow trout		water exposure 0.038 ng/L	1.0 µg/kg[5]	28-day LOEC (survival, growth)	4.41			[13]	L
		water exposure 0.382 ng/L	10.95 ng/g[5] (whole body)		4.46				L; 6-hour exposure period
Oncorhynchus mykiss, Rainbow trout			0.00388 mg/kg (extractable lipid)[4]	Growth, LOED				[32]	L; reduced growth, exposed fish weighed 50 g vs. 130 g for controls
			0.00371 mg/kg (liver)[4]	Growth, LOED				[32]	L; reduced growth, exposed fish weighed 50 g vs. 130 g for controls
			0.00026 mg/kg (muscle)[4]	Growth, LOED				[32]	L; reduced growth, exposed fish weighed 50 g vs. 130 g for controls

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDD

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]
			0.00065 mg/kg (whole body)[4]	Growth, LOED				[32]	L; reduced growth, exposed fish weighed 50 g vs. 130 g for controls
			0.00388 mg/kg (etractable lipid)[4]	Morphology, LOED				[32]	L; livers enlarged to nearly twice the size of control fish livers, fin rot
			0.00371 mg/kg (liver)[4]	Morphology, LOED				[32]	L; livers enlarged to nearly twice the size of control fish livers, fin rot
			0.00026 mg/kg (muscle)[4]	Morphology, LOED				[32]	L; livers enlarged to nearly twice the size of control fish livers, fin rot
			0.00065 mg/kg (whole body)[4]	Morphology, LOED				[32]	L; livers enlarged to nearly twice the size of control fish livers, fin rot
			0.00388 mg/kg (extractable lipid)[4]	Mortality, LOED				[32]	L; lethal to 7 of 90 fish over 139 days
			0.00371 mg/kg (liver)[4]	Mortality, LOED				[32]	L; lethal to 7 of 90 fish over 139 days
			0.00026 mg/kg (muscle)[4]	Mortality, LOED				[32]	L; lethal to 7 of 90 fish over 139 days

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDD

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]**
			0.00065 mg/kg (whole body)[4]	Mortality, LOED				[32]	L; lethal to 7 of 90 fish over 139 days
Oncorhynchus mykiss, Rainbow trout			0.01 mg/kg (whole body)[4]	Mortality, ED50				[36]	L; 80-day LD50 for mortality
			0.001 mg/kg (whole body)[4]	Growth, LOED				[36]	L; reduction in body weight
			0.025 mg/kg (whole body)[4]	Morphology, LOED				[36]	L; fin necrosis, hyperpigmentation
Oncorhynchus mykiss, Rainbow trout			0.000315 mg/kg (carcass)[4]	Growth, NOED				[38]	L; no effect on growth
			0.000102 mg/kg (gastrointestinal tract)[4]	Growth, NOED				[38]	L; no effect on growth
			0.000244 mg/kg (gill)[4]	Growth, NOED				[38]	L; no effect on growth
			0.00007 mg/kg (heart)[4]	Growth, NOED				[38]	L; no effect on growth
			0.000092 mg/kg (kidney)[4]	Growth, NOED				[38]	L; no effect on growth
			0.000072 mg/kg (liver)[4]	Growth, NOED				[38]	L; no effect on growth
			0.000355 mg/kg (pyloric caeca)[4]	Growth, NOED				[38]	L; no effect on growth

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDD

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]**
			0.000029 mg/kg (skeletal muscle)[4]	Growth, NOED				[38]	L; no effect on growth
			0.000201 mg/kg (skin)[4]	Growth, NOED				[38]	L; no effect on growth
			0.000085 mg/kg (spleen)[4]	Growth, NOED				[38]	L; no effect on growth
			0.00327 mg/kg (visceral fat)[4]	Growth, NOED				[38]	L; no effect on growth
			0.00025 mg/kg (whole body)[4]	Growth, NOED				[38]	L; no effect on growth
			0.000315 mg/kg (carcass)[4]	Morphology, NOED				[38]	L; no effect on fin necrosis or hemorrhage
			0.000102 mg/kg (gastrointestinal tract)[4]	Morphology, NOED				[38]	L; no effect on fin necrosis or hemorrhage
			0.000244 mg/kg (gill)[4]	Morphology, NOED				[38]	L; no effect on fin necrosis or hemorrhage
			0.00007 mg/kg (heart)[4]	Morphology, NOED				[38]	L; no effect on fin necrosis or hemorrhage
			0.000092 mg/kg (kidney)[4]	Morphology, NOED				[38]	L; no effect on fin necrosis or hemorrhage

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDD

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]
			0.000072 mg/kg (liver)[4]	Morphology, NOED				[38]	L; no effect on fin necrosis or hemorrhage
			0.000355 mg/kg (pyloric caeca)[4]	Morphology, NOED				[38]	L; no effect on fin necrosis or hemorrhage
			0.000029 mg/kg (skeletal muscle)[4]	Morphology, NOED				[38]	L; no effect on fin necrosis or hemorrhage
			0.000201 mg/kg (skin)[4]	Morphology, NOED				[38]	L; no effect on fin necrosis or hemorrhage
			0.000085 mg/kg (spleen)[4]	Morphology, NOED				[38]	L; no effect on fin necrosis or hemorrhage
			0.00327 mg/kg (visceral fat)[4]	Morphology, NOED				[38]	L; no effect on fin necrosis or hemorrhage
			0.00025 mg/kg (whole body)[4]	Morphology, NOED				[38]	L; no effect on fin necrosis or hemorrhage
			0.000315 mg/kg (carcass)[4]	Mortality, NOED				[38]	L; no effect on mortality
			0.000102 mg/kg (gastrointestinal tract)[4]	Mortality, NOED				[38]	L; no effect on mortality

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDD

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]**
			0.000244 mg/kg (gill)[4]	Mortality, NOED				[38]	L; no effect on mortality
			0.00007 mg/kg (heart)[4]	Mortality, NOED				[38]	L; no effect on mortality
			0.000092 mg/kg (kidney)[4]	Mortality, NOED				[38]	L; no effect on mortality
			0.000072 mg/kg (liver)[4]	Mortality, NOED				[38]	L; no effect on mortality
			0.000355 mg/kg (pyloric caeca)[4]	Mortality, NOED				[38]	L; no effect on mortality
			0.000029 mg/kg (skeletal muscle)[4]	Mortality, NOED				[38]	L; no effect on mortality
			0.000201 mg/kg (skin)[4]	Mortality, NOED				[38]	L; no effect on mortality
			0.000085 mg/kg (spleen)[4]	Mortality, NOED				[38]	L; no effect on mortality
			0.00327 mg/kg (visceral fat)[4]	Mortality, NOED				[38]	L; no effect on mortality
			0.00025 mg/kg (whole body)[4]	Mortality, NOED				[38]	L; no effect on mortality
			0.00452 mg/kg (whole body)[4]	Survival, ED50				[13]	L; exposure concentration is the mean of measured TCDD concentration

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDD

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			0.0000047 mg/kg (liver)[4]	Biochemical, LOED				[40]	L; significant increase in liver ethoxyresorufin O-deethylase (EROD)
			0.000038 mg/kg (liver)[4]	Biochemical, LOED				[40]	L; significant increase in liver ethoxyresorufin O-deethylase (EROD)
			0.000016 mg/kg (liver)[4]	Biochemical, LOED				[40]	L; significant increase in liver ethoxyresorufin O-deethylase (EROD)
Oncorhynchus mykiss, Rainbow trout			0.000439 mg/kg (whole body)[4]	Mortality, ED50				[42]	L; mortality from fertilization to swim-up; exposure dose calculated from text; residue measured in egg at 5-days post exposure; dosed for 48 hours and endpoint measured after approximately 24 days

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDD

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]**
			0.000421 mg/kg (whole body)[4]	Mortality, ED50				[42]	L; mortality from fertilization to swim-up; liposome used to carry dose; 93% of dose retained in egg and assumed to be in swim-up fry, flow rate = 8-12
			0.000279 mg/kg (whole body)[4]	Mortality, LOED				[42]	L; significant increase in mortality from hatch to swim-up at lowest exposure concentration tested; exposure dose calculated from text; residue measured in egg at 5-days post exposure; dosed for 48 hours and endpoint measured after approximately 24 days

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDD

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]**
			0.000437 mg/kg (whole body)[4]	Mortality, LOED				[42]	L; significant increase in mortality from hatch to swim-up; liposome used to carry dose; 93% of dose retained in egg and assumed to be in swim-up fry, flow rate = 8-12
			0.000291 mg/kg (whole body)[4]	Mortality, NOED				[42]	L; no significant increase in mortality from hatch to swim-up; liposome used to carry dose; 93% of dose retained in egg and assumed to be in swim-up fry, flow rate = 8 to 12
Oncorhynchus mykiss, Rainbow trout			0.00017 mg/kg (whole body)[4]	Mortality, ED50				[43]	L; estimated LD50s for 6 strains of rainbow trout, orig_con_wet ranged from 170 to 488; used low value; exposure concentration = 170 to 488

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDD

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]**
Salmo trutta, Brown trout			5.2 pg/g[5] (fillet)			4.25-4.45		[22]	F; locations throughout Maine; a range of mean BAFs is presented; the values are means for locations throughout Maine, and the range is for BAFs calculated using river concentrations from years prior to the sampling date to account for declines in paper mill discharges
Salvelinus fontinalis, Brook trout			0.0006 mg/kg (whole body)[4]	Physiological				[33]	L; induction of hepatic EROD

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDD

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]**
Salvelinus fontinalis, Brook trout			0.0012 mg/kg (whole body)[4]	Growth, NOED				[41]	L; no significant growth effect at highest target body burden; TCDD-spiked diet to produce desired body burden; abstract with minimal information
			0.0012 mg/kg (whole body)[4]	Mortality, NOED				[41]	L; no significant mortality at highest target body burden; TCDD-spiked diet to produce desired body burden; abstract with minimal information
			0.0012 mg/kg (whole body)[4]	Reproduction, LOED				[41]	L; significant delay in spawning; TCDD-spiked diet to produce desired body burden; abstract with minimal information

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDD

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]**
			0.0006 mg/kg (whole body)[4]	Reproduction, NOED				[41]	L; no delay in spawning; TCDD-spiked diet to produce desired body burden; abstract with minimal information
Salvelinus fontinalis, Brook trout			0.0002 mg/kg (whole body)[4]	Mortality, ED50				[43]	L; estimated LD50
Amia calva, Bowfin			11.2 ppt[5] (liver) (n = 1)			-0.59	0.180	[15]	F; northeastern Florida; bleached-kraft paper mill receiving stream; BAF and BSAF calculated using mean of two sediment concen-trations. BAF = (pg TCDD/g tissue) ÷ (pg TCDD / g sediment); BSAF = (pg TCDD/g lipid) ÷ (pg TCDD / g TOC).
			18.6 ppt[5] (liver) (n = 1)			-0.36	0.255		
			46.1 ppt[5] (ovary) (n=1)			0.03	0.281		

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDD

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 kisutch, Coho salmon			0.000478 mg/kg (whole body)[4]	Growth, LOED				[39]	L; reduced growth
			0.000478 mg/kg (whole body)[4]	Mortality, LOED				[39]	L; reduced survival
			0.00217 mg/kg (whole body)[4]	Growth, NA				[39]	L; reduced growth
			0.00217 mg/kg (whole body)[4]	Mortality, NA				[39]	L; reduced survival
			0.000125 mg/kg (whole body)[4]	Behavior, NOED				[39]	L; no effect on food consumption or feeding
			0.000125 mg/kg (whole body)[4]	Growth, NOED				[39]	L; no effect on growth
Carp, (scientific name unknown)		water exposure 62 pg/L	2.2 µg/kg[5]	Death (71 days)				[16]	L
Salvelinus namaycush, Lake trout, (early life stage)		water exposure 20 ng/L	0.055 µg/kg[5] (egg)	48-hour LOEC (mortality)				[21]	L

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDD

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]**
		water exposure 10 ng/L	0.034 µg/kg[5] (egg)	48-hour NOEC (mortality)				[21]	L
		water exposure 62 ng/L	0.226 µg/kg[5] (egg)	48-hour LOEC (hatchability)				[21]	L
Salvelinus namaycush, Lake trout			0.000065 mg/kg (whole body)[4]	Mortality, ED50				[15]	L; lethal to 50% of sac fry
			0.000055 mg/kg (whole body)[4]	Mortality, LOED				[15]	L; lowest statistically significant increase in mortality of sac fry
			0.000226 mg/kg (whole body)[4]	Reproduction, L				[15]	L; reduced hatchability of eggs
			0.000035 mg/kg (whole body)[4]	Mortality, NOED				[15]	L; no effect on mortality of sac fry
Salvelinus namaycush, Lake trout			0.000044 mg/kg (whole body)[4]	Mortality, ED50				[34]	L; LD50 for sac fry mortality

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDD

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]**
Salvelinus namaycush, Lake trout			0.000065 mg/kg (whole body)[4]	Mortality, ED50				[42]	L; mortality from fertilization to swim-up; exposure dose calculated from text; residue measured in egg at 5-days post exposure; dosed for 48 hours and endpoint measured after approximately 24 days
			0.000055 mg/kg (whole body)[4]	Mortality, LOED				[42]	L; significant increase in mortality from hatch to swim-up; exposure dose calculated from text; residue measured in egg at 5-days post exposure; dosed for 48 hours and endpoint measured after approximately 24 days

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDD

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]**
			0.000058 mg/kg (whole body)[4]	Mortality, LOED				[42]	L; significant increase in mortality from hatch to swim-up; high control mortality; liposome used to carry dose; 93% of dose retained in egg and assumed to be in swim-up fry, flow rate = 8-12
			0.000034 mg/kg (whole body)[4]	Mortality, NOED				[42]	L; no significant increase in mortality from hatch to swim-up; exposure dose calculated from text; residue measured in egg at 5-days post exposure; dosed for 48 hours and endpoint measured after approximately 24 days

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDD

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]**
			0.000044 mg/kg (whole body)[4]	Mortality, NOED				[42]	L; no significant increase in mortality from hatch to swim-up; high control mortality; liposome used to carry dose; 93% of dose retained in egg and assumed to be in swim-up fry, flow rate = 8 to 12
Salvelinus namaycush, Lake trout			0.000065 mg/kg (whole body)[4]	Mortality, ED50				[43]	L; estimated LD50
Carassius auratus, Goldfish			0.58-0.63 ng/g[5] (whole body)		4.39			[18]	L; fish were exposed for 120 hr; exposure water contained fly ash extract; concentrations were measured in water, but data were not presented

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDD

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, Carp	170 pg/g[5]		120 pg/g[5]				0.27	[9]	F; Petenwell Reservoir, central Wisconsin; BSAF based on 8% tissue lipid content and 3.1% sediment organic carbon
Cyprinus carpio, Carp			0.0022 mg/kg (whole body)[4]	Behavior, LOED				[15]	L; difficulty swimming
			0.0022 mg/kg (whole body) [4]	Cellular, LOED				[15]	L; edema, body wall ulcers
			0.0022 mg/kg (whole body)[4]	Morphology, LOED				[15]	L; fin erosion, hemorrhage, morphologically resembling Blue Sac Disease
Cyprinus carpio, Carp			0.003 mg/kg (whole body)[4]	Mortality, ED50				[36]	L; 80-day LD50 for mortality
Cyprinus carpio, Carp			0.0022 mg/kg (whole body)[4]	Mortality, LOED				[15]	L; increased mortality

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDD

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]**
Danio rerio, Zebrafish			2.16 ng/g (egg)	ED50 (pericardial edema)				[23]	L; newly fertilized eggs were exposed for 1 hr to water containing graded concentrations of TCDD
			2.43 ng/g (egg)	ED50 (yolk sac edema)					
			2.45 ng/g (egg)	LD50					
Bracydanio rerio, Zebrafish			8.3 µg/kg[5]	LOEC (reproduction)				[24]	L; food exposure
			8.3 µg/kg[5]	LOEC (oogenesis)				[24]	L; food exposure
Pimephales promelas, Fathead minnow			17-2,042 µg/kg[5]	LD100				[17]	L; food exposure

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDD

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]**
Catostomus commerson, White sucker			9.6 pg/g[5] (whole body)			4.89-5.03		[22]	F; locations throughout Maine; a range of mean BAFs is presented; the values are means for locations throughout Maine, and the range is for BAFs calculated using river concentrations from years prior to the sampling date to account for declines in paper mill discharges

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDD

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 nebulosus, Brown bullhead catfish	32.2 -52.8 ppt[5] (TOC = 3.2-3.9%)		1.8 ppt[5] (liver) (% lipid = 3.5)			-1.40	0.043	[15]	F; northeastern Florida; bleached-kraft paper mill receiving stream; BAF and BSAF calculated using mean of two sediment concen-trations. BAF = (pg TCDD/g tissue) ÷ (pg TCDD/ g sediment); BSAF = (pg TCDD/g lipid) ÷ (pg TCDD / g TOC).
			2.6 ppt[5] (liver) (% lipid = 2.9)			-1.22	0.074		
			2.8 ppt[5] (liver) (% lipid = 3.2)			-1.15	0.073		
Ictalurus melas, Black bullhead			0.005 mg/kg (whole body)[4]	Mortality, ED50				[36]	L; 80 day LD50 for mortality
			0.025 mg/kg (whole body)[4]	Morphology, LOED				[36]	L; fin necrosis, hyperpigmentation

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDD

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			0.0044 mg/kg (whole body)[4]	Mortality, ED10				[45]	L; radiolabelled compounds in sediment, compound leached into water for exposure; all fish died between days 14 and 15; body residues from dead fish
Gambusia affinis, Mosquito fish			0.0072 mg/kg (whole body)[4]	Mortality, ED10				[45]	L; radiolabelled compounds in sediment, compound leached into water for exposure; all fish died between days 14 and 15; body residues from dead fish
Oryzias latipes, Japanese medaka		Water exposure 2.2 ng/L	0.24 µg/kg[5] (embryo)	Lesions in embryos				[19]	L

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDD

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]**
Oryzias latipes, Japanese medaka (juveniles)		water exposure 101 ± 26 pg/L (n = 23)	2,408 ±241 pg/g	Obvious signs of TCDD toxicity such as generalized edema, fin erosion, and discoloration in many of the exposed fish	4.38 non-steady state 5.71 predicted steady state			[20]	L; 12-day exposure period; lipid content 7.5%
Oryzias latipes, Japanese medaka			0.24 mg/kg (whole body)[4]	Lesions, ED50				[19]	L; ten replicates per treatment
			0.3 mg/kg (whole body)[4]	Lesions, LOED				[19]	L; 50% of embryos with lesions but no statistical significance analyzed; ten replicates per treatment
			0.1 mg/kg (whole body)[4]	Lesions, NOED				[19]	L; no significant incidence of lesions at lowest doseage tested; 10 replicates per treatment, resd_conc_wet value > 0.1

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDD

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]**
Morone americana, White perch			1.2 pg/g[5]			3.48-3.88		[22]	F; locations throughout Maine; a range of mean BAFs is presented; the values are means for locations throughout Maine, and the range is for BAFs calculated using river concentrations from years prior to the sampling date to account for declines in paper mill discharges
Lepomis macrochirus, Bluegill			0.016 mg/kg (whole body)[4]	Mortality, ED50				[36]	L; 80-day LD50 for mortality
			0.005 mg/kg (whole body)[4]	Growth, LOED				[36]	L; reduction in body weight
			0.025 mg/kg (whole body)[4]	Morphology, LOED				[36]	L; fin necrosis, hyperpigmentation

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDD

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]**
Microperus dolomieu, Smallmouth bass			3.4 pg/g[5] (fillet)			4.06-4.39		[22]	F; locations throughout Maine; a range of mean BAFs is presented; the values are means for locations throughout Maine, and the range is for BAFs calculated using river concentrations from years prior to the sampling date to account for declines in paper mill discharges

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDD

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]**
Micropterus samoides, Largemouth bass	32.2 ppt[5] (TOC=3.2%)		1.8 ppt[5] (liver) (% lipid =3.9)			-1.40	0.038	[15]	F; northeastern Florida; bleached kraft paper mill receiving stream; BAF and BSAF calculated using mean of two sediment concen-trations. BAF = (pg TCDD / g tissue) ÷ (pg TCDD/ g sediment); BSAF = (pg TCDD/g lipid) ÷ (pg TCDD/ g TOC).
	52.8 ppt[5] (TOC=3.9%)		2.9 ppt[5] (liver) (% lipid =2.4)			-1.15	0.100		
			8.8 ppt[5] (ovary) (% lipid =7.6)			-0.68	0.096		
Micropterus salmoides, Largemouth bass			0.011 mg/kg (whole body)[4]	Mortality, ED50				[36]	L; 80-day LD50 For Mortality
			0.025 mg/kg (whole body)[4]	Morphology, LOED				[36]	L; Fin Necrosis, Hyperpigment-ation
Perca flavescens, Yellow perch			0.003 mg/kg (whole body)[4]	Mortality, ED50				[36]	L; 80-day LD50 for mortality
			0.005 mg/kg (whole body)[4]	Growth, LOED				[36]	L; reduction in body weight

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDD

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]
			0.025 mg/kg (whole body)[4]	Morphology, LOED				[36]	L; fin necrosis, hyperpigmentation
			0.000129 mg/kg (carcass)[4]	Growth, NOED				[37]	L; no effect on growth
			0.000148 mg/kg (gastrointestinal tract)[4]	Growth, NOED				[37]	L; no effect on growth
			0.000155 mg/kg (gill)[4]	Growth, NOED				[37]	L; no effect on growth
			0.000077 mg/kg (heart)[4]	Growth, NOED				[37]	L; no effect on growth
			0.000119 mg/kg (kidney)[4]	Growth, NOED				[37]	L; no effect on growth
			0.000466 mg/kg (liver)[4]	Growth, NOED				[37]	L; no effect on growth
			0.000143 mg/kg (pyloric caeca)[4]	Growth, NOED				[37]	L; no effect on growth
			0.000009 mg/kg (skeletal muscle)[4]	Growth, NOED				[37]	L; no effect on growth
			0.000041 mg/kg (skin)[4]	Growth, NOED				[37]	L; no effect on growth
			0.000166 mg/kg (spleen)[4]	Growth, NOED				[37]	L; no effect on growth
			0.00277 mg/kg (visceral fat)[4]	Growth, NOED				[37]	L; no effect on growth

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDD

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]**
			0.000143 mg/kg (whole body)[4]	Growth, NOED				[37]	L; no effect on growth
			0.000129 mg/kg (carcass)[4]	Morphology, NOED				[37]	L; no effect on fin necrosis or hemorrhage
			0.000148 mg/kg (gastrointestinal tract)[4]	Morphology, NOED				[37]	L; no effect on fin necrosis or hemorrhage
			0.000155 mg/kg (gill)[4]	Morphology, NOED				[37]	L; no effect on fin necrosis or hemorrhage
			0.000077 mg/kg (heart)[4]	Morphology, NOED				[37]	L; no effect on fin necrosis or hemorrhage
			0.000119 mg/kg (kidney)[4]	Morphology, NOED				[37]	L; no effect on fin necrosis or hemorrhage
			0.000466 mg/kg (liver)[4]	Morphology, NOED				[37]	L; no effect on fin necrosis or hemorrhage
			0.000143 mg/kg (pyloric caeca)[4]	Morphology, NOED				[37]	L; no effect on fin necrosis or hemorrhage
			0.000009 mg/kg (skeletal muscle)[4]	Morphology, NOED				[37]	L; no effect on fin necrosis or hemorrhage

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDD

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]**
			0.000041 mg/kg (skin)[4]	Morphology, NOED				[37]	L; no effect on fin necrosis or hemorrhage
			0.000166 mg/kg (spleen)[4]	Morphology, NOED				[37]	L; no effect on fin necrosis or hemorrhage
			0.00277 mg/kg (visceral fat)[4]	Morphology, NOED				[37]	L; no effect on fin necrosis or hemorrhage
			0.000143 mg/kg (whole body)[4]	Morphology, NOED				[37]	L; no effect on fin necrosis or hemorrhage
			0.000129 mg/kg (carcass)[4]	Mortality, NOED				[37]	L; no effect on mortality
			0.000148 mg/kg (gastrointestinal tract)[4]	Mortality, NOED				[37]	L; no effect on mortality
			0.000155 mg/kg (gill)[4]	Mortality, NOED				[37]	L; no effect on mortality
			0.000077 mg/kg (heart)[4]	Mortality, NOED				[37]	L; no effect on mortality
			0.000119 mg/kg (kidney)[4]	Mortality, NOED				[37]	L; no effect on mortality
			0.000466 mg/kg (liver)[4]	Mortality, NOED				[37]	L; no effect on mortality

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDD

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]**
			0.000143 mg/kg (pyoric ceca)[4]	Mortality, NOED				[37]	L; no effect on mortality
			0.000009 mg/kg (skeletal muscle)[4]	Mortality, NOED				[37]	L; no effect on mortality
			0.000041 mg/kg (skin)[4]	Mortality, NOED				[37]	L; no effect on mortality
			0.000166 mg/kg (spleen)[4]	Mortality, NOED				[37]	L; no effect on mortality
			0.00277 mg/kg (visceral fat)[4]	Mortality, NOED				[37]	L; no effect on mortality
			0.000143 mg/kg (whole body)[4]	Mortality, NOED				[37]	L., no effect on mortality
Salmonids							0.059	[46]	F

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDD

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]**
Wildlife									
Aix sponsa, Wood duck			pg/g (eggs):	% eggs hatched:				[29]	F; central Arkansas; egg TEFs and hatching success and duckling production were negatively correlated; clutch size was similar among wetland Sites 1-3 which were 9, 17, and 58 km downstream from point source of contamination. respectively, and Site 4 which was 111 km away on a separate drainage; duckling abnor-malities were also noted
			Site 1 geometric mean: 36 (1.6 to 482)	47% (9.7 SE)					
			Site 2 geometric mean: 14 (0.8-74)	62% (10.1 SE)					
			Site 3 geometric mean: 4.2 (<1 to 19)	79% (3.8 SE)					
			Site 4 geometric mean: 0.01 (<1 to 0.5)	93% (3.4 SE)					
Aix sponsa, Wood duck									Threshold range of reduced productivity was > 20-50 ppt TEF.

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDD

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]**
Falco peregrinus, Peregrine falcon			11 ng/g (eggs) (n= 6)	11.4% eggshell thinning				[26]	F; Kola Peninsula, Russia
Haliaeetus leucocephalus, Bald eagle chicks			Powell River site: 2,200 ng/kg lipid weight basis (yolk sac) Reference site: 300 ng/kg lipid weight basis (yolk sac)	A nearly 6-fold greater incidence of an hepatic cytochrome P4501A cross-reactive protein was induced in chicks from Powell River site as compared to the reference ($p < 0.05$). No significant concentration-related effects were found for morphological, physiological, or histological parameters.				[25]	F; southern coast of British Columbia; eggs were collected from nests and hatched in the lab; ~ indicates value was taken from a figure.

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDD

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]**
Sterna forsteri, Forster's tern			Lake Poygan site: 8.0 pg/g; (egg) (n = 6) Green Bay site: 37.3 pg/g; (egg) (n = 6)	Birds from Green Bay had increased incubation period, reduced hatchability, lower body weight, increased liver to body weight ratio, and occurrence of edema when compared to birds from Lake Poygan. There was a significantly higher incidence of congenital abnormalities in dead embryos and chicks from Green Bay.				[27]	F; Green Bay, Lake Michigan, and Lake Poygan, Wisconsin

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDD

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]**
Ardea herodias, Great blue heron chicks			Nicomekl site: 10±0.9 ng/kg; (egg) (n = 11)					[28]	L; eggs were collected from three British Columbia colonies with different levels of contamination and incubated in the laboratory
			Vancouver site: 135±49.6 ng/kg (egg) (n = 12)	Depression of growth compared to Nicomekl site. Presence of edema.					
			Crofton site: 2 11±33.7 ng/kg (egg) (n = 6)	Depression of growth compared to Nicomekl site. Presence of edema.					

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDD

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]**
Mustela vison, Mink	Diet: 2 pg/g[5]		21 pg/g[5] (liver)	LOAEL; reduced kit body weights followed by reduced survival		log BMF= 1.05		[30]	L; BMF = biomagnification factor = v_l/v_d, v_l = lipid-normalized concentration in tissue; v_d = lipid-normalized dietary concentration
	Diet: 3 pg/g[5]		34 pg/g[5] (liver)	Reduced kit body weights followed by reduced survival		log BMF = 1.06			
	Diet: 7 pg/g[5]		50 pg/g[5] (liver)	Significant decrease in number of live kits whelped per female		log BMF = 1.04			

[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.
[5] Not clear from reference if concentration is based on wet or dry weight.

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Chemical Category: PESTICIDE (ORGANOCHLORINE)

Chemical Name (Common Synonyms): **CASRN:** 72-54-8
1,1′-(2,2-DICHLOROETHYLIDENE)BIS(4-CHLOROBENZENE),
p,p′-DICHLORODIPHENYLDICHLOROETHANE
4,4′-DICHLORODIPHENYLDICHLOROETHANE

Chemical Characteristics

Solubility in Water: 0.16 mg/L at 24°C [1]

Half-Life: 2.0-15.6 years based on biodegradation of DDD in aerobic soils under field conditions [2]

Log K_{ow}: 6.10 [3]

Log K_{oc}: 6.0 L/kg organic carbon

Human Health

Oral RfD: Not available [4]

Confidence: —

Critical Effect: Lung tumors in male and female mice, liver tumors in male mice, thyroid tumors in male rats

Oral Slope Factor: 2.4 x 10^{-1} per (mg/kg)/day [4]

Carcinogenic Classification: B2 [4]

Wildlife

Partitioning Factors: Partitioning factors for DDD in wildlife were not calculated in the studies reviewed. However, based on the data presented in one study reviewed, log BCFs for birds from the lower Detroit River ranged from 4.97 to 5.22. Concentrations of DDD in birds were 3.5 to 6.1 times higher than those in sediment.

Food Chain Multipliers: Biomagnification factors of 3.2 and 85 were determined for DDT and DDE, respectively, from alewife to herring gulls in Lake Ontario [5]. A study of arctic marine food chains measured biomagnification factors for DDE that ranged from 17.6 to 62.2 for fish to seal, 0.3 to 0.7 for seal to bear, and 10.7 for fish to bear [6].

Aquatic Organisms

Partitioning Factors: Partitioning factors for DDD in aquatic organisms were not calculated in the studies reviewed. However, the data from one study reviewed showed BCFs of 17,600 for oligochaetes and 565,000 for carp. Ratios of DDD in tissue to sediment were 0.65 for oligochaetes and 21 for carp. BSAFs for clams ranged from 0.120 to 2.745 [22,25]. BSAFs for fish ranged from 0.079 to 2.379 [21,23,24,25].

Food Chain Multipliers: 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) [28].

Toxicity/Bioaccumulation Assessment Profile

DDT is very persistent in the environment due to its low vapor pressure, high fat solubility, and resistance to degradation and photooxidation. DDT is degraded to DDE under aerobic conditions and to DDD in anoxic systems [7]. These metabolites, DDD and DDE, are similar to DDT in both their stability and toxicity. Chronic effects of DDT and its metabolites on ecological receptors include changes in enzyme production, hormonal balance, and calcium metabolism, which may cause changes in behavior and reproduction. The high octanol-water partition coefficient of DDT indicates that it is easily accumulated in tissues of aquatic organisms. Laboratory studies have shown that these compounds are readily bioconcentrated in aquatic organisms, with reported log BCFs for DDT ranging from 3.08 to 7.65 and for DDE ranging from 4.80 to 5.26 [8].

Invertebrate species are generally more susceptible than fish species to effects associated with exposure to DDT in the water column [8]. In general, the low solubility of DDT and its metabolites in water suggests that water column exposures are likely to be lower than exposures from ingestion of food or sediment. Sediments contaminated with pesticides, including DDT, have been shown to affect benthic communities at low concentrations. Results of laboratory and field investigations suggest that chronic effects generally occur at total DDT concentrations in sediment exceeding 2 µg/kg [9]. Equilibrium partitioning methods predict that chronic effects occur at DDT concentrations in sediment of 0.6 to 1.7 µg/kg [10].

For fish, the primary route of uptake is via prey items, but both DDT and its metabolites can be accumulated through the skin or gills upon exposure to water. Short-term exposure to DDT concentrations of less than 1 µg/L have been reported to elicit toxic responses in both freshwater and marine fish [8]. DDT may also be transfered to embryos from contaminated adults. DDT concentrations of 1.1 to 2.4 mg/kg in fish embryos have been associated with fry mortality [11,12].

Eggshell thinning, embryo mortality, and decreased hatchling survival have been linked to chronic exposure to DDT and its metabolites in the diet of birds. Of the three compounds, evidence strongly indicates that DDE is responsible for most reproductive toxicity in avian species [13]. Measurements of residues in eggs of birds are a reliable indicator of adverse effects. There is a large amount of variability in sensitivity to DDT and its metabolites among bird species, with waterfowl and raptor species showing the greatest sensitivities. Studies have shown the brown pelican to be most susceptible to adverse effects, with eggshell thinning and depressed productivity occurring at 3.0 µg/g of DDE in the egg and total reproductive failure when residues exceed 3.7 µg/g [13].

Summary of Biological Effects Tissue Concentrations for *p,p′*-DDD

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									
Tubifex sp., Oligochaetes	0.023 mg/kg n = 1	water = 0.85 ng/L n = 1	0.015 mg/kg n = 1					[14]	F; lower Detroit River
Macomona liliana, Mollusk	66.7 µg/kg OC		76.3 µg/kg lipid				1.144	[22]	F; %lipid = 2.95; %sed OC = 0.30
	1,096.0 µg/kg OC		765.2 µg/kg lipid				0.698	[22]	F; %lipid = 2.33; %sed OC = 0.73
	286.4 µg/kg OC		75.1 µg/kg lipid				0.262	[22]	F; %lipid = 2.57; %sed OC = 0.22
	20.0 µg/kg OC		54.9 µg/kg lipid				2.745	[22]	F; %lipid = 2.04; %sed OC = 0.25
	25.0 µg/kg OC		22.4 µg/kg lipid				0.894	[22]	F; %lipid = 3.13; %sed OC = 0.48
Austrovnus stutchburyi, Mollusk	66.7 µg/kg OC		42.4 µg/kg lipid				0.635	[22]	F; %lipid = 5.62; %sed OC = 0.30
	286.4 µg/kg OC		34.4 µg/kg lipid				0.120	[22]	F; %lipid = 4.85; %sed OC = 0.22
	20 µg/kg OC		27.7 µg/kg lipid				1.383	[22]	F; %lipid = 3.87; %sed OC = 0.25
	25 µg/kg OC		25.1 µg/kg lipid				1.002	[22]	F; %lipid = 4.27; %sed OC = 0.48

Summary of Biological Effects Tissue Concentrations for *p,p′*-DDD

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	58.8 µg/kg OC		82 µg/kg lipid				1.394	[25]	F; %lipid = 0.61; %sed OC = 1.19
	159.7 µg/kg OC		82 µg/kg lipid				0.513	[25]	F; %lipid = 0.61; %sed OC = 1.19
Fish									
Anguilla anguilla, Eel	126 µg/kg OC		10 µg/kg lipid				0.079	[26]	F; %lipid = 13; %sed OC = 32
Corogonus autumnalis, Omul (endemic whitefish)		particulate: 1.0 pg/L ± 1.0 n = 7 dissolved: 17 pg/L ±7.3 n = 7	0.0086-0.15 mg/kg lipid (whole body) n = 1						
Oncorhynchus, Salmo, Salvelinus sp., Salmonids	2,667 µg/kg OC		754.5 µg/kg lipid				0.283	[24]	F; %lipid = 11; %sed OC = 2.7
		0.000093 µg/L	83 µg/kg			5.93		[24]	F; %lipid = 11
Salvelinus fontinalis, Brook trout			4.79 mg/kg (whole body)[4]	Behavior, NOED				[18]	L; temperature selection after 24 h exposure to chemical

Summary of Biological Effects Tissue Concentrations for *p,p′*-DDD

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]**
Salvelinus namaycush, Lake trout			0.9 mg/kg (whole body)[4]	Mortality, LOED				[20]	L; survival of fry reduced
Leuciscus cephalus cabeda, Chub	478 µg/kg OC		378 µg/kg lipid				0.790	[21,27]	F; %lipid = 1.27; %sed OC = 2.76
Alburnus alburnus alborella, Bleak	478 µg/kg OC		769 µg/kg lipid				1.608	[21,27]	F; %lipid = 1.95; %sed OC = 2.76
Cyprinus carpio, Carp	0.023 mg/kg n = 1	water = 0.85 ng/L n = 1	0.48 ± 0.26 mg/kg n = 9					[14]	F; lower Detroit River; value is mean ± SD
Pimephales promelas, Fathead minnow			0.6 mg/kg (whole body)[4]	Reproduction, LOED				[17]	L; significantly different from control (p = 0.05)
Gambusia affinis, Mosquito fish			5.3 mg/kg (whole body)[4]	Mortality, NOED				[19]	L; no effect on survivorship after 3 days
Catastoma macrocheilus, Largescale sucker	530 µg/kg OC		1,261 µg/kg lipid				2.379	[23]	F; %lipid = 11.1; %sed OC = 1.0

Summary of Biological Effects Tissue Concentrations for *p,p′*-DDD

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]**
Cottus cognatus, Slimy sculpin	2667 µg/kg OC		587.5 µg/kg lipid				0.220	[24]	F; %lipid = 8; %sed OC = 2.7
		0.000093 µg/L	47 µg g/kg			5.70		[24]	F; %lipid = 8
Comephorus dybowskii, Pelagic sculpin		particulate: 1.0 pg/L ±1.0 n = 7 dissolved: 17 pg/L ±7.3 n = 7	0.12-0.16 mg/kg lipid (whole body) n = 1					[15]	F; Lake Baikal, Siberia
Wildlife									
Bucephala clangula, Goldeneye	0.023 mg/kg n = 1	water = 0.85 ng/L n = 1	0.080 ± 0.024 mg/kg n = 3					[14]	F; lower Detroit River; value is mean ± SD
Aythya affinis, Lesser scaup	0.023 mg/kg n = 1	water = 0.85 ng/L n = 1	0.093 ± 0.027 mg/kg n = 7					[14]	F; lower Detroit River; value is mean ± SD
Aythya marila, Greater scaup	0.023 mg/kg n = 1	water = 0.85 ng/L n = 1	0.14±0.045 mg/kg n = 3					[14]	F; lower Detroit River; value is mean ± SD

Summary of Biological Effects Tissue Concentrations for *p,p′*-DDD

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]**
Falco peregrinus, Peregrine falcon (eggs)			9 ng/g (eggs) n = 6	11.4% eggshell thinning				[16]	F; Kola Penninsula, Russia; n = number of clutches sampled
Phoca siberica, Baikal seal		particulate: 1.0 pg/L ± 1.0 n = 7 dissolved: 17 pg/L ± 7.3 n = 7	2.0-2.2 mg/kg[5] lipid (blubber) n = 1					[15]	F; Lake Baikal, Siberia

[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.
[5] Not clear from reference if concentration is based on wet or dry weight.

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Chemical Category: PESTICIDE (ORGANOCHLORINE)

Chemical Name (Common Synonyms): **CASRN:** 72-55-9
1,1′-(DICHLOROETHYLIDENE)BIS(4-CHLOROBENZENE),
p,p′-DICHLORODIPHENYLDICHLOROETHYLENE
4,4′-DICHLORODIPHENYLDICHLOROETHYLENE

Chemical Characteristics

Solubility in Water: 0.065 mg/L at 24°C [1]

Half-Life: 2.0 - 15.6 years based on biodegradation of DDD in aerobic soils under field conditions [2]

Log K_{ow}: 6.76 [3]

Log K_{oc}: 6.65 L/kg organic carbon

Human Health

Oral RfD: No data [4]

Confidence: —

Critical Effect: Liver tumors in mice and hamsters, thyroid tumors in female rats

Oral Slope Factor: 3.4 x 10^{-1} per (mg/kg)/day [4]

Carcinogenic Classification: B2 [4]

Wildlife

Partitioning Factors: Based on the data presented in one study, log BCFs for birds collected from the lower Detroit River ranged from 5.92 to 6.36. Concentrations of DDE in birds were 40 to 108 times higher than in sediment. BSAFs were calculated for red-winged blackbird eggs and tree swallow eggs during a study in the Great Lakes area, with values ranging from 13 to 870 as reported in the attached summary table. BSAFs for tree swallow nestlings were 5 and 49.

Food Chain Multipliers: Biomagnification factors of 3.2 and 85 were determined for DDT and DDE, respectively, from alewife to herring gulls in Lake Ontario [5]. A study of arctic marine food chains measured biomagnification factors for DDE that ranged from 17.6 to 62.2 for fish to seal, 0.3 to 0.7 for seal to bear, and 10.7 for fish to bear [6].

Aquatic Organisms

Partitioning Factors: Partitioning factors for DDE in aquatic organisms were not calculated in the studies reviewed. However, the data showed ratios of DDT in tissue to sediment of 0.49 for oligochaetes and 32 for fish from the lower Detroit River. Ratios of DDT in lipid to sediment for three fish species from Rio de la Plata, Argentina ranged from 87 to 26,000. BSAFs for clams ranged from 1.2313 to

107.7 [15,41,36]. BSAFs for dover sole collected in southern California ranged from 1.7 to 3.4. BSAFs for other species ranged from 1.274 to 140.

Food Chain Multipliers: Food chain multipliers (FCMs) for trophic level 3 aquatic organisms were 23.7 (all benthic food web), 1.7 (all pelagic food web), and 14.4 (benthic and pelagic food web). FCMs for trophic level 4 aquatic organisms were 57.5 (all benthic food web), 3.7 (all pelagic food web), and 26.7 (benthic and pelagic food web) [46].

Toxicity/Bioaccumulation Assessment Profile

DDT is very persistent in the environment due to its low vapor pressure, high fat solubility, and resistance to degradation and photooxidation. DDT is degraded to DDE under aerobic conditions and to DDD in anoxic systems [7]. These metabolites, DDD and DDE, are similar to DDT in both their stability and toxicity. Chronic effects of DDT and its metabolites on ecological receptors include changes in enzyme production, hormonal balance, and calcium metabolism, which may cause changes in behavior and reproduction. The high octanol-water partition coefficient of DDT indicates that it is easily accumulated in tissues of aquatic organisms. Laboratory studies have shown that these compounds are readily bioconcentrated in aquatic organisms, with reported log BCFs for DDT ranging from 3.08 to 6.65 and for DDE ranging from 4.80 to 5.26 [8].

Invertebrate species are generally more susceptible than fish species to effects associated with exposure to DDT in the water column [8]. In general, the low solubility of DDT and its metabolites in water suggests that water column exposures are likely to be lower than exposures from ingestion of food or sediment. Sediments contaminated with pesticides, including DDT, have been shown to impact benthic communities at low concentrations. Results of laboratory and field investigations suggest that chronic effects generally occur at total DDT concentrations in sediment exceeding 2 µg/kg [9]. Equilibrium partitioning methods predict that chronic effects occur at DDT concentrations in sediment of 0.6 to 1.7 µg/kg [10].

For fish, the primary route of uptake is via prey items, but both DDT and its metabolites can be accumulated through the skin or gills upon exposure to water. Short-term exposure to DDT concentrations of less than 1 µg/L have been reported to elicit toxic responses in both freshwater and marine fish [8]. DDT may also be transferred to embryos from contaminated adults. DDT concentrations of 1.1 to 2.4 mg/kg in fish embryos have been associated with fry mortality [11,12].

Eggshell thinning, embryo mortality, and decreased hatchling survival have been linked to chronic exposure to DDT and its metabolites in the diet of birds. Of the three compounds, evidence strongly indicates that DDE is responsible for most reproductive toxicity in avian species [13]. Measurements of residues in eggs of birds are a reliable indicator of adverse effects. There is a large amount of variability in sensitivity to DDT and its metabolites among bird species, with waterfowl and raptor species showing the greatest sensitivities. Studies have shown the brown pelican to be most susceptible to adverse effects, with eggshell thinning and depressed productivity occurring at 3.0 µg/g of DDE in the egg and total reproductive failure when residues exceed 3.7 µg/g [13].

Summary of Biological Effects Tissue Concentrations for *p,p′*-DDE

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									
Tubifex sp., Oligochaetes	0.012 mg/kg (n = 1)	Surface water 0.57 ng/L (n = 1)	0.0059 mg/kg (n = 1)					[14]	F; lower Detroit River; value is mean ± SD
Viviparus conectus, Gastropod mollusk	294 µg/kg OC		368 µg/kg lipid				1.2517	[36]	F; %lipid = 7.06; %sed OC = 1.02
Unio elongatulus, Bivalve mollusk	294 µg/kg OC		362 µg/kg lipid				1.2313	[36]	F; %lipid = 10.49; %sed OC = 1.02
Mollusks (unspecified)	99.67 µg/kg OC		229 µg/kg lipid				2.298	[37]	F; %lipid = 1.1; %sed OC = 2.8
Macomona liliana, Mollusk	36.67 µg/kg OC		522.20 µg/kg lipid				14.241	[38]	F; %lipid = 2.95; %sed OC = 0.30
	35.62 µg/kg OC		573.39 µg/kg lipid				16.097	[38]	F; %lipid = 2.33; %sed OC = 0.73
	36.36 µg/kg OC		278.21 µg/kg lipid				7.652	[38]	F; %lipid = 2.57; %sed OC = 0.22
	20 µg/kg OC		328.92 µg/kg lipid				16.446	[38]	F; %lipid = 2.04; %sed OC = 0.25

Summary of Biological Effects Tissue Concentrations for *p,p′*-DDE

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]**
	6.25 µg/kg OC		61.34 µg/kg lipid				9.814	[38]	F; %lipid = 3.13; %sed OC = 0.48
Austrovenus stutchburyi, Mollusk	36.67 µg/kg OC		141.64 µg/kg lipid				3.863	[38]	F; %lipid = 5.62; %sed OC = 0.30
	35.62 µg/kg OC		148.75 µg/kg lipid				4.176	[38]	F; %lipid = 5.21; %sed OC = 0.73
	36.36 µg/kg OC		57.94 µg/kg lipid				1.594	[38]	F; %lipid = 4.85; %sed OC = 0.22
	20 µg/kg OC		59.95 µg/kg lipid				2.998	[38]	F; %lipid = 3.87; %sed OC = 0.25
	6.25 µg/kg OC		10.54 µg/kg lipid				1.686	[38]	F; %lipid = 4.27; %sed OC = 0.48
Corbicula fluminea, Asian clam	13 µg/kg OC		1,400 µg/kg lipid				107.7	[15]	F; %lipid not reported; %sed OC = 2.3
Corbicula fluminea, Asian clam	(0-5 cm) 0.3 ng/g dw	Surface water 1.8 ng/L	1.4 µg/g lipid (whole tissue)					[15]	F; Rio de La Plata, Argentina; lipid content 2.4-3.8%
	0.6 ng/g dw		1.4 µg/g lipid (whole tissue)						
Corbicula fluminea, Asian clam	9,664 µg/kg OC		540,984 µg/kg lipid				55.979	[41]	F; %lipid = 0.61; %sed OC = 0.19
	168 µg/kg OC		4,098 µg/kg lipid				24.393	[41]	F; %lipid = 0.61; %sed OC = 0.19

Summary of Biological Effects Tissue Concentrations for *p,p′*-DDE

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]**
	210 µg/kg OC		2,131 µg/kg lipid				10.148	[41]	F; %lipid = 0.61; %sed OC = 0.19
Astacidae, Crayfish	99.67 µg/kg OC		177 µg/kg lipid				1.776	[37]	F; %lipid = 1.3; %sed OC = 2.8
Chironomus riparius, Midge			1.6 mg/kg (whole body)[4]	Behavior, NOED				[31]	L; no effect on swimming behavior
			0.27 mg/kg (whole body)[4]	Behavior, NOED				[31]	L; no effect on swimming behavior
			0.1 mg/kg (whole body)[4]	Behavior, NOED				[31]	L; no effect on swimming behavior
Chironomus riparius, Midge			7.35 mg/kg (whole body)[4]	Development, LOED				[34]	L; development time from egg to 4th instar decreased from 22-25 days to 19-21 days
			3.75 mg/kg (whole body)[4]	Development, NOED				[34]	L; no effect on developmental period of larvae
Fishes									
Anguilla anguilla, Eel	5 µg/kg OC		156 µg/kg lipid				31.200	[43]	F; %lipid = 7; %sed OC = 7

Summary of Biological Effects Tissue Concentrations for *p,p′*-DDE

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 µg/kg OC		213 µg/kg lipid				42.600	[43]	F; %lipid = 7; %sed OC = 14
	32 µg/kg OC		2117 µg/kg lipid				66.156	[43]	F; %lipid = 6; %sed OC = 18
	76 µg/kg OC		849 µg/kg lipid				11.171	[43]	F; %lipid = 10; %sed OC = 12
	23 µg/kg OC		658 µg/kg lipid				28.609	[43]	F; %lipid = 10; %sed OC = 12
	72 µg/kg OC		2,176 µg/kg lipid				30.222	[43]	F; %lipid = 13; %sed OC = 32
Oncorhynchus mykiss, Rainbow trout			0.15 mg/kg (fat)[4]	Growth, ED40				[29]	L; 40% decrease in growth relative to control
			0.15 mg/kg (fat)[4]	Physiological, ED30				[29]	L; 30% decrease in hemoglobin content relative to control
			0.15 mg/kg (fat)[4]	Physiological, ED30				[29]	L; 30% increase in liver size relative to control
			0.08 mg/kg (fat)[4]	Physiological, ED35				[29]	L; 35% increase in kidney size relative to control

Summary of Biological Effects Tissue Concentrations for *p,p′*-DDE

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, Salmo, *Salvelinus* spp., Salmonids	1,889 µg/kg OC		7,817 µg/kg lipid				4.139	[40]	F; %lipid = 11; %sed OC =2.7
		0.000076 µg/L	860 µg/kg			7.05		[40]	F; %lipid = 11
Oncorhynchus sp., Salmon	99.67 µg/kg OC		925 µg/kg lipid				9.281	[37]	F; %lipid = 13.1; %sed OC = 2.8
Prosopium williamsoni, Mountain whitefish	544.4 µg/kg OC		2,333 µg/kg lipid				4.285	[39]	F; %lipid = 12.0, %sed OC = 0.9
	3,500 µg/kg OC		4,460 µg/kg lipid (arithmetic mean of two samples)				1.274	[39]	F; %lipid = 12.25 %sed OC = 0.3
Coregonus autumnalis, Omul (endemic whitefish)		particulate: <14 pg/L n = 7 dissolved: 17±7.1 pg/L n = 7	0.31-0.50 mg/kg lipid n = 2						

Summary of Biological Effects Tissue Concentrations for *p,p′*-DDE

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]**
Salvelinus fontinalis, Brook trout			44.9 mg/kg (whole body)[4]	Behavior, LOED				[30]	L; temperature selection after 24 h exposure to chemical
Salvelinus namaycush, Lake trout			1.09 mg/kg (whole body)[4]	Mortality, LOED				[33]	L; survival of fry reduced
Alburnus alburnus alborella, Bleak fish	294 µg/kg OC		1,092 µg/kg lipid				3.7143	[36]	F; %lipid = 21.43; %sed OC = 1.02
Alburnus alburnus alborella, Bleak fish	358 µg/kg OC		2,113 µg/kg lipid				5.9022	[35, 45]	F; %lipid = 1.95; %sed OC = 2.76
Chondrostoma soetta	294 µg/kg OC		1,179 µg/kg lipid				4.0102	[36]	F; %lipid = 9.75; %sed OC = 1.02
Cyprinus carpio, Common carp	99.67 µg/kg OC		4,209 µg/kg lipid				42.229	[37]	F, %lipid = 13.9; %sed OC = 2.8
Cyprinus carpio, Common carp	174 µg/kg OC		1,905 µg/kg lipid				10.948	[42]	F, %lipid = 8.4; %sed OC = 2.13

Summary of Biological Effects Tissue Concentrations for *p,p′*-DDE

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, Carp	0.012 mg/kg (n = 1)	Surface water 0.57 ng/L (n = 1)	0.38 ± 0.15 mg/kg (n = 9)					[14]	F; lower Detroit River; value is mean ± SD
Scardinius erythrophalmus, Rudd	294 µg/kg OC		1,473 µg/kg lipid				6.546	[36]	F; %lipid = 11.66; %sed OC = 1.02
Leuciscus cephalus, Chub	294 µg/kg OC		1,473 µg/kg lipid				5.0102	[36]	F; %lipid = 9.98; %sed OC = 1.02
Leuciscus cephalus cabeda, Chub	358 µg/kg OC		1,953 µg/kg lipid				5.4553	[35, 45]	F; %lipid = 1.27; %sed OC = 2.76
Rutilus pigus	294 µg/kg OC		728 µg/kg lipid				2.4762	[36]	F; %lipid = 12.63; %sed OC = 1.02
Rutilus rubilio	294 µg/kg OC		1,167 µg/kg lipid				3.9694	[36]	F; %lipid = 11.05; %sed OC = 1.02
Catostomus commersoni, White sucker	208 µg/kg OC		1,519 µg/kg lipid				7.303	[42]	F; %lipid = 7.9; %sed OC = 1.44

Summary of Biological Effects Tissue Concentrations for *p,p′*-DDE

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]**
mixed *Catastoma* sp., Suckers	3,500 µg/kg OC		3,253 µg/kg lipid (arithmetic mean of two samples)				0.929	[39]	F; %lipid = 9.2; %sed OC = 0.3
Catastoma macrocheilus, Largescale sucker	3,010 µg/kg OC		7,477 µg/kg lipid				2.484	[39]	F; %lipid = 11.1; %sed OC = 1.0
Barbus barbus, Barbel	294 µg/kg OC		1,333 µg/kg lipid				4.5340	[36]	F; %lipid = 16.43; %sed OC = 1.02
Siluris glanis, Wels fish, juveniles	294 µg/kg OC		731 µg/kg lipid				2.4864	[36]	F; %lipid = 3.83; %sed OC = 1.02
Siluris glanis, Wels fish, adults	294 µg/kg OC		1,613 µg/kg lipid				5.4864	[36]	F; %lipid = 5.38; %sed OC = 1.02
Pimelodus albicans, Mandi	0.2 ng/g dw		0.6 µg/g lipid (n = 2) (muscle)					[15]	F; Rio de La Plata, Argentina; lipid content 4%
Pimelodus albicans, Mandi	20 µg/kg OC		600 µg/kg lipid				30.0	[15]	F; %lipid not reported; %sed OC = 1.0
Gambusia affinis, Mosquito fish			29.2 mg/kg (whole body)[4]	Mortality, NOED				[32]	L; no effect on survivorship after 3 days

Summary of Biological Effects Tissue Concentrations for *p,p′*-DDE

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]**
Ambloplites rupestris, Rock bass	99.67 µg/kg OC		365 µg/kg lipid				3.662	[37]	F; %lipid = 0.7; %sed OC = 2.8
Sunfish (unspecified)	99.67 µg/kg OC		254 µg/kg lipid				2.548	[37]	F; %lipid = 3.7; %sed OC = 2.8
Roccus chrysops, White bass	99.67 µg/kg OC		1,586 µg/kg lipid				15.913	[37]	F; %lipid = 1.8; %sed OC = 2.8
Micropterus salmoides, Smallmouth bass	99.67 µg/kg OC		1,352 µg/kg lipid				13.565	[37]	F; %lipid = 0.6; %sed OC = 2.8
Dorosoma cepedianum, Gizzard shad	99.67 µg/kg OC		382 µg/kg lipid				3.833	[37]	F; %lipid = 6.8; %sed OC = 2.8
Perca fluviatilis, Perch	294 µg/kg OC		3,390 µg/kg lipid				11.5306	[36]	F; %lipid = 5.84; %sed OC = 1.02
Stizostedion vitreum, Walleye	99.67 µg/kg OC		2,593 µg/kg lipid				26.016	[37]	F; %lipid = 1.2; %sed OC = 2.8

Summary of Biological Effects Tissue Concentrations for *p,p′*-DDE

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	27 µg/g dw (n = 5)		16.0 µg/g (n = 5) (muscle) 210 µg/g (n = 3) (liver)			log MBAF -0.26 0.79	 1.7 2.0	[16]	F; Southern California Bight; modified bioaccumulation factor (MBAF) = C_{org} ww/ C_{sed} dw; water content of tissue was not measured
	0.09 µg/g dw (n = 10)		0.24 µg/g (n = 10) (muscle) 0.80 µg/g (n = 6) (liver)			log MBAF 0.43 1.79	 1.8 3.4		
Oligosarcus jenynsi, Common name not available	5.7 ng/g dw		0.5 µg/g lipid (n = 7) (muscle)					[15]	F; Rio de La Plata, Argentina; lipid content 0.32%
Prochilodus platensis, Curimata	20 µg/kg OC		2,800 µg/kg lipid				140	[15]	F, %lipid not reported; %sed OC = 1.0
Prochilodus platensis, Curimata	0.2 ng/g dw		Three composite samples: 1.2 (n = 4), 5.2 (n = 4) and 2 (n = 5) µg/g lipid (muscle)					[15]	F; Rio de La Plata, Argentina; lipid content 1-12.7%

Summary of Biological Effects Tissue Concentrations for *p,p′*-DDE

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]**
Gar pike (unspecified)	99.67 µg/kg OC		11,986 µg/kg lipid				120.257	[37]	F; %lipid = 0.8; %sed OC = 2.8
Comephorus bybowskii, Pelagic sculpin,		particulate: <14 pg/L n = 7 dissolved: 17 pg/L ± 7.1 n = 7	0.74-0.76 mg/kg lipid n = 1					[17]	F; Lake Baikal, Siberia
Cottus cognatus, Slimy sculpin	1,889 µg/kg OC		2,375 µg/kg lipid				1.257	[40]	F; %lipid = 8; %sed OC = 2.7
		0.000076 µg/L	190 µg/kg			6.40		[40]	F; %lipid =8; %sed OC = 2.7
Wildlife									
Bucephala clangula, Goldeneye	0.012 mg/kg (n = 1) seston = 0.10 mg/kg	Surface water 0.57 ng/L (n = 1)	0.48 ± 0.18 mg/kg (whole body) (n = 3)					[14]	F; lower Detroit River; value is mean ± SD

Summary of Biological Effects Tissue Concentrations for *p,p′*-DDE

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]**
Aythya affinis, Lesser scaup	0.012 mg/kg (n = 1)	Surface water 0.57 ng/L (n = 1)	0.80 ± 0.33 mg/kg (whole body) (n = 7)					[14]	F; lower Detroit River; value is mean ± SD
Aythya marila, Greater scaup	0.012 mg/kg (n = 1)	Surface water 0.57 ng/L (n = 1)	1.3 ± 0.25 mg/kg (whole body) (n = 3)					[14]	F; lower Detroit River; value is mean ± SD
Falco peregrinus, Peregrine falcon			µg/g (egg): ≤15 15-30 >30	Young produced per active nest: 1.8 2.0 1.0				[26]	F; Alaska; young produced not adjusted for sample egg collected

Summary of Biological Effects Tissue Concentrations for *p,p′*-DDE

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]**
Aquila chrysaetos, Golden eagle			µg/g (egg): 0.1 0.1 0.2 0.3 0.3	Mean % eggshell thinning = 7% 1% 3% 4% 5%				[24]	F; Great Britain; percentage of thinning based on thickness index [24]
Haliaeetus leucocephalus, Bald eagle			10 µg/g (egg)	Mean percent eggshell thinning= 10%				[22]	F; Oregon and Washington
			µg/g (egg): <2.2 2.2-3.5 3.6-6.2 6.3-11.9 ≥12	Young produced per active nest: 1.0 1.0 0.5 0.3 0.2				[23]	F
Ardea herodias, Great blue heron			4 µg/g (egg) 5 µg/g (egg)	Mean percent eggshell thinning = 10% 13%				[18]	F; Washington

Summary of Biological Effects Tissue Concentrations for *p,p′*-DDE

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]**
Plegadis chihi, White-faced ibis				Mean percent eggshell thinning=				[20]	F; Nevada
			2 µg/g (egg)	12%					
			1 µg/g (egg)	8%					
				Young produced per active nest:				[21]	F; Nevada; young produced not adjusted for sample egg collected
			µg/g (egg):						
			≤1	1.8					
			1-4	1.8					
			4-8	1.3					
			8-16	0.8					
			>16	0.6					
Egretta thula, Snowy egret				Mean percent eggshell thinning=				[20]	F; Nevada; young produced not adjusted for sample egg collected
			1 µg/g (egg)	3%					
			2 µg/g (egg)	12%					
				Young produced per active nest:					
			µg/g (egg):						
			≤1	2.2					
			1-5	2.4					
			5-10	1.0					
			10-20	1.0					

Summary of Biological Effects Tissue Concentrations for *p,p′*-DDE

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]**
Sula bassanus, Northern gannet			µg/g (egg) 19	Mean % eggshell thinning = 17%				[19]	F; Quebec
Larus californicus, California gull			430 mg/kg (brain)[4]	Mortality, not available (NA)				[28]	L
			175 mg/kg (breast)[4]	Mortality, NA				[28]	L
			3,100 mg/kg (liver)[4]	Mortality, NA				[28]	L
			220 mg/kg (brain)[4]	NA, NA				[28]	L
			490 mg/kg (breast)[4]	NA, NA				[28]	L
			800 mg/kg (liver)[4]	NA				[28]	L
			750 mg/kg (liver)[4]	NA				[28]	L
Pelecanus occidentalis, Brown pelican			4.4 mg/kg (brain)[4]	Mortality, NA				[28]	L
			59.5 mg/kg (breast)[4]	Mortality, NA				[28]	L
			7.15 mg/kg (liver)[4]	Mortality, NA				[28]	L

Summary of Biological Effects Tissue Concentrations for *p,p′*-DDE

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]**
Agelaius phoeniceus, Red-winged blackbird (eggs)	40.5 ng/g TOC = 2.5%		3,088.1 ng/g				41.4	[25]	F; Great Lakes/St. Lawrence River basin; 12 wetlands sites; sediment concentration reported as wet weight concentration which may be a typographical error
	7.9 ng/g TOC = 21.0%		777.7 ng/g				372.7		
	373.1 ng/g TOC = 7.5%		648.7 ng/g				12.9		
	1,160.7 ng/g TOC = 12%		1,299.6 ng/g				13.2		
	10.4 ng/g TOC-18.5%		305.7 ng/g				113.3		
	65.4 ng/g TOC = 11.5%		826.2 ng/g				30.3		
	1.6 ng/g TOC = 10.5%		416.1 ng/g				582.4		
	0.8 ng/g TOC = 13.8%		145.1 ng/g				522		
	1.3 ng/g TOC = 11.1%		183.5 ng/g				326.4		
	3.0 ng/g TOC = 23.9%		117.6 ng/g				203.7		

Summary of Biological Effects Tissue Concentrations for *p,p′*-DDE

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]**
Tachycineta bicolor, Tree swallow Nestlings			(whole body minus feet, beak, wings, and feathers)					[25]	F; Great Lakes/St. Lawrence River basin; 12 wetlands sites; sediment concentration reported as wet weight concentration which may be a typographical error
	65.4 ng/g TOC = 11.5%		288.2 ng/g				548.9		
	0.8 ng/g TOC = 13.8%		22.4 ng/g						
Eggs	65.4 ng/g TOC = 11.5%		794.7 ng/g				16.2		
	0.8 ng/g TOC = 13.8%		458.2 ng/g				868.6		
			3.5 µg/g (egg) (n = 6)	11.4% eggshell thinning				[27]	F; Kola Penninsula, Russia; n = number of clutches sampled

Summary of Biological Effects Tissue Concentrations for *p,p′*-DDE

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]**
Phoca siberica, Baikal seal		particulate: <14 pg/L n = 7 dissolved: 17 pg/L ± 7.1 n = 7	43-44 mg/kg lipid n = 1					[17]	F; Lake Baikal, Siberia

[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: PESTICIDE (ORGANOCHLORINE)

Chemical Name (Common Synonyms): **CASRN:** 50-29-3
1,1′-(2,2,2-TRICHLOROETHYLIDENE)BIS(4-CHLOROBENZENE),
p,p′-DICHLORODIPHENYLTRICHLOROETHANE,
4,4′-DICHLORODIPHENYLTRICHLOROETHANE

Chemical Characteristics

Solubility in Water: 0.0031 - 0.0034 mg/L at 25°C [1]

Half-Life: 2.0 - 15.6 years based on biodegradation of DDD in aerobic soils under field conditions [2]

Log K_{ow}: 6.83 [3]

Log K_{oc}: 6.71 L/kg organic carbon

Human Health

Oral RfD: 5×10^{-4} mg/kg/day [4]

Confidence: Medium, uncertainty factor = 100

Critical Effect: Liver lesions in rats, liver tumors in mice and rats

Oral Slope Factor: 3.4×10^{-1} per (mg/kg)/day [4]

Carcinogenic Classification: B2 [4]

Wildlife

Partitioning Factors: Partitioning factors for DDT in wildlife were not calculated in the studies reviewed. However, based on the data in one study, log BCFs for birds from the lower Detroit River ranged from 4.81 to 5.01. Concentrations of DDT in birds were 2.1 to 3.3 times higher than in sediment.

Food Chain Multipliers: Biomagnification factors of 3.2 and 85 were determined for DDT and DDE, respectively, from alewife to herring gulls in Lake Ontario [5]. A study of arctic marine food chains measured biomagnification factors for DDE that ranged from 17.6 to 62.2 for fish to seal, 0.3 to 0.7 for seal to bear, and 10.7 for fish to bear [6].

Aquatic Organisms

Partitioning Factors: Based on the results from one study reviewed, the log BCF for carp collected from the lower Detroit River was 4.77. Ratios of DDT in lipids to sediment were 450 in clams and 1,250 to 11,000 in fish from the Rio de la Plata, Argentina. BSAFs for clams ranged from 0.060 to 302.326 [14,33,36]. BSAFs for fish ranged from 0.120 to 88.07.

Food Chain Multipliers: Food chain multipliers (FCMs) for trophic level 3 aquatic organisms were 22.5 (all benthic food web), 1.7 (all pelagic food web), and 13.7 (benthic and pelagic food web). FCMs for trophic level 4 aquatic organisms were 52.5 (all benthic food web), 3.6 (all pelagic food web), and 24.6 (benthic and pelagic food web) [39].

Toxicity/Bioaccumulation Assessment Profile

DDT is very persistent in the environment due to its low vapor pressure, high fat solubility, and resistance to degradation and photooxidation. DDT is degraded to DDE under aerobic conditions and to DDD in anoxic systems [7]. These metabolites, DDD and DDE, are similar to DDT in both their stability and toxicity. Chronic effects of DDT and its metabolites on ecological receptors include changes in enzyme production, hormonal balance, and calcium metabolism, which may cause changes in behavior and reproduction. The high octanol-water partition coefficient of DDT indicates that it is easily accumulated in tissues of aquatic organisms. Laboratory studies have shown that these compounds are readily bioconcentrated in aquatic organisms, with reported log BCFs for DDT ranging from 3.08 to 6.65 and for DDE ranging from 4.80to 5.26 [8].

Invertebrate species are generally more susceptible than fish species to effects associated with exposure to DDT in the water column [8]. In general, the low solubility of DDT and its metabolites in water suggests that water column exposures are likely to be lower than exposures from ingestion of food or sediment. Sediments contaminated with pesticides, including DDT, have been shown to affect benthic communities at low concentrations. Results of laboratory and field investigations suggest that chronic effects generally occur at total DDT concentrations in sediment exceeding 2 µg/kg [9]. Equilibrium partitioning methods predict that chronic effects occur at DDT concentrations in sediment of 0.6 to 1.7 µg/kg [10].

For fish, the primary route of uptake is via prey items, but both DDT and its metabolites can be accumulated through the skin or gills upon exposure to water. Short-term exposure to DDT concentrations of less than 1 µg/L have been reported to elicit toxic responses in both freshwater and marine fish [8]. DDT may also be transfered to embryos from contaminated adults. DDT concentrations of 1.1 to 2.4 mg/kg in fish embryos have been associated with fry mortality [11,12].

Eggshell thinning, embryo mortality, and decreased hatchling survival have been linked to chronic exposure to DDT and its metabolites in the diet of birds. Of the three compounds, evidence strongly indicates that DDE is responsible for most reproductive toxicity in avian species [13]. Measurements of residues in eggs of birds are a reliable indicator of adverse effects. There is a large amount of variability in sensitivity to DDT and its metabolites among bird species, with waterfowl and raptor species showing the greatest sensitivities. Studies have shown the brown pelican to be most susceptible to adverse effects, with eggshell thinning and depressed productivity occurring at 3.0 µg/g of DDE in the egg and total reproductive failure when residues exceed 3.7 µg/g [13].

Summary of Biological Effects Tissue Concentrations for p,p′-DDT

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									
Macomona liliana, Mollusk	63.33 µg/kg OC		13.22 µg/kg lipid				0.209	[33]	F; %lipid = 2.95; %sed OC = 0.30
	76.71 µg/kg OC		33.91 µg/kg lipid				0.442	[33]	F; %lipid = 2.33; %sed OC = 0.73
	127.27 µg/kg OC		24.12 µg/kg lipid				0.190	[33]	F; %lipid = 2.57; %sed OC = 0.22
	20.83 µg/kg OC		7.35 µg/kg lipid				0.353	[33]	F; %lipid = 3.13; %sed OC = 0.48
Austrovenus stutchburyi, Mollusk	63.33 µg/kg OC		8.01 µg/kg lipid				0.126	[33]	F; %lipid = 5.62; %sed OC = 0.30
	76.71 µg/kg OC		7.29 µg/kg lipid				0.095	[33]	F; %lipid = 5.21; %sed OC = 0.73
	127.71 µg/kg OC		7.63 µg/kg lipid				0.060	[33]	F; %lipid = 4.85; %sed OC = 0.22
Corbicula fluminea, Asian clam	4.3 µg/kg OC		1,300 µg/kg lipid				302.326	[14]	F; %lipid = not reported; %sed OC = 2.3
Corbicula fluminea, Asian clam	3277 µg/kg OC		108,197 µg/kg lipid				33.017	[36]	F; %lipid = 0.61; %sed OC = 1.19

Summary of Biological Effects Tissue Concentrations for p,p′-DDT

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]**
	67 µg/kg OC		508 µg/kg lipid				7.563	[36]	F; %lipid = 0.61; %sed OC = 1.19
	92 µg/kg OC		164 µg/kg lipid				1.774	[36]	F; %lipid = 0.61; %sed OC = 1.19
Corbicula fluminea, Asian clam	(0-5 cm) 2.9 ng/g dw		1.3 µg/g lipid (whole tissue)					[14]	F; Rio de La Plata, Argentina; lipid content 2.4-3.8%
Mercenaria mercenaria, Quahog clam			0.126 mg/kg (whole body)[4]	Behavior, NOED				[29]	L; No effect on feeding activity
Mya arenaria, Soft shell clam				NOED				[29]	L; no effect on feeding activity
Daphnia magna, Cladoceran			1.83 mg/kg (whole body)[4]	Mortality, NOED				[23]	L; no effect on survivorship in 3 days
Diporeia spp., Amphipod			19.7 mg/kg (whole body)[4]	Mortality, NOED				[22]	L; no increase in mortality in 96 hours

Summary of Biological Effects Tissue Concentrations for p,p′-DDT

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]**
Gammarus fasciatus, Amphipod			0.336 mg/kg (whole body)[4]	Mortality, NOED				[23]	L; no effect on survivorship in 3 days
Palaemonetes kadiakensis, Grass shrimp			0.1 mg/kg (whole body)[4]	Mortality, NOED				[23]	L; no effect on survivorship in 3 days
Orconectes nais, Crayfish			0.0466 mg/kg (whole body)[4]	Mortality, NOED				[23]	L; no effect on survivorship in 3 days
Ephemera danica, Mayfly			6 mg/kg (whole body)[4]	Growth, NOED				[20]	L
			6 mg/kg (whole body)[4]	Mortality, NOED					L
Hexagenia bilineata, Mayfly			0.336 mg/kg (whole body)[4]	Mortality, NOED				[23]	L; no effect on survivorship after 3 days
Siphlonurus sp., Mayfly			0.216 mg/kg (whole body)[4]	Mortality, NOED				[23]	L; no effect on survivorship after 3 days

Summary of Biological Effects Tissue Concentrations for p,p′-DDT

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]**
Libellula sp., Dragonfly			0.0144 mg/kg (whole body)[4]	Mortality, NOED				[23]	L; no effect on survivorship after 2 days
Ischnura verticalis, Damselfly			0.075 mg/kg (whole body)[4]	Mortality, NOED				[23]	L; no effect on survivorship after 2 days
Chironomus sp., Midge			0.44 mg/kg (whole body)[4]	Mortality, NOED				[23]	L; no effect on survivorship in 3 days
Chironomus riparius, Midge			0.83 mg/kg (whole body)[4]	Behavior, LOED				[24]	L; reduced swimming ability
			0.18 mg/kg (whole body)[4]	Behavior, NOED				[24]	L; no effect on swimming behavior
			0.08 mg/kg (whole body)[4]	Behavior, NOED				[24]	L; no effect on swimming behavior
Fishes									
Squalus acanthias, Spiny dogfish			0.1 mg/kg (whole body)[4]	Mortality, NOED				[32]	L; no effect on mortality in 24 hours

Summary of Biological Effects Tissue Concentrations for p,p′-DDT

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]**
Anguilla anguilla, Eel	23 µg/kg OC		158 µg/kg lipid				6.87	[37]	F; %lipid = 7; %sed OC = 7
	8 µg/kg OC		135 µg/kg lipid				16.88	[37]	F; %lipid = 7; %sed OC = 14
	14 µg/kg OC		1233 µg/kg lipid				88.07	[37]	F; %lipid = 6; %sed OC = 18
	25 µg/kg OC		221 µg/kg lipid				8.84	[37]	F; %lipid = 10; %sed OC = 12
	34 µg/kg OC		287 µg/kg lipid				8.44	[37]	F; %lipid = 10; %sed OC = 12
	144 µg/kg OC		1064 µg/kg lipid				7.39	[37]	F; %lipid = 13; %sed OC = 32
Oncorhynchus, Salmo, Salvelinus sp., Salmonids	667 µg/kg OC		727 µg/kg lipid				1.091	[35]	F; %lipid = 11; %sed OC = 2.7
		0.000019 µg/L	80 µg/kg lipid			6.62		[35]	F; %lipid = 11
Salmonids							1.67	[38]	F
Oncorhynchus kisutch, Coho salmon			95 mg/kg (whole body)[4]	Mortality, ED50				[27]	L; 50% mortality in 31 days

Summary of Biological Effects Tissue Concentrations for p,p′-DDT

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]**
Prosopium williamsoni, Mountain whitefish	244 µg/kg OC		417 µg/kg lipid				1.706	[34]	F; %lipid = 12.0; %sed OC = 0.9
	6,433 µg/kg OC		772 µg/kg lipid				0.120	[34]	F; %lipid = 12.25; %sed OC = 0.30
Corogonus autumnalis, Omul (endemic whitefish)		particulate: 5.1pg/L ± 2.3 n = 7 dissolved: 50 pg/L ± 23 n = 7	0.16-0.27 mg/kg[5] lipid (whole body) n = 2					[16]	F; Lake Baikal, Siberia
Salmo salar, Atlantic salmon			3 mg/kg (whole body)[4]	Morphology, NOED				[26]	L; no effect on metabolic rate or growth, resd_conc_wet value range 3.0-5.0
Salvelinus namaycush, Lake trout			3.9 mg/kg (whole body)[4]	Behavior, LOED				[21]	L; temperature selection after 24-hour exposure to chemical

Summary of Biological Effects Tissue Concentrations for p,p′-DDT

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]**
			27.8 mg/kg (whole body)[4]	Behavior, LOED				[21]	L; temperature selection after 24-hour exposure to chemical
Salvelinus namaycush, Lake trout			3.66 mg/kg (whole body)[4]	Mortality, LOED				[28]	L; survival of fry reduced
			2 mg/kg (whole body)[4]	Mortality, LOED				[28]	L; survival of fry reduced
Carassius auratus, Goldfish			5.1 mg/kg (whole body)[4]	Behavior, LOED				[31]	L; behavioral changes, loss of equilibrium, convulsions
Pimephales promelas, Fathead minnow			3.8 mg/kg (whole body)[4]	Reproduction, LOED				[19]	L; significantly different from control (p = 0.05)
			24 mg/kg (whole body)[4]	Reproduction, LOED				[19]	L; significantly different from control (p = 0.05)
Cyprinus carpio, Carp	0.012 mg/kg (n = 1)	Surface water 0.39 ng/L (n = 1)	0.023 ± 0.012 mg/kg (n = 9)					[15]	F; lower Detroit River; value is mean ± SD

Summary of Biological Effects Tissue Concentrations for p,p′-DDT

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]**
Mixed *Catastoma* sp., Suckers	6433 µg/kg OC		869 µg/kg lipid				0.135	[34]	F; %lipid = 9.2; %sed OC = 0.30
Catastoma macrocheilus, Largescale sucker	340 µg/kg OC		811 µg/kg lipid				2.385	[34]	F; %lipid = 11.1; %sed OC = 1.0
Pimelodus albicans, (Marine catfish)	0.4 ng/g dw		0.5 µg/g lipid (n = 7) (muscle)					[14]	F; Rio de La Plata, Argentina; lipid content 4%
Pimelodus albicans, Mandi	40.0 µg/kg OC		500 µg/kg lipid					[14]	F; %lipid = not reported; %sed OC = 1.0
Gambusia affinis, Mosquito fish			18.6 mg/kg (whole body)[4]	Mortality, NOED				[25]	L; no effect on survivorship after 3 days
Leuciscus idus, Golden ide			95 mg/kg (whole body)[4]	Mortality, NOED				[30]	L; no effect on survivorship in 3 days

Summary of Biological Effects Tissue Concentrations for p,p′-DDT

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			4.2 mg/kg (whole body)[4]	Behavior, LOED				[31]	L; behavioral changes, loss of equilibrium, convulsions
Comephorus dybowskii, Pelagic sculpin		particulate: 5.1pg/L ± 2.3 n = 7 dissolved: 50 pg/L ± 23 n = 7	0.52-0.64 mg/kg lipid (whole body) n = 1					[16]	F; Lake Baikal, Siberia
Cottus cognatus, Slimy sculpin	667 µg/kg OC		362 µg/kg lipid				0.544	[35]	F; %lipid = 8; %sed OC = 2.7
		0.000019 µg/L	29 µg/kg lipid			6.18		[35]	F; %lipid = 8
Prochilodus platensis, common name not available	0.4 ng/g dw		Three composite samples (µg/g lipid): 2.4 (n = 4) (muscle) 9.3 (n = 4) (muscle) 4.3 (n = 5) (muscle)					[14]	F; Rio de La Plata, Argentina; lipid content 1-12.7%
Prochilodus platensis, Curimata	40.0 µg/kg OC		5,333.33 µg/kg lipid					[14]	F; %lipid = not reported; %sed OC = 1.0

Summary of Biological Effects Tissue Concentrations for p,p′-DDT

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]**
Wildlife									
Bucephala clangula, Goldeneye	0.012 mg/kg (n = 1)	water = 0.39 ng/L (n = 1)	0.040 mg/kg (whole body) (n = 3)					[15]	F; lower Detroit River; value is mean ± SD
Aythya affinis, Lesser scaup	0.012 mg/kg (n = 1)	surface water 0.39 ng/L (n = 1)	0.025 mg/kg (whole body) (n = 7)					[15]	F; lower Detroit River; value is mean ± SD
Aythya marila, Greater scaup	0.012 mg/kg (n = 1)	surface water 0.39 ng/L (n = 1)	0.040 ± 0.0094 mg/kg (whole body) (n = 3)					[15]	F; lower Detroit River; value is mean ± SD
Falco peregrinus, Peregrine falcon (eggs)			22 ng/g (eggs) (n = 6)	11.4% eggshell thinning				[17]	F; Kola Penninsula, Russia; n = number of clutches sampled
Larus californicus, California gull			440 mg/kg (brain)[4]	Mortality, NA				[18]	L
			183 mg/kg (breast)[4]	Mortality, NA				[18]	L
			3200 mg/kg (liver)[4]	Mortality, NA				[18]	L

Summary of Biological Effects Tissue Concentrations for p,p′-DDT

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]**
Pelecanus occidentalis, Brown pelican			4.55 mg/kg (brain)[4]	Mortality, NA				[18]	L
			66 mg/kg (breast)[4]	Mortality, NA				[18]	L
			7.8 mg/kg (liver)[4]	Mortality, NA				[18]	L
Phalacrocorax penicillatus, Brandts cormorant			230 mg/kg (brain)[4]	Mortality, NA				[18]	L
			500 mg/kg (breast)[4]	Mortality, NA				[18]	L
			840 mg/kg (liver)[4]	Mortality, NA				[18]	L
			810 mg/kg (Liver)[4]	Mortality, NA				[18]	L
Phoca siberica, Baikal seal		particulate: 5.1pg/L ± 2.3 n = 1 dissolved: 50 pg/L ± 23 n = 1	17-21 mg/kg[5] lipid (blubber) n = 1					[16]	F; Lake Baikal, Siberia

[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.
[5] Not clear from reference if concentration is based on wet or dry weight.

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Chemical Category: PESTICIDE (ORGANOPHOSPHATE)

Chemical Name (Common Synonyms): DIAZINON **CASRN:** 333-41-5

Chemical Characteristics

Solubility in Water: 0.004% at 20°C [1]

Half-Life: No data [2]

Log K_{ow}: 3.70 [3]

Log K_{oc}: 3.64 L/kg organic carbon

Human Health

Oral RfD: 9×10^{-4} mg/kg/day [4]

Confidence: —

Critical Effect: Decreased cholinesterase activity

Oral Slope Factor: No data [4,5]

Carcinogenic Classification: No data [4,5]

Wildlife

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

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

Aquatic Organisms

Partitioning Factors: Partitioning factors for diazinon in aquatic organisms were not found in the literature. Log BCFs ranged from 0.69 to 1.23 (invertebrates) and from 1.59 to 2.90 (fishes).

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

Toxicity/Bioaccumulation Assessment Profile

Diazinon is relatively toxic to aquatic organisms. The acute toxicity for aquatic invertebrates ranged from 0.9 µg/L (48-h LC50) for *Daphnia pulex* [6] to 200µg/L (96-h LC50) for *Gammarus lacustris* [7], while chronic toxicity ranged from 0.27 µg/L (30-d LC50) for *Gammarus pseudolimneaus* to 4.6 µg/L (30-d LC50) for *Acroneuria lycorias* [8]. The maximum acceptable concentration (MATC) for diazinon based on the exposure with sheepshead minnows, was 0.47 µg/L [5], and 3.2 µg/L based on the exposure with fathead minnows [9].

The mode of toxic action of organophosphorus compounds is related to the inhibition of acetylcholinesterase in tissue of animals [10]. A representative of organophosphorus insecticides, diazinon shows species-selective toxicity in fish [11]. For example, diazinon was about 10 times more toxic to the guppy than to the zebra fish [12] and 22 times more potent to loach than killifish [10]. Both the guppy and zebra fish metabolized diazinon to 2-isopropyl-6-methyl-4-pyrimidinol (pyrimidinol). The species-specific oxidative transformation of diazinon or inhibition of acetylcholinesterase are responsible for the differences in diazinon toxicity. During the exposure of pretreated fish (guppies and zebra fish) to diazinon [13], the tissue concentration of pyrimidinol initially increased, then declined to very low levels. Keizer et al. [13] hypothesized that the toxicity of diazinon to guppy is due to its metabolism to a highly toxic metabolite, e.g., diazoxon whereas toxicity to zebra fish is related to bioaccumulation of the parent compound. Fish reached an apparent steady state after 48 hours [12] or 96 hours [14].

Diazinon was most rapidly excreted from the gallbladder followed by liver, muscle, and kidney [11]. The slow excretion rate from kidney was probably because diazinon was transported from all parts of the fish to the kidney before excretion [15]. The log BCFs for eels exposed to 56 µg/L of diazinon were 2.90 in liver, 3.20 in muscle, and 3.36 in gill tissue [16]. Diazinon elimination from the selected tissues was rapid; it was not detected in any tissue after 24-hour exposure in clean water [16]. The results of the study by Kanazawa [17] showed that the concentration of diazinon in tissue of the freshwater fish reached a maximum after 4 days and then decreased gradually. The uptake of diazinon by killifish was not influenced if the fish were exposed to the individual pesticide, or to a pesticide mixture [18].

Diazinon was identified as a major toxicant in municipal effluents [19], indicating persistence of this pesticide in the environment. According to Lee et al. [20], the toxicity of diazinon can be induced by dissolved organic materials.

Summary of Biological Effects Tissue Concentrations for Diazinon

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									
Cipangopoludina malleata, Pond snail		10 µg/L			0.77			[21]	L
Procambarus clarkii, Crayfish		10 µg/L			0.69			[21]	L
Indoplanorbis exustus, Red snail		10 µg/L			1.23			[21]	L
Fishes									
Pseudorasbora parva, Topmouth gudgeon		50 µg/L	11.3 ng/g		2.32			[22]	L
Anguilla anguilla, Eel		10 µg/L	80 ng/g (liver)		2.90			[16]	L
Anguilla anguilla, Eel		10 µg/L	160 ng/g (muscle)		2.90			[16]	L
Gnathopogon caerulescens, Willow shiner					2.39			[23]	F

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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]**
Pseudorasbora parva, Topmouth gudgeon					2.18			[23]	F
Pseudorasbora parva, Motsugo		0.7 mg/kg	211 mg/kg (4-day) 17 mg/kg (30 day)	Bleeding, abnormal swimming	1.81			[17]	L
Brachydanio rerio, Zebra fish	Ingestion		1,550 mg/kg (whole body)[4]	Mortality, ED100				[13]	L; Lethal body burden
Zacco slatypus, Pale chub					2.18			[23]	F
Plecoglossus altivelis, Ayu sweetfish					1.79			[23]	F
Cyprinodon variegatus, Sheepshead minnow		1.8 µg/L	0.26 mg/kg in 4d, 0.11 mg/kg in 7d, 0.31 mg/kg in 14d		2.17			[14]	L
		3.5 µg/L	0.38 mg/kg in 4d, 0.21 mg/kg in 7d, 0.49 mg/kg in 14d		2.17			[14]	L
		6.5 µg/L	1.3 mg/kg in 4d, 0.5 mg/kg in 7d, 1.4 mg/kg in 14d		2.33			[14]	L
Cyprinodon variegatus, Sheepshead minnow			0.3 mg/kg (whole body)[4]	Morphology, LOED				[14]	L; body darkened, lateral curvature of body

Summary of Biological Effects Tissue Concentrations for Diazinon

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]
			1.4 mg/kg (whole body)[4]	Morphology, not applicable				[14]	L; body darkened, lateral curvature of body
			0.5 mg/kg (whole body)[4]	Morphology, not applicable				[14]	L; body darkened, lateral curvature of body
			0.05 mg/kg (whole body)[4]	Mortality, NOED				[14]	L; no effect on morphology or appearance
			1.4 mg/kg (whole body)[4]	Mortality, NOED				[14]	L; no effect on mortality
			0.5 mg/kg (whole body)[4]	Mortality, NOED				[14]	L; no effect on mortality
			0.3 mg/kg (whole body)[4]	Mortality, NOED				[14]	L; no effect on mortality
			0.05 mg/kg (whole body)[4]	Mortality, NOED				[14]	L; no effect on mortality
			0.05 mg/kg (whole body)[4]	Physiological, LOED				[14]	L; inhibition of acetylcholinesterase activity
			1.4 mg/kg (whole body)[4]	Physiological, NA				[14]	L; 71% inhibition of acetylcholinesterase activity
			0.5 mg/kg (whole body)[4]	Physiological, NA				[14]	L; inhibition of acetylcholinesterase activity
			0.3 mg/kg (whole body)[4]	Physiological, NA				[14]	L; inhibition of acetylcholinesterase activity
			1.4 mg/kg (whole body)[4]	Reproduction, ED50				[14]	L; 45-55% reduction in average number of eggs produced

Summary of Biological Effects Tissue Concentrations for Diazinon

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]
			0.5 mg/kg (whole body)[4]	Reproduction, ED50				[14]	L; 45-55% reduction in average number of eggs produced
			0.3 mg/kg (whole body)[4]	Reproduction, ED50				[14]	L; 45-55% reduction in average number of eggs produced
			0.05 mg/kg (whole body)[4]	Reproduction, LOED				[14]	L; statistically significant reduction in number of eggs produced
Poecilia reticulata, Guppy		0.8 mg/L	25.8 µg/g in 24h, 90.3 µg/g in 48h, 167.7 µg/g in 96h, 109 mg/kg (whole body)[4]	Mortality, ED100				[13]	L; lethal body burden
Poecilia reticulata, Guppy			2,430 mg/kg (whole body)[4]	Mortality, ED100				[24]	L; lifestage: 2-3 months
			2,430 mg/kg (whole body)[4]	Mortality, ED100				[24]	L; lifestage: 2-3 months

[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: PESTICIDE (ORGANOCHLORINE)

Chemical Name (Common Synonyms): DICOFOL **CASRN:** 115-32-2

Chemical Characteristics

Solubility in Water: 1.2 mg/L at 20° C (99% purity) [1]

Half-Life: No data [2]

Log K_{ow}: No data [3]

Log K_{oc}: —

Human Health

Oral RfD: 1 x 10^{-3} mg/kg/day [4]

Confidence: —

Critical Effect: Increase in liver to body weight ratios in rats

Oral Slope Factor: No data [5]

Carcinogenic Classification: C [6]

Wildlife

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

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

Aquatic Organisms

Partitioning Factors: Log BCFs ranging from 4.02-4.16 were reported in a study exposing fathead minnows to dicofol [10].

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

Toxicity/Bioaccumulation Assessment Profile

Dicofol is an organochlorine compound used as a miticide. The principal commercial dicofol product, Kelthane, is made from DDT [7]. Clark et al. [7] reported reduction in eggshell weight and thickness of American kestrels due to dicofol. They also observed that 10 µg/g of dicofol reduced hatchability of eggs. They suggested that dicofol concentrations above 3 µg/g in food may affect bird reproduction. The 48-h and 100-h LC50s for grass shrimp (*Crangon franciscorum*) exposed to dicofol (Kelthane) were 590 and 100 µg/L, respectively [8].

The major metabolite of dicofol is 1,1-bis(4-chlorophenol)2,2dichloroethanol (pp-DCD) [9]. Because dicofol is more lipophilic than its metabolites, it was abundant in every tissue except for liver and brain. The dicofol metabolites are less toxic than dicofol and they have less impact on the formation of normal eggshells by doves [9]. The bioconcentration of dicofol in fathead minnows was reduced by 35 percent by clay particles (65 mg/L) indicating that more than 30 percent of the dicofol sorbed onto clay and was biologically unavailable to the fish [10]. Bioconcentration factors at the two dicofol concentrations were not significantly different and steady-state concentrations occured with 40 to 60 days of exposure at 10,500 to 13,900 times water levels.

Summary of Biological Effects Tissue Concentrations for Dicofol

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									
Pimephales promelas, Fathead minnow		12.38 µg/L			4.02-4.16			[10]	L
		1.15 µg/L			4.12-4.14			[10]	L
Wildlife									
Streptopelia risoria, Ring neck dove	32 mg/kg (diet)		116.5µg/g in fat 1.07µg/g in liver 4.55µg/g in heart 0.37µg/g in brain					[9]	L

[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: PESTICIDE (ORGANOCHLORINE)

Chemical Name (Common Synonyms): DIELDRIN **CASRN:** 60-57-1

Chemical Characteristics

Solubility in Water: 186 µg/L at 25°C [1]

Half-Life: 175 days to 3 years, based on unacclimated aerobic soil grab sample data and reported half-life in soil based on field data [2]

Log K_{ow}: 5.37 [3]

Log K_{oc}: 5.28 L/kg organic carbon

Human Health

Oral RfD: 5 x 10^{-5} mg/kg/day [4]

Confidence: Medium, uncertainty factor = 100

Critical Effect: Liver lesions (focal proliferation and focal hyperplasia) in rats, liver carcinomas in mice

Oral Slope Factor: 1.6 x 10^{+1} per (mg/kg)/day [4]

Carcinogenic Classification: B2 [4]

Wildlife

Partitioning Factors: Log BCFs for tadpole and juvenile frogs have been measured at 2.20 to 3.33, whereas log BCFs for adult frogs were 1.57 to 2.58. Dieldrin appears to bioconcentrate to a lesser extent in frogs than in fish. Mallard ducklings exposed to dieldrin-contaminated water for drinking and swimming had log BCFs ranging from 1.69 to 2.21 in liver, 0.98 to 1.97 in muscle, 2.25 to 2.84 in skin, and 2.85 to 3.30 in lipid. Mallard ducklings exposed for longer periods had log BCFs up to 9.30. BSAFs were calculated for red-winged blackbirds and tree swallow eggs during a study in the Great Lakes area, with values ranging from 7.5 to 448, as reported in the attached summary table. The BSAF for tree swallow nestings was 341.

Food Chain Multipliers: A biomagnification factor of 16 has been reported for dieldrin for herring gulls feeding on alewife in Lake Ontario [5]. A study of arctic marine food chains measured biomagnification factors for dieldrin that ranged from 2.2 to 2.4 for fish to seal, 4.9 to 5.5 for seal to bear, and 11.4 for fish to bear [6].

Aquatic Organisms

Partitioning Factors: In older studies, the following log BCFs have been reported for dieldrin: 4.51 in freshwater alga [7]; from 3.38 to 4.83 in fish [8]; and log 3.20 in a saltwater mussel [9]. A log BCF of 5.36 was found for rainbow trout [34]. BSAFs ranging from 1.120 to 7.134 were reported to bivalves [33].

Food Chain Multipliers: Food chain multipliers (FCMs) for trophic level 3 aquatic organisms were 8.6 (all benthic food web), 1.2 (all pelagic food web), and 5.5 (benthic and pelagic food web). FCMs for trophic level 4 aquatic organisms were 10.8 (all benthic food web), 1.9 (all pelagic food web), and 5.8 (benthic and pelagic food web) [36].

Toxicity/Bioaccumulation Assessment Profile

Dieldrin is the name of an insecticide that was used in the United States for locust and mosquito control until production and importation were banned. In addition to man-made production, dieldrin is derived from the oxidation of aldrin, which is also an insecticide. Aldrin is readily converted to dieldrin under normal environmental conditions [10]. In addition, aldrin is readily metabolized to dieldrin, so the effects seen in animals exposed to aldrin may be caused by dieldrin [11]. Dieldrin is one of the most persistent of the chlorinated hydrocarbons, and is highly resistant to biodegradation and abiotic degradation. In water, volatilization of dieldrin to the atmosphere is probably an important process, but transformation processes in soils and sediment are slow. Dieldrin sorbs tightly to soil and sediment, particularly if substantial amounts of organic carbon are present.

Dieldrin is toxic to aquatic organisms, birds, and mammals and is capable of producing carcinogenic, teratogenic, and reproductive effects [10]. Teratogenic effects include cleft palate, webbed foot, and skeletal anomalies. Reproductive effects include decreased fertility, increased fetal death, and effects on gestation [10].

In aquatic organisms, the acute toxicity of dieldrin ranges from 0.5 to 740 µg/L for freshwater and 0.7 to >100 µg/L for saltwater organisms [12]. Differences between dieldrin concentrations causing acute lethality and chronic toxicity in species acutely sensitive to this insecticide are small; acute-chronic ratios ranged from 2.4 to 12.8 for three species [12]. Dieldrin is generally an order of magnitude more toxic to fish than is aldrin [11]. LC50s for freshwater and saltwater aquatic invertebrates exposed to sediment spiked with dieldrin in the laboratory have been shown to range from 0.0041 to 386 µg/g dw [12]. Bioconcentration factors for dieldrin in various aquatic organisms range from 400 to 68,000 [8], indicating that dieldrin will show moderate to significant bioaccumulation in various aquatic species.

Mammals appear to be more sensitive to dieldrin poisoning than birds. Brain concentrations associated with lethality in mammals are 5 mg/kg and in birds are 10 mg/kg [11]. Concentrations as low as 1 mg/kg in the brain might trigger irreversible starvation in sensitive individuals of birds [13]. Major effects on reproduction in wildlife are not expected to occur at dieldrin concentrations of less than one half those causing mortality [11]. Dieldrin is commonly found in the brain, tissues, and eggs of fish-eating birds that also have residues of organochlorines such as DDE and PCBs. Based on a number of literature studies, the State of New York proposed a dietary fish flesh criterion of 0.12 mg/kg to protect piscivorous wildlife [14]. There are limited studies relating aldrin concentrations to toxicity because of the rapid conversion of aldrin into dieldrin.

Summary of Biological Effects Tissue Concentrations for Dieldrin

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, Eastern oyster			107 mg/kg (whole body)[4]	Cellular, NOED				[23]	L; no histological effects on structure of gill, gut or mantle
			11 mg/kg (whole body)[4]	Cellular, NOED				[23]	
			1.03 mg/kg (whole body)[4]	Cellular, NOED				[23]	
Crassostrea virginica, Eastern oyster			1.44 mg/kg (whole body)[4]	Behavior, LOED				[29]	L; erratic shell movements, extended shell closure indicated irritation
			18.6 mg/kg (whole body)[4]	Behavior, NA				[29]	
			1.44 mg/kg (whole body)[4]	Mortality, NOED				[29]	L; no effect on mortality within 168 hours
			18.6 mg/kg (whole body)[4]	Mortality, NOED				[29]	
Crassostrea virginica, Eastern oyster			13.9 mg/kg (whole body)[4]	Growth, NOED				[31]	L; estimated NOED - no statistical summary in text

Summary of Biological Effects Tissue Concentrations for Dieldrin

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]**
Macomona liliana, Mollusk	73.3 µg/kg OC		201.7 µg/kg lipid				2.752	[33]	F, %lipid = 2.95; %sed OC = 0.30
	52.1 µg/kg OC		371.7 µg/kg lipid				7.134	[33]	F, %lipid = 2.33; %sed OC = 0.73
	72.7 µg/kg OC		172.0 µg/kg lipid				2.366	[33]	F, %lipid = 2.57; %sed OC = 0.22
	60.0 µg/kg OC		76.0 µg/kg lipid				1.267	[33]	F, %lipid = 2.04; %sed OC = 0.25
	20.8 µg/kg OC		80.2 µg/kg lipid				3.856	[33]	F, %lipid = 3.13; %sed OC = 0.48
Austrovenus stutchburyi, Mollusk	73.3 µg/kg OC		102.7 µg/kg lipid				1.401	[33]	F, %lipid = 5.62; %sed OC = 0.30
	52.1 µg/kg OC		127.6 µg/kg lipid				2.449	[33]	F, %lipid = 5.21; %sed OC = 0.73
	72.7 µg/kg OC		105.2 µg/kg lipid				1.447	[33]	F, %lipid = 4.85; %sed OC = 0.22
	60.0 µg/kg OC		67.2 µg/kg lipid				1.120	[33]	F, %lipid = 3.87; %sed OC = 0.25
	20.8 µg/kg OC		58.6 µg/kg lipid				2.817	[33]	F, %lipid = 4.27; %sed OC = 0.48
Mercenaria mercenaria, Quahog clam			0.38 mg/kg (whole body)[4]	Behavior, NOED				[22]	L; no effect on feeding activity

Summary of Biological Effects Tissue Concentrations for Dieldrin

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]**
Mya arenaria, Soft shell clam			0.87 mg/kg (whole body)[4]	Behavior, NOED				[22]	L; no effect on feeding activity
Chlamydotheca arcuata, Ostracod			1 mg/kg (whole body)[4]	Mortality, LOED				[28]	L; immobility, mortality, resd_conc_wet value > 1.0
Palaemonetes pugio, Grass shrimp			2.1 mg/kg (whole body)[4]	Mortality, LOED				[31]	L; estimated loed - no statistical summary in text
			0.09 mg/kg (whole body)[4]	Mortality, NOED				[31]	L; estimated noed - no statistical summary in text
Penaeus duorarum, Pink shrimp			0.23 mg/kg (whole body)[4]	Mortality, ED50				[31]	L; ED50 via Spearman Karber 1.5 (msl)
			0.08 mg/kg (whole body)[4]	Mortality, LOED				[31]	L; estimated LOED - no statistical summary in text
			0.01 mg/kg (whole body)[4]	Mortality, NOED				[31]	L; estimated NOED - no statistical summary in text
Chironomus riparius, Midge			1.9 mg/kg (whole body)[4]	Mortality, ED10				[24]	L; all larvae moribund in 2 hours

Summary of Biological Effects Tissue Concentrations for Dieldrin

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]**
			1.1 mg/kg (whole body)[4]	Behavior, ED50				[24]	L; 50 - 75% mortality, lethargy within 2 hours
			1.1 mg/kg (whole body)[4]	Mortality, ED50				[24]	
			1.1 mg/kg (whole body)[4]	Behavior, ED50				[24]	L; 50 - 75% mortality, lethargy within 2 hours
			1.1 mg/kg (whole body)[4]	Mortality, ED50				[24]	
Fishes									
Squalus acanthias, Spiny dogfish			1 mg/kg (whole body)[4]	Mortality, NOED				[27]	L; no effect on mortality in 24 hours
Oncorhynchus mykiss, Rainbow trout			0.14 mg/kg (fat)[4]	Physiological, ED30				[32]	L; 30% decrease in hemoglobin content relative to control
			0.14 mg/kg (fat)[4]	Physiological, ED30				[32]	L; 30% increase in liver size relative to control
			0.05 mg/kg (fat)[4]	Physiological, ED35				[32]	L; 35% increase in kidney size relative to control
			0.14 mg/kg (fat)[4]	Growth, ED40				[32]	L; 40% decrease in growth relative to control

Summary of Biological Effects Tissue Concentrations for Dieldrin

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 (juveniles)					5.36			[34]	L
Salmonids							6.65	[35]	F
Carassius auratus, Goldfish			3.8 mg/kg (whole body)[4]	Behavior, LOED				[26]	L; hyperexcit-ability
Leuciscus idus, Golden ide			151 mg/kg (whole body)[4]	Mortality, NOED				[25]	L; no effect on survivorship in 3 days
Cyprinodon variegatus, Sheepshead minnow			52.9 mg/kg (whole body)[4]	Mortality, ED50				[31]	L; ED50 via Spearman Karber 1.5 (msl)
			34 mg/kg (whole body)[4]	Mortality, LOED				[31]	L; estimated NOED - no statistical summary in text
			12.8 mg/kg (whole body)[4]	Mortality, NOED				[31]	
Gambusia affinis, Mosquito fish			28 mg/kg (whole body)[4]	Mortality, NOED				[30]	L; no effect on survivorship after 3 days

Summary of Biological Effects Tissue Concentrations for Dieldrin

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]**
Poecilia reticulata, Guppy			10.7 mg/kg (whole body)[4]	Growth, NA				[21]	L; decreased biomass of guppy population in laboratory ecosystem
Lepomis macrochirus, Bluegill			3.7 mg/kg (whole body)[4]	Behavior, LOED				[26]	L; behavioral changes, loss of equilibrium, convulsions
Wildlife									
Xenopus laevis, African clawed frog (tadpole stage)		water exposure 2.3±0.2 µg/L	0.7 mg/kg[5] (whole body)		2.48			[15]	L; 28-day exposure; insufficient tissue for replicates; values are mean ± SE
		water exposure 1.1±0.1 µg/L	1.8±1.2 mg/kg[5] (whole body)		3.21±3.04			[15]	L; 28-day exposure
		(water exposure) µg/L 2.0±0.0 4.2±0.1 9.3±0.2 20.5±0.4	mg/kg[5] (whole body): 0.8 20.0±0 3.0±0.6 7.0		 2.60 2.68±0 2.51±0.07 2.53			[15]	L; 28-day exposure; insufficient tissue for replicates for all exposures; values are mean ± SE
		µg/L (water exposure): 0.9±0.1 1.8±0.2 3.8±0.3 9.7±0.4	mg/kg[5] (whole body) 0.4±0 0.8±0.2 1.5±0.5 3.0±1.0	 NOAEL LOAEL	 2.67±0 2.62±1.92 2.59±2.11 2.49±2.01			[15]	L; 24-day exposure; values are mean ± SE; effects based on mortality

Summary of Biological Effects Tissue Concentrations for Dieldrin

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]**
Xenopus laevis, African clawed frog (tadpole stage)		water exposure 5.5 µg/L	1.8 mg/kg[5] (whole body)	LC50				[15]	L; 24-day exposure; LC50 tissue dieldrin estimated by graphical extrapolation
Xenopus laevis, African clawed frog (juvenile stage)		water exposure 2.1±0.2 µg/L	4.5±0.3 mg/kg[5] (whole body)		3.33±2.38			[15]	L; 28-day exposure; values are mean ± SE
Rana pipiens, Leopard frog (tadpole stage)		water exposure 0.8±0.1 µg/L	0.6±0.2 mg/kg[5] (whole body)		2.84±2.28			[15]	L; 28-day exposure; values are mean ± SE
		water exposure 2.1±0.1 µg/L	0.8±0.1 mg/kg[5] (whole body)		2.59±1.60			[15]	L; 28-day exposure; values are mean ± SE
		water exposure (µg/L)	whole body[5] (mg/kg)					[15]	L; 28-day exposure; values are mean ± SE; effects based on mortality
		0.8±0.1	0.4±0.1	NOAEL	2.64±1.18				
		1.9±0.2	0.4±0	LOAEL	2.32±0				
		4.1±0.3	0.6±0.1		2.20±1.08				
		10.0±0.3	2.0±0.1		2.30±0				
Rana pipiens, Leopard frog (tadpole stage)		water exposure 8.3 µg/L	1.7 mg/kg[5] (whole body)	LC50				[15]	L; 24-day exposure; LC50 tissue dieldrin estimated by graphical extrapolation

Summary of Biological Effects Tissue Concentrations for Dieldrin

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]**
Rana pipiens, Leopard frog (adult stage)		water exposure 10.7±1.3 µg/L	0.4±0.4 mg/kg[5] (skin) 0.9±0.1 mg/kg[5] (muscle) 1.5±0.5 mg/kg[5] (liver)		1.57±1.57 1.92±0.95 2.15±1.67			[15]	L; 28-day exposure; values are mean ± SE
		water exposure 56.2±4.1 µg/L	7.3±2.8 mg/kg[5] (skin) 17.8±7.8 mg/kg[5] (muscle) 21.5±3.3 mg/kg[5] (liver)		2.11±1.69 2.51±2.15 2.58±1.64			[15]	L; 28-day exposure; values are mean ± SE
		water exposure 53.4 µg/L	5.5 mg/kg[5] (skin) 10.0 mg/kg[5] (muscle) 13.0 mg/kg[5] (liver)	LC50 LC50 LC50				[15]	L; 28-day exposure; LC50 tissue dieldrin estimated by graphical extrapolation
Anas platyrhynchos, Mallard (ducklings)		0.014±1 mg/L	24.5 mg/kg (lipid) 2.3 mg/kg (liver) 1.3 mg/kg (muscle)	No mortality or effects on behavior or survival observed	3.24			[18]	L; 34-day exposure; 1-day-old birds had access to dieldrin-contaminated water for drinking and swimming
		0.052±4 mg/L	68.9 mg/kg (lipid) 3.4 mg/kg (liver) 1.15 mg/kg (muscle)	No mortality or effects on behavior or survival observed	3.12				

Summary of Biological Effects Tissue Concentrations for Dieldrin

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]**
		0.118±11 mg/L	128 mg/kg (lipid) 7.4 mg/kg (liver) 1.1 mg/kg (muscle)	No mortality or effects on behavior or survival observed	3.04				
Anas platyrhynchos, Mallard (ducklings)		0.019±2 mg/L	37.9 mg/kg (lipid) 13 mg/kg (skin) 1.9 mg/kg (liver)	No mortality or effects on behavior or survival observed	3.30			[18]	L; 24-day exposure; 1-day old birds had access to dieldrin-contaminated water for drinking and swimming
		0.075±1 mg/L	107 mg/kg (lipid) 39.5 mg/kg (skin) 4.8 mg/kg (liver)	No mortality or effects on behavior or survival observed	3.15				
		0.193±8 mg/L	217 mg/kg (lipid) 75 mg/kg (skin) 11.3 mg/kg (liver)	No mortality or effects on behavior or survival observed	3.05				
		0.177±11 mg/L	125 mg/kg (lipid) 31.5 mg/kg (skin) 8.6 mg/kg (liver) 2.3 mg/kg (brain) 0.97 mg/kg muscle) 0.97 mg/kg (blood)		2.84 2.25 1.69 1.11 0.74 0.51			[18]	L; 8-day exposure; 14-day old birds had access to dieldrin-contaminated water for drinking and swimming

Summary of Biological Effects Tissue Concentrations for Dieldrin

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]**
			915 mg/kg (lipid) 305 mg/kg (skin) 52 mg/kg (liver)	96-Hour LC50	0.74 0.26 −0.52			[18]	L; 24-day exposure; birds were dosed with food spiked with dieldrin at measured concentrations of 0.3 to 165 mg/kg.
			395 mg/kg (lipid) 193 mg/kg (skin) 12 mg/kg (liver) 5 mg/kg (brain) 2 mg/kg (muscle)	24-Day LC50	1.13 0.81 −0.40 −0.70 −1.00				
			180 mg/kg (lipid) 102 mg/kg (skin) 7 mg/kg (liver) 2.5 mg/kg (brain) <1 mg/kg (muscle)	24-Day LOAEL	1.05 0.81 −0.40 −0.70				
			4 mg/kg (lipid) 2 mg/kg (skin) <1 mg/kg (liver) <1 mg/kg (brain) <1 mg/kg (muscle)	24-Day NOAEL	1.12 0.83				
Falco peregrinus, Peregrine falcon (eggs)			59 ng/g (eggs) (n = 6)	11.4% eggshell thinning				[19]	F; Kola Peninsula, Russia; n = number of clutches sampled
Falco tinnunculus, European kestrel			6-30 mg/kg (liver)	mortality				[16]	F

Summary of Biological Effects Tissue Concentrations for Dieldrin

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]**
Agelaius phoeniceus, Red-winged blackbird	1.2 ng/g TOC=21.0%		16.6 ng/g				7.5	[20]	F; Great Lakes/St. Lawrence River basin; 12 wetlands sites; sediment concentration reported as wet weight concentration which may be a typographical error. 203.7 117.6 ng/g ww
(eggs)	11.0 ng/g TOC=7.5%		31.0 ng/g				21		
	127.8 ng/g TOC=12%		84.6 ng/g				7.8		
	0.6 ng/g TOC=18.5%		8.9 ng/g				57.2		
	0.7 ng/g TOC=11.5%		9.1 ng/g				31.1		
	0.1 ng/g TOC=10.5%		20.0 ng/g				448		
Tachycineta bicolor, Tree swallow			(whole body minus feet, beak, wings, and feathers)					[20]	F; Great Lakes/ St. Lawrence River basin; 12 wetlands sites; sediment concentration reported as wet weight concentration which may be a typographical error.
(nestlings)	0.7 ng/g TOC=11.5%		211.4 ng/g				340.5		
(eggs)	0.7 ng/g TOC=11.5%		19.3 ng/g				36.9		

Summary of Biological Effects Tissue Concentrations for Dieldrin

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]**
Tyto alba, Barn owl			6-44 mg/kg (liver)	mortality				[17]	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.
[5] Not clear from reference if concentration is based on wet or dry weight.

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Chemical Category: PESTICIDE (ORGANOPHOSPHATE)

Chemical Name (Common Synonyms): DISULFOTON **CASRN:** 298-04-4

Chemical Characteristics

Solubility in Water: 25 ppm at 23°C [1]

Half-Life: 3 days - 21 days based on aerobic soil field data [2]

Log K_{ow}: 3.98 [3]

Log K_{oc}: 3.91 L/kg organic carbon

Human Health

Oral RfD: 4 X 10^{-5} mg/kg/day [4]

Confidence: Medium, uncertainty factor = 1000 [4]

Critical Effect: Cholinesterase inhibition and optic nerve degeneration in dogs

Oral Slope Factor: No data [4]

Carcinogenic Classification: D [6]

Wildlife

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

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

Aquatic Organisms

Partitioning Factors: Partitioning factors for disulfoton in aquatic organisms were not found in the literature.

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

Toxicity/Bioaccumulation Assessment Profile

The toxicity of insecticidally active organophosphorus compounds like disulfoton to animals is attributed to their ability to inhibit acetylcholinesterase, which is a class of enzymes that catalyzes the hydrolysis of the neurotransmitting agent acetylcholine [7].

Disulfoton is relatively toxic to aquatic organisms. The acute toxicity for aquatic invertebrates ranged from 5 µg/L (96-h LC50) for *Pteronarcys californica* [8] to 52 µg/L (96-h LC50) for *Gammarus lacustris* [9], while chronic toxicity ranged from 1.4 µg/L (30-d LC50) for *Acroneuria pacifica* to 1.9 µg/L (30-d LC50) for *Pteronarcys californica* [10]. Fish are less sensitive to disulfoton. The 96-h LC50 based on the exposure with fathead minnows was 3700 µg/L [11]. The toxicity of disulfoton and its most important degradation products were measured using *Daphnia magna* [12]. The toxicity of disulfoton (24-h LC50 of 55 µg/L) was similar to two of its degradation products (disulfoton-sulfoxide and disulfoton). The remaining degradation products were much less toxic than the parent compound.

Summary of Biological Effects Tissue Concentrations for Disulfoton

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

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Chemical Category: POLYCHLORINATED DIBENZOFURANS

Chemical Name (Common Synonyms): **CASRN:** 70648-26-9
1,2,3,4,7,8-HEXACHLORODIBENZOFURAN

Chemical Characteristics

Solubility in Water: No data [1]

Half-Life: No data [2]

Log K_{ow}: No data [3]

Log K_{oc}: —

Human Health

Oral RfD: No data [4]

Confidence: —

Critical Effect: —

Oral Slope Factor: No data [4]

Carcinogenic Classification: —

Wildlife

Partitioning Factors: Partitioning factors for 1,2,3,4,7,8-hexaCDF in wildlife were not found in the studies reviewed.

Food Chain Multipliers: Limited information was found reporting on specific biomagnification factors of PCDDs and PCDFs through terrestrial wildlife. Due to the toxicity, high K_{ow} values, and highly persistent nature of the PCDDs and PCDFs, they possess a high potential to bioaccumulate and biomagnify through the food web. PCDDs and PCDFs have been identified in fish and wildlife throughout the global aquatic and marine environments [5]. Studies conducted in Lake Ontario indicated that biomagnification of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD) appears to be significant between fish and fish-eating birds but not between fish and their food. When calculated for older predaceous fish such as lake-trout-eating young smelt, the biomagnification factor (BMF) can equal 3. The BMF from alewife to herring gulls in Lake Ontario was 32 for 2,3,7,8-TCDD [6]. Log BMFs of 1.70 to 1.81 were reported for mink from 1,2,3,4,7,8-hexaCDF-contaminated diet exposures.

EPA has developed risk-based concentrations of 2,3,7,8-TCDD in different media that present low and high risk to fish, mammalian, and avian wildlife. These concentrations were developed based on toxic effects of 2,3,7,8-TCDD and its propensity to bioaccumulate in fish, mammals, and birds.

Environmental Concentrations Associated With 2,3,7,8-TCDD Risk to Aquatic Life and Associated Wildlife [7]

Organism	Fish Concentration (pg/g)	Sediment Concentration (pg/g dry wt.)	Water Concentration (pg/L)	
			POC=0.2	POC=1.0
Low Risk				
Fish	50	60	0.6	3.1
Mammalian Wildlife	0.7	2.5	0.008	0.04
Avian Wildlife	6	21	0.07	0.35
High Risk to Sensitive Species				
Fish	80	100	1	5
Mammalian Wildlife	7	25	0.08	0.4
Avian Wildlife	60	210	0.7	3.5

Note: POC - Particulate organic carbon
Fish lipid of 8% and sediment organic carbon of 3% assumed where needed.
For risk to fish, BSAF of 0.3 used; for risk to wildlife, BSAF of 0.1 used.
Low risk concentrations are derived from no-effects thresholds for reproductive effects (mortality in embryos and young) in sensitive species.
High risk concentrations are derived from TCDD doses expected to cause 50 to 100% mortality in embryos and young of sensitive species.

Aquatic Organisms

Partitioning Factors: Partitioning factors for 1,2,3,4,7,8-hexaCDF in aquatic organisms were not found in the studies reviewed.

Food Chain Multipliers: No specific food chain multipliers were identified for 1,2,3,7,8-hexaCDF. Food chain multiplier information was only available for 2,3,7,8-TCDD. Biomagnification of 2,3,7,8-TCDD does not appear to be significant between fish and their prey. Limited data for the base of the Lake Ontario lake trout food chain indicated little or no biomagnification between zooplankton and forage fish. BMFs based on fish consuming invertebrate species are probably close to 1.0 because of the 2,3,7,8-TCDD biotransformation by forage fish. BMFs greater than 1.0 might exist between some zooplankton species and their prey due to the lack of 2,3,7,8-TCDD biotransformation in invertebrates [7]. Log BMFs of 1.70 to 1.81 were determined for mink [13].

Toxicity/Bioaccumulation Assessment Profile

The polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) each consist of 75 isomers that differ in the number and position of attached chlorine atoms. The PCDDs and PCDFs are polyhalogenated aromatic compounds and exhibit several properties common to this group of compounds. These compounds tend to be highly lipophilic and the degree of lipophilicity is increased with increasing ring chlorination [5]. In general, the PCDDs and PCDFs exhibit relative inertness to acids, bases, oxidation, reduction, and heat, increasing in environmental persistence and chemical stability

with increasing chlorination [8,5]. Because of their lipophilic nature, the PCDDs and PCDFs have been detected in fish, wildlife, and human adipose tissue, milk, and serum [5].

Each isomer has its own unique chemical and toxicological properties. The most toxic of the PCDD and PCDF isomers is 2,3,7,8-TCDD, one of the 22 possible congeners of tetrachlorodibenzo-*p*-dioxin [9]. Toxicity equivalency factors (TEFs) have been developed by EPA relating the toxicities of other PCDD and PCDF isomers to that of 2,3,7,8-TCDD [9]. The biochemical mechanisms leading to the toxic response resulting from exposure to PCDDs and PCDFs are not known in detail, but experimental data suggest that an important role in the development of systemic toxicity resulting from exposure to these chemicals is played by an intracellular protein, the Ah receptor. This receptor binds halogenated polycyclic aromatic molecules, including PCDDs and PCDFs. In several mouse strains, the expression of toxicity of 2,3,7,8-TCDD-related compounds, including cleft palate formation, liver damage, effects on body weight gain, thymic involution, and chloracnegenic response, has been correlated with their binding affinity for the Ah receptor, and with their ability to induce several enzyme systems [9].

Toxicity Equivalency Factors (TEF) for PCDD and PCDF Isomers [9]

Isomer	TEF
Total TetraCDD	1
2,3,7,8-TCDD	1
Other TCDDs	0.01
Total PentaCDDs	0.5
2,3,7,8-PentaCDDs	0.5
Other PentaCDDs	0.005
Total HexaCDDs	0.04
2,3,7,8-HexaCDDs	0.04
Other HexaCDDs	0.0004
Total HeptaCDDs	0.001
2,3,7,8-HeptaCDDs	0.001
Other HeptaCDDs	0.00001
Total TetraCDFs	0.1
2,3,7,8-TetraCDF	0.1
Other TetraCDFs	0.001
Total PentaCDFs	0.1
2,3,7,8-PentaCDFs	0.1
Other PentaCDFs	0.001
Total HexaCDFs	0.01
2,3,7,8-HexaCDFs	0.01
Other HexaCDFs	0.0001
Total HeptaCDFs	0.001
2,3,7,8-HeptaCDFs	0.001
Other HeptaCDFs	0.00001

In natural systems, PCDDs and PCDFs are typically associated with sediments, biota, and the organic carbon fraction of ambient waters [7]. Congener-specific analyses have shown that the 2,3,7,8-substituted PCDDs and PCDFs were the major compounds present in most sample extracts [5]. Results from limited epidemiology studies are consistent with laboratory-derived threshold levels to 2,3,7,8-TCDD impairment of reproduction in avian wildlife. Population declines in herring gulls (*Larus argentatus*) on Lake Ontario during the early 1970s coincided with egg concentrations of 2,3,7,8-TCDD and related chemicals expected to cause reproductive failure based on laboratory experiments (2,3,7,8-TCDD concentrations in excess of 1,000 pg/g). Improvements in herring gull reproduction through the mid-1980s were correlated with declining 2,3,7,8-TCDD concentrations in eggs and lake sediments [7]. Based on limited information on isomer-specific analysis from animals at different trophic levels, it appears that at higher trophic levels, i.e., fish-eating birds and fish, there is a selection of the planar congeners with the 2,3,7,8-substituted positions [10].

PCDDs and PCDFs are accumulated by aquatic organisms through exposure routes that are determined by the habitat and physiology of each species. With log K_{ow}>5, exposure through ingestion of contaminated food becomes an important route for uptake in comparison to respiration of water [7]. The relative contributions of water, sediment, and food to uptake of 2,3,7,8-TCDD by lake trout in Lake Ontario was examined by exposing yearling lake trout to Lake Ontario smelt and sediment from Lake Ontario along with water at a 2,3,7,8-TCDD concentration simulated to be at equilibrium with the sediments. Food ingestion was found to contribute approximately 75 percent of total 2,3,7,8-TCDD [7]. There have been a number of bioconcentration studies of 2,3,7,8-TCDD using model ecosystem and single species exposure. Although there is variation in the actual log BCF values, in general, the algae and plants have the lowest BCF values, on the order of a few thousand. A value of 4.38 has been reported for the snail *Physa* sp. Crustacea and insect larva appear to have the next highest log BCF values, followed by several species of fish, with the highest log BCF value of 4.79 [10].

Exposure of juvenile rainbow trout to 2,3,7,8-TCDD and 2,3,7,8-TCDF in water for 28 days resulted in adverse effects on survival, growth, and behavior at extremely low concentrations. A no-observed-effects concentration (NOEC) for 2,3,7,8-TCDD could not be determined because the exposure to the lowest dose of 0.038 ng/L resulted in significant mortality [11]. A number of biological effects have been reported in fish following exposure to 2,3,7,8-TCDD including enzyme induction, immunological effects, wasting syndrome, dermatological effects, hepatic effects, hematological effects, developmental effects, and cardiovascular effects [10].

Summary of Biological Effects Tissue Concentrations for 1,2,3,4,7,8-HexaCDF

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									
Salmonids							0.0045	[14]	F
Wildlife									
Falco peregrinus, Peregrine falcon			3.2 ng/g (eggs) (n = 6)	11.4% eggshell thinning				[12]	F; Kola Peninsula, Russia
Mustela vison, Mink	Diet: 1 pg/g[4]		33 pg/g[4] (liver)	LOAEL; reduced kit body weights followed by reduced survival		No BMF reported		[13]	L; BMF = lipid-normalized concentration in the liver divided by the lipid-normalized dietary concentration
	2 pg/g[4]		73 pg/g[4] (liver)	Reduced kit body weights followed by reduced survival		Log BMF = 1.70			
	3 pg/g[4]		130 pg/g[4] (liver)	Significant decrease in number of live kits whelped per female		Log BMF = 1.81			

[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] Not clear from reference if concentration is based on wet or dry weight.

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Chemical Category: POLYCHLORINATED DIBENZOFURANS

Chemical Name (Common Synonyms): **CASRN:** 57117-41-6
1,2,3,7,8-PENTACHLORODIBENZOFURAN

Chemical Characteristics

Solubility in Water: No data [1]

Half-Life: No data [2]

Log K_{ow}: No data [3]

Log K_{oc}: —

Human Health

Oral RfD: No data [4]

Confidence: —

Critical Effect: —

Oral Slope Factor: No data [4]

Carcinogenic Classification: —

Wildlife

Partitioning Factors: Partitioning factors for 1,2,3,7,8-pentaCDF in wildlife were not found in the studies reviewed.

Food Chain Multipliers: Limited information was found reporting on specific biomagnification factors of PCDDs and PCDFs through terrestrial wildlife; no information was available for 1,2,3,7,8-pentaCDF, specifically. Due to the toxicity, high K_{ow} values, and highly persistent nature of the PCDDs and PCDFs, they possess a high potential to bioaccumulate and biomagnify through the food web. PCDDs and PCDFs have been identified in fish and wildlife throughout the global aquatic and marine environments [5]. Studies conducted in Lake Ontario indicated that biomagnification of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD) appears to be significant between fish and fish-eating birds but not between fish and their food. When calculated for older predaceous fish such as lake-trout-eating young smelt, the log biomagnification factor (BMF) can equal 0.48. The log BMF from alewife to herring gulls in Lake Ontario was 1.51 for 2,3,7,8-TCDD [6].

EPA has developed risk-based concentrations of 2,3,7,8-TCDD in different media that present low and high risk to fish, mammalian, and avian wildlife. These concentrations were developed based on toxic effects of 2,3,7,8-TCDD and its propensity to bioaccumulate in fish, mammals, and birds.

Environmental Concentrations Associated With 2,3,7,8-TCDD Risk to Aquatic Life and associated Wildlife [7]

Organism	Fish Concentration (pg/g)	Sediment Concentration (pg/g dry wt.)	Water Concentration (pg/L)	
			POC=0.2	POC=1.0
Low Risk				
Fish	50	60	0.6	3.1
Mammalian Wildlife	0.7	2.5	0.008	0.04
Avian Wildlife	6	21	0.07	0.35
High Risk to Sensitive Species				
Fish	80	100	1	5
Mammalian Wildlife	7	25	0.08	0.4
Avian Wildlife	60	210	0.7	3.5

Note: POC - Particulate organic carbon
Fish lipid of 8% and sediment organic carbon of 3% assumed where needed.
For risk to fish, BSAF of 0.3 used; for risk to wildlife, BSAF of 0.1 used.
Low risk concentrations are derived from no-effects thresholds for reproductive effects (mortality in embryos and young) in sensitive species.
High risk concentrations are derived from TCDD doses expected to cause 50 to 100% mortality in embryos and young of sensitive species.

Aquatic Organisms

Partitioning Factors: Partitioning factors for 1,2,3,7,8-pentaCDF in aquatic organisms were not found in the studies reviewed.

Food Chain Multipliers: No specific food chain multipliers were identified for 1,2,3,7,8-pentaCDF. Food chain multiplier information was only available for 2,3,7,8-TCDD. Biomagnification of 2,3,7,8-TCDD does not appear to be significant between fish and their prey. Limited data for the base of the Lake Ontario lake trout food chain indicated little or no biomagnification between zooplankton and forage fish. BMFs based on fish consuming invertebrate species are probably close to 1.0 because of the 2,3,7,8-TCDD biotransformation by forage fish. BMFs greater than 1.0 might exist between some zooplankton species and their prey due to the lack of 2,3,7,8-TCDD biotransformation in invertebrates [7].

Toxicity/Bioaccumulation Assessment Profile

The polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) each consist of 75 isomers that differ in the number and position of attached chlorine atoms. The PCDDs and PCDFs are polyhalogenated aromatic compounds and exhibit several properties common to this group of compounds. These compounds tend to be highly lipophilic and the degree of lipophilicity is increased with increasing ring chlorination [5]. In general, the PCDDs and PCDFs exhibit relative inertness to acids, bases, oxidation, reduction, and heat, increasing in environmental persistence and chemical stability with increasing chlorination [8,5]. Because of their lipophilic nature, the PCDDs and PCDFs have been detected in fish, wildlife, and human adipose tissue, milk, and serum [5].

Each isomer has its own unique chemical and toxicological properties. The most toxic of the PCDD and PCDF isomers is 2,3,7,8-TCDD, one of the 22 possible congeners of tetrachlorodibenzo-*p*-dioxin [8]. Toxicity equivalency factors (TEFs) have been developed by the U.S. EPA relating the toxicities of other PCDD and PCDF isomers to that of 2,3,7,8-TCDD [9]. The biochemical mechanisms leading to the toxic response resulting from exposure to PCDDs and PCDFs are not known in detail, but experimental data suggest that an important role in the development of systemic toxicity resulting from exposure to these chemicals is played by an intracellular protein, the Ah receptor. This receptor binds halogenated polycyclic aromatic molecules, including PCDDs and PCDFs. In several mouse strains, the expression of toxicity of 2,3,7,8-TCDD-related compounds, including cleft palate formation, liver damage, effects on body weight gain, thymic involution, and chloracnegenic response has been correlated with their binding affinity for the Ah receptor, and with their ability to induce several enzyme systems [9].

Toxicity Equivalency Factors (TEF) for PCDD and PCDF Isomers [9]

Isomer	TEF
Total TetraCDD	1
2,3,7,8-TCDD	1
Other TCDDs	0.01
Total PentaCDDs	0.5
2,3,7,8-PentaCDDs	0.5
Other PentaCDDs	0.005
Total HexaCDDs	0.04
2,3,7,8-HexaCDDs	0.04
Other HexaCDDs	0.0004
Total HeptaCDDs	0.001
2,3,7,8-HeptaCDDs	0.001
Other HeptaCDDs	0.00001
Total TetraCDFs	0.1
2,3,7,8-TetraCDF	0.1
Other TetraCDFs	0.001
Total PentaCDFs	0.1
2,3,7,8-PentaCDFs	0.1
Other PentaCDFs	0.001
Total HexaCDFs	0.01
2,3,7,8-HexaCDFs	0.01
Other HexaCDFs	0.0001
Total HeptaCDFs	0.001
2,3,7,8-HeptaCDFs	0.001
Other HeptaCDFs	0.00001

In natural systems, PCDDs and PCDFs are typically associated with sediments, biota, and the organic carbon fraction of ambient waters [7]. Congener-specific analyses have shown that the 2,3,7,8-substituted PCDDs and PCDFs were the major compounds present in most sample extracts [5]. Results from limited

epidemiology studies are consistent with laboratory-derived threshold levels to 2,3,7,8-TCDD impairment of reproduction in avian wildlife. Population declines in herring gulls (*Larus argentatus*) on Lake Ontario during the early 1970s coincided with egg concentrations of 2,3,7,8-TCDD and related chemicals expected to cause reproductive failure based on laboratory experiments (2,3,7,8-TCDD concentrations in excess of 1,000 pg/g). Improvements in herring gull reproduction through the mid-1980s were correlated with declining 2,3,7,8-TCDD concentrations in eggs and lake sediments [7]. Based on limited information on isomer-specific analysis from animals at different trophic levels, it appears that at higher trophic levels, i.e., fish-eating birds and fish, there is a selection of the planar congeners with the 2,3,7,8-substituted positions [10].

PCDDs and PCDFs are accumulated by aquatic organisms through exposure routes that are determined by the habitat and physiology of each species. With log K_{ow}>5, exposure through ingestion of contaminated food becomes an important route for uptake in comparison to respiration of water [7]. The relative contributions of water, sediment, and food to uptake of 2,3,7,8-TCDD by lake trout in Lake Ontario was examined by exposing yearling lake trout to Lake Ontario smelt and sediment from Lake Ontario along with water at a 2,3,7,8-TCDD concentration simulated to be at equilibrium with the sediments. Food ingestion was found to contribute approximately 75 percent of total 2,3,7,8-TCDD [7]. There have been a number of bioconcentration studies of 2,3,7,8-TCDD using model ecosystem and single species exposure. Although there is variation in the actual log BCF values, in general, the algae and plants have the lowest BCF values, on the order of a few thousand. A value of 4.38 has been reported for the snail *Physa* sp. Crustacea and insect larva appear to have the next highest BCF values, followed by several species of fish, with the highest log BCF value of 4.78 [10].

Exposure of juvenile rainbow trout to 2,3,7,8-TCDD and -TCDF in water for 28 days resulted in adverse effects on survival, growth, and behavior at extremely low concentrations. A no-observed-effects concentration (NOEC) for 2,3,7,8-TCDD could not be determined because the exposure to the lowest dose of 0.038 ng/l resulted in significant mortality [11]. A number of biological effects have been reported in fish following exposure to 2,3,7,8-TCDD including enzyme induction, immunological effects, wasting syndrome, dermatological effects, hepatic effects, hematological effects, developmental effects, and cardiovascular effects [10].

Summary of Biological Effects Tissue Concentrations for 1,2,3,7,8-PentaCDF

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									
Salmonids							0.013	[15]	F
Wildlife									
Falco peregrinus, Peregrine falcon			4.0 ng/g (eggs) (n = 6)	11.4% eggshell thinning				[13]	F; Kola Peninsula, Russia
Haliaeetus leucocephalus, Bald eagle chicks			Powell River site: ~160 ng/kg lipid weight basis (yolk sac) Reference site: ~30 ng/kg lipid weight basis (yolk sac)	A hepatic cytochrome P4501A crossreactive protein (CYP1A) was induced nearly six-fold in chicks from Powell River site compared to the reference ($p<0.05$). No significant concentration-related effects were found for morphological, physiological, or histological parameters.				[12]	F; southern coast of British Columbia; eggs were collected from nests and hatched in the lab; ~ indicates value was taken from a figure.

Summary of Biological Effects Tissue Concentrations for 1,2,3,7,8-PentaCDF

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]**
Mustela vison, Mink	Diet: 1 pg/g[4]		1 pg/g[4] (liver)	LOAEL; reduced kit body weights followed by reduced survival				[14]	L
	2 pg/g[4]		2 pg/g[4] (liver)	Reduced kit body weights followed by reduced survival					
	4 pg/g[4]		2 pg/g[4] (liver)	Significant decrease in number of live kits whelped per female					

[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] Not clear from reference if concentration is based on wet or dry weight.

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15. USEPA. 1995. *Great Lakes Water Quality Initiative technical support document for the procedure to determine bioaccumulation factors*. EPA-820-B-95-005. U.S. Environmental Protection Agency, Office of Water, Washington, DC.

Chemical Category: POLYCHLORINATED DIBENZOFURANS

Chemical Name (Common Synonyms): **CASRN:** 57117-31-4
2,3,4,7,8-PENTACHLORODIBENZOFURAN

Chemical Characteristics

Solubility in Water: 0.24 µg/L [1]

Half-Life: No data [2,3]

Log K_{ow}: No data [4], 6.92 [2]

Log K_{oc}: 6.80 L/kg organic carbon

Human Health

Oral RfD: No data [5]

Confidence: —

Critical Effect: —

Oral Slope Factor: No data [5]

Carcinogenic Classification: —

Wildlife

Partitioning Factors: Partitioning factors for 2,3,4,7,8-pentaCDF in wildlife were not found in the studies reviewed.

Food Chain Multipliers: Limited information was found reporting on specific biomagnification factors of PCDDs and PCDFs through terrestrial wildlife; no information was available for 2,3,4,7,8-pentaCDF, specifically. Due to the toxicity, high K_{ow} values, and highly persistent nature of the PCDDs and PCDFs, they possess a high potential to bioaccumulate and biomagnify through the food web. PCDDs and PCDFs have been identified in fish and wildlife throughout the global aquatic and marine environments [6]. Studies conducted in Lake Ontario indicated that biomagnification of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD) appears to be significant between fish and fish-eating birds but not between fish and their food. When calculated for older predaceous fish such as lake-trout-eating young smelt, the biomagnification factor (BMF) can equal 3. The BMF from alewife to herring gulls in Lake Ontario was 32 for 2,3,7,8-TCDD [7]. Log BMFs of 1.73 to 1.74 were determined for mink [18].

EPA has developed risk-based concentrations of 2,3,7,8-TCDD in different media that present low and high risk to fish, mammalian, and avian wildlife. These concentrations were developed based on toxic effects of 2,3,7,8-TCDD and its propensity to bioaccumulate in fish, mammals, and birds.

Environmental Concentrations Associated With 2,3,7,8-TCDD Risk to Aquatic Life and Associated Wildlife [8]

Organism	Fish Concentration (pg/g)	Sediment Concentration	Water Concentration (pg/L)	
			POC=0.2	POC=1.0
Low Risk				
Fish	50	60	0.6	3.1
Mammalian Wildlife	0.7	2.5	0.008	0.04
Avian Wildlife	6	21	0.07	0.35
High Risk to Sensitive Species				
Fish	80	100	1	5
Mammalian Wildlife	7	25	0.08	0.4
Avian Wildlife	60	210	0.7	3.5

Note: POC - Particulate organic carbon
Fish lipid of 8% and sediment organic carbon of 3% assumed where needed.
For risk to fish, BSAF of 0.3 used; for risk to wildlife, BSAF of 0.1 used.
Low risk concentrations are derived from no-effects thresholds for reproductive effects (mortality in embryos and young) in sensitive species.
High risk concentrations are derived from TCDD doses expected to cause 50 to 100% mortality in embryos and young of sensitive species.

Aquatic Organisms

Partitioning Factors: In one study, the BSAF for carp collected from a reservoir in central Wisconsin was 0.28. The log BCF for goldfish measured during a laboratory exposure of 120 hours was 4.48

Food Chain Multipliers: No specific food chain multipliers were identified for 2,3,4,7,8-pentaCDF. Food chain multiplier information was only available for 2,3,7,8-TCDD. Biomagnification of 2,3,7,8-TCDD does not appear to be significant between fish and their prey. Limited data for the base of the Lake Ontario lake trout food chain indicated little or no biomagnification between zooplankton and forage fish. BMFs based on fish consuming invertebrate species are probably close to 1.0 because of the 2,3,7,8-TCDD biotransformation by forage fish. BMFs greater than 1.0 might exist between some zooplankton species and their prey due to the lack of 2,3,7,8-TCDD biotransformation in invertebrates [8].

Toxicity/Bioaccumulation Assessment Profile

The polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) each consist of 75 isomers that differ in the number and position of attached chlorine atoms. The PCDDs and PCDFs are polyhalogenated aromatic compounds and exhibit several properties common to this group of compounds. These compounds tend to be highly lipophilic and the degree of lipophilicity is increased with increasing ring chlorination [6]. In general, the PCDDs and PCDFs exhibit relative inertness to acids, bases, oxidation, reduction, and heat, increasing in environmental persistence and chemical stability with increasing chlorination [6,9]. Because of their lipophilic nature, the PCDDs and PCDFs have been detected in fish, wildlife, and human adipose tissue, milk, and serum [6].

Each isomer has its own unique chemical and toxicological properties. The most toxic of the PCDD and PCDF isomers is 2,3,7,8-TCDD, one of the 22 possible congeners of tetrachlorodibenzo-*p*-dioxin [8]. Toxicity equivalency factors (TEFs) have been developed by the EPA relating the toxicities of other PCDD and PCDF isomers to that of 2,3,7,8-TCDD [10]. The biochemical mechanisms leading to the toxic response resulting from exposure to PCDDs and PCDFs are not known in detail, but experimental data suggest that an important role in the development of systemic toxicity resulting from exposure to these chemicals is played by an intracellular protein, the Ah receptor. This receptor binds halogenated polycyclic aromatic molecules, including PCDDs and PCDFs. In several mouse strains, the expression of toxicity of 2,3,7,8-TCDD-related compounds, including cleft palate formation, liver damage, effects on body weight gain, thymic involution, and chloracnegenic response, has been correlated with their binding affinity for the Ah receptor, and with their ability to induce several enzyme systems [10].

Toxicity Equivalency Factors (TEF) for PCDD and PCDF Isomers [10]

Isomer	TEF
Total TetraCDD	1
2,3,7,8-TCDD	1
Other TCDDs	0.01
Total PentaCDDs	0.5
2,3,7,8-PentaCDDs	0.5
Other PentaCDDs	0.005
Total HexaCDDs	0.04
2,3,7,8-HexaCDDs	0.04
Other HexaCDDs	0.0004
Total HeptaCDDs	0.001
2,3,7,8-HeptaCDDs	0.001
Other HeptaCDDs	0.00001
Total TetraCDFs	0.1
2,3,7,8-TetraCDF	0.1
Other TetraCDFs	0.001
Total PentaCDFs	0.1
2,3,7,8-PentaCDFs	0.1
Other PentaCDFs	0.001
Total HexaCDFs	0.01
2,3,7,8-HexaCDFs	0.01
Other HexaCDFs	0.0001
Total HeptaCDFs	0.001
2,3,7,8-HeptaCDFs	0.001
Other HeptaCDFs	0.00001

In natural systems, PCDDs and PCDFs are typically associated with sediments, biota, and the organic carbon fraction of ambient waters [7]. Congener-specific analyses have shown that the 2,3,7,8-substituted PCDDs and PCDFs were the major compounds present in most sample extracts [6]. Results from limited

epidemiology studies are consistent with laboratory-derived threshold levels to 2,3,7,8-TCDD impairment of reproduction in avian wildlife. Population declines in herring gulls (*Larus argentatus*) on Lake Ontario during the early 1970s coincided with egg concentrations of 2,3,7,8-TCDD and related chemicals expected to cause reproductive failure based on laboratory experiments (2,3,7,8-TCDD concentrations in excess of 1,000 pg/g). Improvements in herring gull reproduction through the mid-1980s were correlated with declining 2,3,7,8-TCDD concentrations in eggs and lake sediments [8]. Based on limited information on isomer-specific analysis from animals at different trophic levels, it appears that at higher trophic levels, i.e., fish-eating birds and fish, there is a selection of the planar congeners with the 2,3,7,8-substituted positions [11].

PCDDs and PCDFs are accumulated by aquatic organisms through exposure routes that are determined by the habitat and physiology of each species. With log K_{ow}>5, exposure through ingestion of contaminated food becomes an important route for uptake in comparison to respiration of water [8]. The relative contributions of water, sediment, and food to uptake of 2,3,7,8-TCDD by lake trout in Lake Ontario was examined by exposing yearling lake trout to Lake Ontario smelt and sediment from Lake Ontario along with water at a 2,3,7,8-TCDD concentration simulated to be at equilibrium with the sediments. Food ingestion was found to contribute approximately 75 percent of total 2,3,7,8-TCDD [8]. There have been a number of bioconcentration studies of 2,3,7,8-TCDD using model ecosystem and single species exposure. Although there is variation in the actual log BCF values, in general, the algae and plants have the lowest BCF values, on the order of a few thousand. A value of 4.38 has been reported for the snail *Physa* sp. Crustacea and insect larva appear to have the next highest BCF values, followed by several species of fish, with the highest log BCF value of 4.79 [11].

Exposure of juvenile rainbow trout to 2,3,7,8-TCDD and -TCDF in water for 28 days resulted in adverse effects on survival, growth, and behavior at extremely low concentrations. A no-observed-effects concentration (NOEC) for 2,3,7,8-TCDD could not be determined because the exposure to the lowest dose of 0.038 ng/L resulted in significant mortality [12]. A number of biological effects have been reported in fish following exposure to 2,3,7,8-TCDD including enzyme induction, immunological effects, wasting syndrome, dermatological effects, hepatic effects, hematological effects, developmental effects, and cardiovascular effects [11].

Summary of Biological Effects Tissue Concentrations for 2,3,4,7,8-PentaCDF

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
					Log BCF	Log BAF			
Taxa	**Sediment**	**Water**	**Tissue (Sample Type)**	**Effects**			**BSAF**	**Reference**	**Comments[3]**
Fishes									
Carassius auratus, Goldfish			2.69/2.5 ng/g[4] (whole body)		4.48			[14]	L; fish were exposed for 120 hr; exposure water contained fly ash extract; concen-trations were measured in water, but data were not presented
Cyprinus carpio, Carp	8 pg/g[4]		4.4 pg/g[4]				0.28	[13]	F; Petenwell Reservoir, central Wisconsin; BSAF based on 8% tissue lipid content and 3.1% sediment organic carbon
Salmonids							0.095	[19]	F
Wildlife									
Falco peregrinus, Peregrine falcon			27 ng/g (eggs) (n = 6)	11.4% eggshell thinning				[17]	F; Kola Peninsula, Russia

Summary of Biological Effects Tissue Concentrations for 2,3,4,7,8-PentaCDF

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
					Log BCF	Log BAF			
Taxa	**Sediment**	**Water**	**Tissue (Sample Type)**	**Effects**			**BSAF**	**Reference**	**Comments[3]**
Haliaeetus leucocephalus, Bald eagle chicks			Powell River site: ~800 ng/kg lipid weight basis (yolk sac) Reference site: ~100 ng/kg lipid weight basis (yolk sac)	A nearly 6-fold increase in hepatic cytochrome P4501A cross-reactive protein (CYP1A) was induced in chicks from Powell River site compared to the reference ($p<0.05$). No significant concentration-related effects were found for morphological, physiological, or histological parameters.				[15]	F; southern coast of British Columbia; eggs were collected from nests and hatched in the lab; ~ indicates value was taken from a figure.

Summary of Biological Effects Tissue Concentrations for 2,3,4,7,8-PentaCDF

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
					Log BCF	Log BAF			
Taxa	**Sediment**	**Water**	**Tissue (Sample Type)**	**Effects**			**BSAF**	**Reference**	**Comments[3]**
Ardea herodias, Great blue heron chicks			Nicomekl site: <2 ng/kg (egg) (n = 11)					[16]	L; eggs were collected from three British Columbia colonies with different levels of contamination and incubated in the laboratory
			Vancouver site: 33±18.5 ng/kg (egg) (n = 12)	Depression of growth compared to Nicomekl site. Presence of edema.					
			Crofton site: 33±7.6 ng/kg (egg) (n = 6)	Depression of growth compared to Nicomekl site. Presence of edema.					

Summary of Biological Effects Tissue Concentrations for 2,3,4,7,8-PentaCDF

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
					Log BCF	Log BAF	BSAF		
Taxa	**Sediment**	**Water**	**Tissue (Sample Type)**	**Effects**				**Reference**	**Comments[3]**
Mustela vison, Mink	Diet: 4 pg/g[4]		170 pg/g[4] (liver)	LOAEL; reduced kit body weights followed by reduced survival		No BMF reported		[18]	L; BMF = lipid-normalized concentration in the liver divided by the lipid-normalized dietary concentration
	6 pg/g[4]		320 pg/g[4] (liver)	Reduced kit body weights followed by reduced survival		Log BMF = 1.74			
	14 pg/g[4]		490 pg/g[4] (liver)	Significant decrease in number of live kits whelped per female		Log BMF = 1.73			

[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] Not clear from reference if concentration is based on wet or dry weight.

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Chemical Category: POLYCHLORINATED DIBENZOFURANS

Chemical Name (Common Synonyms): **CASRN:** 51207-31-9
2,3,7,8-TETRACHLORODIBENZOFURAN

Chemical Characteristics

Solubility in Water: No data [1], 0.42 µg/L [2] **Half-Life:** No data [3]

Log K_{ow}: No data [4], 6.53 [2] **Log K_{oc}:** —

Human Health

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

Critical Effect: —

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

Wildlife

Partitioning Factors: Partitioning factors for 2,3,7,8-TCDF in wildlife were not found in the studies reviewed.

Food Chain Multipliers: Limited information was found reporting on specific biomagnification factors of PCDDs and PCDFs through terrestrial wildlife; no information was available for 2,3,7,8-TCDF, specifically. Due to the toxicity, high K_{ow} values, and highly persistent nature of the PCDDs and PCDFs, they possess a high potential to bioaccumulate and biomagnify through the food web. PCDDs and PCDFs have been identified in fish and wildlife throughout the global aquatic and marine environments [6]. Studies conducted in Lake Ontario indicated that biomagnification of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD) appears to be significant between fish and fish-eating birds but not between fish and their food. When calculated for older predaceous fish such as lake-trout-eating young smelt, the biomagnification factor (BMF) can equal 3. The BMF from alewife to herring gulls in Lake Ontario was 32 for 2,3,7,8-TCDD [7]. A log BMF of -0.40 was determined for mink [2].

EPA has developed risk-based concentrations of 2,3,7,8-TCDD in different media that present low and high risk to fish, mammalian, and avian wildlife. These concentrations were developed based on toxic effects of 2,3,7,8-TCDD and its propensity to bioaccumulate in fish, mammals, and birds.

Environmental Concentrations Associated With 2,3,7,8-TCDD Risk to Aquatic Life and Associated Wildlife [8]

Organism	Fish Concentration (pg/g)	Sediment Concentration (pg/g dry wt.)	Water Concentration (pg/L)	
			POC=0.2	POC=1.0
Low Risk				
Fish	50	60	0.6	3.1
Mammalian Wildlife	0.7	2.5	0.008	0.04
Avian Wildlife	6	21	0.07	0.35
High Risk to Sensitive Species				
Fish	80	100	1	5
Mammalian Wildlife	7	25	0.08	0.4
Avian Wildlife	60	210	0.7	3.5

Note: POC - Particulate organic carbon
Fish lipid of 8% and sediment organic carbon of 3% assumed where needed.
For risk to fish, BSAF of 0.3 used; for risk to wildlife, BSAF of 0.1 used.
Low risk concentrations are derived from no-effects thresholds for reproductive effects (mortality in embryos and young) in sensitive species.
High risk concentrations are derived from TCDD doses expected to cause 50 to 100% mortality in embryos and young of sensitive species.

Aquatic Organisms

Partitioning Factors: In one study, steady-state BSAFs for invertebrates exposed to 2,3,7,8-TCDF in the laboratory ranged from about 0.3 to 0.7. The BSAF for carp collected from a reservoir in central Wisconsin was 0.06.

Food Chain Multipliers: No specific food chain multipliers were identified for 2,3,7,8-TCDF. Food chain multiplier information was only available for 2,3,7,8-TCDD. Biomagnification of 2,3,7,8-TCDD does not appear to be significant between fish and their prey. Limited data for the base of the Lake Ontario lake trout food chain indicated little or no biomagnification between zooplankton and forage fish. BMFs based on fish consuming invertebrate species are probably close to 1.0 because of the 2,3,7,8-TCDD biotansformation by forage fish. BMFs greater than 1.0 might exist between some zooplankton species and their prey due to the lack of 2,3,7,8-TCDD biotransformation in invertebrates [8].

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The polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) each consist of 75 isomers that differ in the number and position of attached chlorine atoms. The PCDDs and PCDFs are polyhalogenated aromatic compounds and exhibit several properties common to this group of compounds. These compounds tend to be highly lipophilic and the degree of lipophilicity is increased with increasing ring chlorination [6]. In general, the PCDDs and PCDFs exhibit relative inertness to acids, bases, oxidation, reduction, and heat, increasing in environmental persistence and chemical stability

with increasing chlorination [9,6]. Because of their lipophilic nature, the PCDDs and PCDFs have been detected in fish, wildlife, and human adipose tissue, milk, and serum [6].

Each isomer has its own unique chemical and toxicological properties. The most toxic of the PCDD and PCDF isomers is one of the 22 possible congeners of tetrachlorodibenzo-*p*-dioxin [9]. Toxicity equivalency factors (TEFs) have been developed by EPA relating the toxicities of other PCDD and PCDF isomers to that of 2,3,7,8-TCDD [10]. The biochemical mechanisms leading to the toxic response resulting from exposure to PCDDs and PCDFs are not known in detail, but experimental data suggest that an important role in the development of systemic toxicity resulting from exposure to these chemicals is played by an intracellular protein, the Ah receptor. This receptor binds halogenated polycyclic aromatic molecules, including PCDDs and PCDFs. In several mouse strains, the expression of toxicity of 2,3,7,8-TCDD-related compounds, including cleft palate formation, liver damage, effects on body weight gain, thymic involution, and chloracnegenic response, has been correlated with their binding affinity for the Ah receptor, and with their ability to induce several enzyme systems [10].

Toxicity Equivalency Factors (TEF) for PCDD and PCDF Isomers [10]

Isomer	TEF
Total TetraCDD	1
2,3,7,8-TCDD	1
Other TCDDs	0.01
Total PentaCDDs	0.5
2,3,7,8-PentaCDDs	0.5
Other PentaCDDs	0.005
Total HexaCDDs	0.04
2,3,7,8-HexaCDDs	0.04
Other HexaCDDs	0.0004
Total HeptaCDDs	0.001
2,3,7,8-HeptaCDDs	0.001
Other HeptaCDDs	0.00001
Total TetraCDFs	0.1
2,3,7,8-TetraCDF	0.1
Other TetraCDFs	0.001
Total PentaCDFs	0.1
2,3,7,8-PentaCDFs	0.1
Other PentaCDFs	0.001
Total HexaCDFs	0.01
2,3,7,8-HexaCDFs	0.01
Other HexaCDFs	0.0001
Total HeptaCDFs	0.001
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Other HeptaCDFs	0.00001

In natural systems, PCDDs and PCDFs are typically associated with sediments, biota, and the organic carbon fraction of ambient waters [7]. Congener-specific analyses have shown that the 2,3,7,8-substituted PCDDs and PCDFs were the major compounds present in most sample extracts [6]. Results from limited epidemiology studies are consistent with laboratory-derived threshold levels to 2,3,7,8-TCDD impairment of reproduction in avian wildlife. Population declines in herring gulls (*Larus argentatus*) on Lake Ontario during the early 1970s coincided with egg concentrations of 2,3,7,8-TCDD and related chemicals expected to cause reproductive failure based on laboratory experiments (2,3,7,8-TCDD concentrations in excess of 1,000 pg/g). Improvements in herring gull reproduction through the mid-1980s were correlated with declining 2,3,7,8-TCDD concentrations in eggs and lake sediments [8]. Based on limited information on isomer-specific analysis from animals at different trophic levels, it appears that at higher trophic levels, i.e., fish-eating birds and fish, there is a selection of the planar congeners with the 2,3,7,8-substituted positions [11].

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Exposure of juvenile rainbow trout to 2,3,7,8-TCDD and -TCDF in water for 28 days resulted in adverse effects on survival, growth, and behavior at extremely low concentrations. A no-observed-effects concentration (NOEC) for 2,3,7,8-TCDD could not be determined because the exposure to the lowest dose of 0.038 ng/L resulted in significant mortality [12]. A number of biological effects have been reported in fish following exposure to 2,3,7,8-TCDD including enzyme induction, immunological effects, wasting syndrome, dermatological effects, hepatic effects, hematological effects, developmental effects, and cardiovascular effects [11].

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDF

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 virens, Sandworm	334±6 pg/g dw n = 6		112±51 pg/g dw (whole body)				~0.25	[13,14]	L; 180-day exposure; sediment TOC was 57 mg/kg; ~ indicates approximate value, as numbers were estimated from bar graphs
Macoma nasuta, Clam	334±6 pg/g dw n = 6		51.4±6.8 pg/g dw				~0.7	[13,14]	L; 120-day exposure; sediment TOC was 57 mg/kg; ~ indicates approximate value, as numbers were estimated from bar graphs
Palaemonetes pugio, Grass shrimp	334±6 pg/g dw n = 6		58.8±7.7 pg/g dw				~0.6	[13,14]	L; 28-day exposure; sediment TOC was 57 mg/kg; ~ indicates approximate value, as numbers were estimated from bar graphs

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDF

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									
Oncorhynchus mykiss (*Salmo gairdneri*), Rainbow trout		Water exposure 0.41 ng/L	2.5 µg/kg[4]	28-Day NOEC (growth)				[15]	L
		Water exposure 1.79 ng/L	7.6 µg/kg[4]	28-Day NOEC (survival)				[15]	
Oncorhynchus mykiss, Rainbow trout			0.00009 mg/kg (whole body)[4]	Growth, NOED				[15]	L
Salmonids							0.047	[22]	F
Cyprinus carpio, Carp	182 pg/g[4]		28 pg/g[4]				0.06	[16]	F; Petenwell Reservoir, central Wisconsin; BSAF based on 8% tissue lipid content and 3.1% sediment organic carbon

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDF

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]**
Wildlife									
Falco peregrinus, Peregrine falcon			30 ng/g (eggs) (n=6)	11.4% eggshell thinning				[19]	F; Kola Peninsula, Russia
Haliaeetus leucocephalus, Bald eagle chicks			Powell River site: 8,000 ng/kg lipid weight basis (yolk sac) Reference site: 500 ng/kg lipid weight basis (yolk sac)	A hepatic cytochrome P4501A cross-reactive protein (CYP1A) was induced nearly 6-fold in chicks from Powell River site compared to the reference ($p<0.05$). No significant concentration-related effects were found for morphological, physiological, or histological parameters.				[17]	F; southern coast of British Columbia; eggs were collected from nests and hatched in the laboratory.

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDF

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]**
Ardea herodias, Great blue heron chicks			Nicomekl site: <1 ng/kg (egg) n = 11					[18]	L; eggs were collected from three British Columbia colonies with different levels of contamination and incubated in the laboratory
			Vancouver site: 11±4.3 ng/kg (egg) n = 12	Depression of growth compared to Nicomekl site. Presence of edema.					
			Crofton site: 8±2.3 ng/kg (egg) n = 6	Depression of growth compared to Nicomekl site. Presence of edema.					

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDF

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]**
Aix sponsa, Wood duck			pg/g (eggs):	% eggs hatched:				[20]	F; central Arkansas; egg TEFs, hatching success, and duckling prod-uction were negatively corre-lated; clutch size was similar among wetland Sites 1-3, 9, 17, and 58 km downstream from point source of contamination, respectively, and Site 4, which was 111 km away on a separate drainage; duckling abnormalities were also noted; threshold range of reduced productivity was >20-50 ppt TEF
			Site 1 geometric mean: 26 (2.4-244)	47 (9.7 SE)					
			Site 2 geometric mean: 11 (1.4-60)	62 (10.1 SE)					
			Site 3 geometric mean: 5.4 (<1-22)	79 (3.8 SE)					
			Site 4 geometric mean: 0.3 (<1-3.2)	93 (3.4 SE)					

Summary of Biological Effects Tissue Concentrations for 2,3,7,8-TCDF

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]**
Mustela vison, Mink	Diet: 2 pg/g[5]		2 pg/g[5] (liver)	LOAEL; reduced kit body weights followed by reduced survival		No BMF reported		[21]	L; BMF= lipid-normalized concentration in the liver divided by the lipid-normalized dietary concentration
	4 pg/g[5]		2 pg/g[5] (liver)	Reduced kit body weights followed by reduced survival		Log BMF= -0.4			
	12 pg/g[5]		3 pg/g[5] (liver)	Significant decrease in number of live kits whelped per female		Log BMF= -0.4			

[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.
[5] Not clear from reference if concentration is based on wet or dry weight.

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Chemical Category: POLYNUCLEAR AROMATIC HYDROCARBON (high molecular weight)

Chemical Name (Common Synonyms): FLUORANTHENE **CASRN:** 206-44-0

Chemical Characteristics

Solubility in Water: 0.20-0.26 mg/L [1]

Half-Life: 140-440 days, aerobic soil die-away test [2]

Log K_{ow}: 5.12 [3]

Log K_{oc}: 5.03 L/kg organic carbon

Human Health

Oral RfD: 4 x 10^{-2} mg/kg-day [4]

Confidence: Low, uncertainty factor = 3000

Critical Effect: Nephropathy, increased liver weights, hematological alterations, and clinical effects

Oral Slope Factor: No data [4]

Carcinogenic Classification: D [4]

Wildlife

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

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

Aquatic Organisms

Partitioning Factors: The water quality criterion tissue level (WQCTL) for fluoranthene, which is calculated by multiplying the water quality chronic value (16 µg/L) by the BCF (1741.8), is 27,869 µg/kg [5]. Salinity and particle size of the sediment had no or very little effect on survival of three amphipod species during exposure to fluoranthene [6]. Log BCFs ranged from -0.92 for *Lumbriculues variegatus* [16] to 0.63 for *Hyallela azteca* [9]. Log BAFs of 0.36 to 0.56 were calculated for the midges *Chironomus tentans* [24].

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

Toxicity/Bioaccumulation Assessment Profile

Polynuclear aromatic hydrocarbons, (PAHs) are readily metabolized and excreted by fish and invertebrates [7], affecting bioaccumulation kinetics and equilibrium tissue residues. According to McCarty et al. [8], the toxic body residue of individual PAHs in tissues ranged from 513 to 4,248 mg/kg.

The concentration of 382 ppb produced biological effects in environmental samples (Puget Sound). The LC50 values for fluoranthene using freshwater amphipods ranged from 11.7 to 150.3 nmol/g dry weight [9].

Fluoranthene is relatively toxic to aquatic species (10-day EC50 = 2.3 to 7.4 µg/L for *H. azteca*, 10-day EC50 = 3.0 to 8.7 µg/L *C. tentans*). Its toxicity increased 6- to 17-fold under UV light [10]. *H. azteca* accumulated up to 1,131 µg/g of fluoranthene during 10 days of exposure to the LC50 concentration. Below the toxic level, the concentration of fluoranthene in amphipod tissue reached 200 to 400 µg/g within the first 48 hours and then dropped to 100 µg/g [9]. During 30-day bioaccumulation exposures, fed *H. azteca* accumulated significantly more fluoranthene than unfed organisms [11]. Furthermore, in exposures in which food was added, organisms gained weight and reproduced, even when sediment was dosed with concentrations approximately 20 to 90 times the 10-day LC50 value, with sediment containing levels of organic carbon comparable to the Suedel et al. [12] experiments. These data suggest that animals in fed exposures preferentially consumed the food, given the relatively high accumulation of compound in animal tissue. Mortality due to narcosis, the mechanism thought to be responsible for PAH toxicity, ranged from 2 to 8 µmol/g for acute responses and 0.2 to 0.8 µmol/g for chronic exposures in fish [13]. In the study by Harkey et al. [11], animals accumulated up to 1.4 µmol/g after 30 days in the highest (1,004 nmol/g) sediment concentration. Previous water-only exposures [14] predicted that a body burden of 5.6 µmol/g in *H. azteca* needs to be attained to produce 50 percent mortality. The body burden of fluoranthene associated with 50 percent mortality of *Leptocheirus plumulosus* was 0.69 µmol/g wet wt, which is lower than the predicted critical body residue for nonpolar narcotic compounds [15].

Summary of Biological Effects Tissue Concentrations for Fluoranthene

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									
Lumbriculus variegatus, Oligochaete worm					-0.92			[16]	F
Nereis succinea, Polychaete worm	0.218 µg/g OC 0.436 µg/g OC 0.48 µg/g OC 1.4 µg/g OC 4.55 µg/g OC 10.2 µg/g OC 19.5 µg/g OC 30.1 µg/g OC		9.20 µg/g lipid – 2.55 µg/g lipid 35.6 µg/g lipid 4.80 µg/g lipid 3.79 µg/g lipid 14.1 µg/g lipid 24.0 µg/g lipid					[17]	F
Nereis virens, Sand worm					-0.096 or -0.10 -0.02 0.52			[18]	F
Modiolus demissus, Northern horse mussel					0.36			[19]	F
Mytilus edulis, Blue mussel					-0.44			[19]	F

Summary of Biological Effects Tissue Concentrations for Fluoranthene

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]**
Mytilus edulis, Mussel			627 mg/kg (whole body)[4]	Physiological, ED50				[27]	L; 50% reduction in feeding rate
			1.9 mg/kg (whole body)[4]	Physiological, ED50				[28]	L; 50% reduction in feeding, clearance rate and tolerance to aerial exposure
			0.112 mg/kg (whole body)	Physiological, LOED				[28]	L; elevated activity of superoxide dimutase (SOD)
			1.5 mg/kg (whole body)[4]	Physiological, LOED				[28]	L; inhibition of superoxide dimutase (SOD) and catalase activity
			1.5 mg/kg (whole body)[4]	Reproduction, LOED				[28]	L; reduced gametogenesis, reproductive success rate
Crassostrea virginica, Eastern oyster			62 mg/kg (whole body)[4]	Morphology, LOED				[30]	L; thickness of digestive epithelium
Crassostrea virginica, Eastern oyster					-0.15 -0.28			[19]	F

Summary of Biological Effects Tissue Concentrations for Fluoranthene

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]**
Macoma balthica, Baltic macoma	0.218 µg/g OC 0.436 µg/g OC 0.48 µg/g OC 1.4 µg/g OC 4.55 µg/g OC 10.2 µg/g OC 19.5 µg/g OC 30.1 µg/g OC		7.62 µg/g lipid 5.12 µg/g lipid – 96.2 µg/g lipid 7.48 µg/g lipid 5.73 µg/g lipid 17.2 µg/g lipid –					[17]	F
Macoma nasuta, Clam					0.58 0.39 -0.26			[18]	F
Mercenaria mercenaria, Northern quahog					-0.05			[19]	F
Mya arenaria, Softshell					-0.08			[19]	F
Daphnia magna, Cladoceran		9 µg/L	77 nM/g		0.51			[20]	L

Summary of Biological Effects Tissue Concentrations for Fluoranthene

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]**
Hyalella azteca, Amphipod		14.2 µg/L	25.6 µg/g		0.51			[9]	L
			44.0 µg/g		0.54				
			44.8 µg/g		0.54				
			65.7 µg/g		0.56				
			78.4 µg/g		0.57				
Hyalella azteca, Amphipod		56.7 µg/L	169 µg/g		0.59			[9]	L
			320 µg/g		0.55				
			458 µg/g		0.57				
			751 µg/g		0.54				
		86.2 µg/L	350 µg/g		0.60			[9]	L
			531 µg/g		0.59				
			714 µg/g		0.58				
			800 µg/g		0.62				
			1,192 µg/g		0.61				
		100.8 µg/L	644 µg/g		0.61				
			898 µg/g		0.59				
			1,074 µg/g		0.60				
			1,199 µg/g		0.56				
			1,248 µg/g		0.58				
		41.5 µg/L	307 µg/g		0.58				
			363 µg/g		0.59				
			515 µg/g		0.60				
			517 µg/g		0.63				
			763 µg/g		0.63				
			815 µg/g		0.60				
			852 µg/g		0.61				
		98.3 µg/L	566 µg/g		0.61				
			825 µg/g		0.61				
			829 µg/g		0.61				
			1,035 µg/g		0.63				
			1,171 µg/g		0.60				
			1,213 µg/g		0.61				
			1,310 µg/g		0.58				

Summary of Biological Effects Tissue Concentrations for Fluoranthene

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]
Hyalella azteca, Amphipod		168.0 µg/L	855 µg/g		0.61				
			884 µg/g		0.59				
			971 µg/g		0.60				
			988 µg/g		0.58				
			1,265 µg/g		0.59				
			1,375 µg/g		0.60				
		184.7 µg/L	746 µg/g		0.57				
			896 µg/g		0.58				
			1,208 µg/g		0.57				
			1,302 µg/g		0.59				
			1,382 µg/g		0.59				
			1,445 µg/g		0.58				
			1,581 µg/g		0.57				
	158 nmol/g		Day 1: 160 nmol/g	no mortality				[11]	L
			Day 2: 140 nmol/g	no mortality					
			Day 3: 60 nmol/g	no mortality					
			Day 10: 90 nmol/g	no mortality					
			Day 17: 110 nmol/g	no mortality					
			Day 30: 120 nmol/g	no mortality					
	634 nmol/g		Day 1: 900 nmol/g	no mortality					
			Day 2: 1,050 nmol/g	no mortality					
			Day 3: 850 nmol/g	no mortality					
			Day 10: 700 nmol/g	no mortality					
			Day 17: 700 nmol/g	40% mortality					
			Day 30:800 nmol/g	40% mortality					
Hyalella azteca, Amphipod	1267 nmol/g		Day 1: 1,000 nmol/g	no mortality					
			Day 2: 850 nmol/g	no mortality					
			Day 3: 950 nmol/g	no mortality					
			Day 10: 700 nmol/g	no mortality					
			Day 17: 800 nmol/g	35% mortality					
			Day 30: 1,100 nmol/g	65% mortality					

Summary of Biological Effects Tissue Concentrations for Fluoranthene

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]**
Leptocheirus plumulosus, Amphipod		38 µg/L or 187 nmol/L	0.68 µmol/g	50% mortality				[15]	L; critical body residue
		36 nmol/L							
		77 nmol/L	78 nmol/g	100% survival					
		143 nmol/L	226 nmol/g	100% survival					
		285 nmol/L	369 nmol/g	93% survival					
			721 nmol/g	46% survival					
Pontoporeia hoyi, Amphipod	60 ng/g	5 ng/mL	2,000 ng/g					[21]	L
	270 ng/g	4 ng/mL	2,000 ng/g						
	1000 ng/g	4 ng/mL	1,000 ng/g						
	21.3 nmol/g		7-12 nmol/g			1.04-1.36		[14]	L
	41.1 nmol/g		28-57 nmol/g						
	119.5 nmol/g		68-149 nmol/g						
	327.0 nmol/g		71-614 nmol/g						
Rhepoxynius abronius, Amphipod	12.09 mg/kg	14.3 µg/L		23% mortality				[22]	L
	14.50 mg/kg			52% mortality					
	25.11 mg/kg			92% mortality					
Chironomus riparius, Midge	4,040 µg/kg		181,000 µg/kg					[23]	L
Chironomus tentans Midge	377 µg/g_{oc}	4 µg/L	9,593 ng/g (larvae)			0.36		[24]	L
			22 ng/g (adult)			0.36			
	1,220 µg/g_{oc}	12 µg/L	33,455 ng/g (larvae)			0.41			
			257 ng/g (adult)			0.41			
	1,853 ug/g_{oc}	19 µg/L	72,790 ng/g (larvae)			0.56			
			9,810 ng/g (adult)			0.56			

Summary of Biological Effects Tissue Concentrations for Fluoranthene

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									
Oncorhynchus mykiss, Rainbow trout			379 µg/g, liver					[25]	F
Cyprinus carpio, Common carp			183 mg/kg (liver)	Physiological, NOED				[29]	L; no significant increase in erod enzyme and P450 1a protein content
Lepomis macrochirus, Bluegill	4,040 µg/kg		600 µg/kg					[23]	L
Pleuronectes vetulus, English sole	320-25,000 ng/g		<6.6 ng/g liver <2.6 ng/g muscle					[26]	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: PESTICIDE (ORGANOCHLORINE)

Chemical Name (Common Synonyms): HEPTACHLOR **CASRN:** 76-44-8

Chemical Characteristics

Solubility in Water: 0.03 mg/L [1]

Half-Life: No data [2]

Log K_{ow}: 6.26 [3]

Log K_{oc}: 6.15 L/kg organic carbon

Human Health

Oral RfD: 5×10^{-4} mg/kg/day [4]

Confidence: Low, uncertainty factor = 300 [4]

Critical Effect: Liver weight increases in rats; benign and malignant liver tumors in mice

Oral Slope Factor: $4.5 \times 10^{+0}$ per (mg/kg)/day [4]

Carcinogenic Classification: B2 [4]

Wildlife

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

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

Aquatic Organisms

Partitioning Factors: Log BCFs ranged from 5.30 to 11.70 for invertebrates and log BCFs for fishes ranged from 3.87 to 19.34.

Food Chain Multipliers: Food chain multipliers (FCMs) for trophic level 3 aquatic organisms were 20.8 (all benthic food web), 1.6 (all pelagic food web), and 12.7 (benthic and pelagic food web). FCMs for trophic level 4 aquatic organisms were 45.8 (all benthic food web), 3.4 (all pelagic food web), and 21.7 (benthic and pelagic food web) [18].

Toxicity/Bioaccumulation Assessment Profile

Hepatchlor is the most widely used insecticide in the organochlorine class [5]. Heptachlor is resistent to degradation and, therefore, persistent in the environment. Heptachlor acute toxic effects in animals are principally due to hyperexcitation in the nervous system and death is frequently ascribed to respiratory failure [5].

Heptachlor is relatively toxic to aquatic invertebrates. The acute toxicity of heptachlor ranged from 0.11 µg/L (96-h LC50) for *Penaeus duorarum* to 1.5 µg/L (96-h LC50) for *Crassostrea virginica* [6]. Fish are also relatively sensitive to heptachlor. The 96-h LC50 values based on the exposure of sheepshead minnows, pinfish, and spot were 3.68, 3.77, and 0.85µg/L, respectively [6].

Laboratory bioaccumulation exposures with spot showed that heptachlor was metabolized to heptachlor epoxide at all concentrations tested [7]. After 3 days of exposure, heptachlor concentrations averaged 52 percent of total residues. At the end of depuration the relative amount of heptachlor decreased to 10 percent, while heptachlor epoxide increased to 44 percent. Cooking (baking, charbroiling, canning, pan frying and deep frying) reduced the heptachlor contents by an average 40 percent in chinook salmon fillets [8].

Heptachlor was among chemicals responsible for the widespread decline of peregrine falcon populations [9]. Heptachlor concentrations above 4 mg/kg in brain is critical and could be associated with falcon mortality, while a concentration above 1.5 mg/kg in eggs was associated with lower reproductive success of falcons [9]. Birds whose life cycle depends on the aquatic environment contained higher residues of heptachlor in their tissue than the seed eaters [10]. The tissues of red-winged blackbirds and tree swallows demonstrated geographically distinct levels of chlorinated hydrocarbons including heptachlor [11]. The spatial variation of heptachlor concentration in eggs correlated significantly with those found in sediments. Higher concentrations of heptachlor in chick tissue rather than in eggs pointed to a local source of uptake through their diet [11].

Summary of Biological Effects Tissue Concentrations for Heptachlor

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, Eastern oyster		0.08 µg/L	0.43 µg/g	30% shell reduction	7.59			[6]	L
		0.4 µg/L	3.1 µg/g	28% shell reduction				[6]	L
		0.91 µg/L	7.7 µg/g	33% shell reduction				[6]	L
		4 µg/L	18 µg/g	78% shell reduction				[6]	L
		14 µg/L	55 µg/g	98% shell reduction				[6]	L
Crassostrea virginica, Eastern oyster			0.021 mg/kg (whole body)[4]	Growth, ED18				[6]	L; exposure media 65% heptachlor (technical grade)
			0.016 mg/kg (whole body)[4]	Growth, NOED				[6]	L; exposure media 65% heptachlor (technical grade)
Mercenaria mercenaria, Quahog clam			0.11 mg/kg (whole body)[4]	Behavior, NOED				[15]	L; no effect on feeding activity
Mya arenaria, Soft shell clam			1.3 mg/kg (whole body)[4]	Behavior, NOED				[15]	L; no effect on feeding activity
Penaeus duorarum, Pink shrimp		0.04 µg/L	0.01 µg/g	5% mortality	5.30			[6]	L
		0.2 µg/L	0.033 µg/g	82% mortality				[6]	L

Summary of Biological Effects Tissue Concentrations for Heptachlor

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]**
Palaemonetes vulgaris, Grass shrimp		0.13 µg/L	0.062 ug/g	6% mortality	11.70			[6]	L
		0.44 µg/L	0.2 µg/g	13% mortality				[6]	L
		2 µg/L	0.97 µg/g	70% mortality				[6]	L
		5 µg/L	3.6 µg/g	95% mortality				[6]	L
Fishes									
Oncorhynchus tshawytscha, Chinook salmon			27.9 µg/kg in eggs	Rearing mortality				[12]	F
Cyprinodon variegatus, Sheepshead minnow		2.7 µg/L	20 µg/g	15% mortality	3.87			[6]	L
		3.3 µg/L	33 µg/g	50% mortality				[6]	L
		3.6 µg/L	34 µg/g	50% mortality				[6]	L
		4.0 µg/L	85 µg/g	60% mortality				[6]	L
Cyprinodon variegatus, Sheepshead minnow		8.8 µg/L	133 µg/g	85% mortality	4.33			[6]	L
Cyprinodon variegatus, Sheepshead minnow			4.5 mg/kg (whole body)[4]	Behavior, LOED				[16]	L; decreased swimming activity
			4.8 mg/kg (whole body)[4]	Behavior, LOED				[16]	L; hyperkinetic behavior
			10.4 mg/kg (whole body)[4]	Behavior, NA				[16]	L; hyperkinetic behavior

Summary of Biological Effects Tissue Concentrations for Heptachlor

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]**
			10.4 mg/kg (whole body)[4]	Mortality, NA				[16]	L; 39% decline in survivorship
			4.5 mg/kg (whole body)[4]	Cellular, NOED				[16]	L; no effect on liver, kidney, pancreas, digestive tract histopathology
			4.8 mg/kg (whole body)[4]	Cellular, NOED				[16]	L; no effect on liver, kidney, pancreas, digestive tract histopathology
			10.4 mg/kg (whole body)[4]	Cellular, NOED				[16]	L; no effect on liver, kidney, pancreas, digestive tract histopathology
			4.5 mg/kg (whole body)[4]	Mortality, NOED				[16]	L; no significant effect on mortality
			4.8 mg/kg (whole body)[4]	Mortality, NOED				[16]	L; no significant effect on mortality
			16 mg/kg (whole body)[4]	Mortality, LOED				[17]	L; increase in fry mortality
			26 mg/kg (whole body)[4]	Reproduction, LOED				[17]	L; decreased egg production of adults
			211 mg/kg (whole body)[4]	Reproduction, LOED				[17]	L; decreased fertility of eggs produced by adults
			0.022 mg/kg (whole body)[4]	Mortality, ED5				[6]	L; exposure media 65% heptachlor (technical grade)
Leiostomus xanthurus, Spot		0.14 µg/L	0.34 µg/g		19.34			[7]	L
		0.26 µg/L	0.64 µg/g	25% mortality				[7]	L
		0.58 µg/L	1.73 µg/g	35% mortality				[7]	L
		1.03 µg/L	3.70 µg/g					[7]	L
		0.5 µg/L	1.5 µg/g					[7]	L
		0.65 µg/L	2.3 µg/g					[7]	L

Summary of Biological Effects Tissue Concentrations for Heptachlor

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]**
Leiostomus xanthurus, Spot			2.6 mg/kg (whole body)[4]	Mortality, ED40				[6]	L; exposure media 65% heptachlor (technical grade)
			0.01 mg/kg (whole body)[4]	Mortality, NOED				[6]	L; exposure media 65% heptachlor (technical grade)
			0.01 mg/kg (whole body)[4]	Mortality, NOED				[6]	L; exposure media 65% heptachlor (technical grade)
Lagodon rhomboides, Pinfish			5.7 mg/kg (whole body)[4]	Mortality, NOED				[6]	L; exposure media 65% heptachlor (technical grade)
Wildlife									
Falco peregrinus anatum, American peregrine			0.018-2.070 mg/kg in eggs (1965-1986)					[9]	F
Falco peregrinus pealei, Peale's peregrine			0.015-0.049 mg/kg in eggs (1965-1986)					[9]	F
Falco peregrinus tundrius, Arctic peregrine			0.087-2.710 mg/kg in eggs (1965-1987)					[9]	F

Summary of Biological Effects Tissue Concentrations for Heptachlor

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]**
Martes americana, Marten			0.3 - 4.5 µg/kg in muscle; 9.1 - 12.7 µg/kg in liver					[14]	F
Martes pennanti, Fishers			1 - 5.7 µg/kg in muscle 5.8 -17µg/kg in liver					[14]	F
Quail			0.86 - 1.15 mg/kg					[13]	F
Woodcock			0.86 - 1.29 mg/kg					[13]	F
Agelaius phoeniceus, Red-winged blackbird	0.2 ng/g 0.2 ng/g 0.2 ng/g		4.1 ng/g in eggs 3.7 ng/g in eggs 4.3 ng/g in eggs		1.05 2.34 1.71			[11] [11] [11]	F F 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: METAL

Chemical Name (Common Synonyms): LEAD **CASRN:** 7439-92-1

Chemical Characteristics

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

Log K_{ow}: – **Log K_{oc}:** –

Human Health

Oral RfD: Not available [2] **Confidence:** –

Critical Effect: Changes in levels of certain blood enzymes, altered neurobehavioral development of children. (These changes may occur at blood lead levels so low as to be essentially without a threshold; therefore, the RfD workgroup determined that it was inappropriate to develop an RfD for inorganic lead.)

Oral Slope Factor: Not available [2] **Carcinogenic Classification:** B2 [2]

Wildlife

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

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

Aquatic Organisms

Partitioning Factors: Lead is most soluble in water and is bioavailable at low pH, low organic content, and low concentrations of calcium, iron, manganese, zinc, and cadmium. Lead is capable of forming insoluble metal sulfides and can easily complex with humic acid. The common forms of dissolved lead are lead sulfate, lead chloride, lead hydroxide, and lead carbonate, but the distribution of salts is highly dependent on the pH of the water. Most lead entering surface waters is precipitated in the sediment as carbonates or hydroxides [8]. Log BCFs of 5.15 (cladoceran) [12] and 3.56 (midge) [9] were reported in the literature.

Food Chain Multipliers: Although methylated lead is rapidly taken out from the water, e.g., by rainbow trout, there is no evidence of biomagnification in the aquatic environment [6 and 7].

Toxicity/Bioaccumulation Assessment Profile

The amount of bioavailable lead in sediment is controlled, in large part, by the concentration of acid volatile sulfides (AVS) and organic mater [3,4,5]. Lead is accumulated by aquatic organisms equally from water and through dietary exposure [6]. In the sediments, a portion of lead can be transformed to trimethyllead and tetraalkyllead compounds through chemical and microbial processes. The organolead compounds are much more toxic to aquatic organisms than are the inorganic lead compounds [7]. Bioaccumulation of organolead compounds is rapid and high; these compounds concentrate in the fatty tissues of aquatic organisms. Babukutty and Chacko [8] and others reported a strong correlation between soft tissue concentration of lead in worms and that in the exchangeable fraction of the sediment.

In vertebrates, lead is known to modify the structure and function of the kidney, bone, central nervous system, and the hematopoietic system. It produces adverse biochemical, histopathological, neuropsychological, ferotoxic, teratogenic, and reproductive effects. Inhibition of blood delta aminolevulnic acid dehydratase (ALAD), an enzyme critical in heme formation, has been observed as a result of exposure to lead in a variety of fish, invertebrates, and birds. At sufficiently high concentrations, lead effects are manifested in aquatic organisms as reduced growth, fecundity, and survivorship [9].

Summary of Biological Effects Tissue Concentrations for Lead

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]**
Eichhornia crassipes, Water hyacinth			4.4 mg/kg (leaf)	Growth, NOED				[20]	L; no effect on growth
			4.6 mg/kg (leaf)	Growth, NOED				[20]	L; no effect on growth
			135 mg/kg (root)	Growth, NOED				[20]	L; no effect on growth
			259 mg/kg (root)	Growth, NOED				[20]	L; no effect on growth
			598 mg/kg (root)	Growth, NOED				[20]	L; no effect on growth
			1030 mg/kg (root)	Growth, NOED				[20]	L; no effect on growth
			6 mg/kg (stem)	Growth, NOED				[20]	L; no effect on growth
			16.6 mg/kg (stem)	Growth, NOED				[20]	L; no effect on growth
			48.8 mg/kg (stem)	Growth, NOED				[20]	L; no effect on growth
			70.6 mg/kg (stem)	Growth, NOED				[20]	L; no effect on growth
			4.4 mg/kg (leaf)	Morphology, NOED				[20]	L; no effect on plant appearance
			4.6 mg/kg (leaf)	Morphology, NOED				[20]	L; no effect on plant appearance
			135 mg/kg (root)	Morphology, NOED				[20]	L; no effect on plant appearance
			259 mg/kg (root)	Morphology, NOED				[20]	L; no effect on plant appearance
			598 mg/kg (root)	Morphology, NOED				[20]	L; no effect on plant appearance
			1,030 mg/kg (root)	Morphology, NOED				[20]	L; no effect on plant appearance
			6 mg/kg (stem)	Morphology, NOED				[20]	L; no effect on plant appearance
			16.6 mg/kg (stem)	Morphology, NOED				[20]	L; no effect on plant appearance
			48.8 mg/kg (stem)	Morphology, NOED				[20]	L; no effect on plant appearance
			70.6 mg/kg (stem)	Morphology, NOED				[20]	L; no effect on plant appearance

Summary of Biological Effects Tissue Concentrations for Lead

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											
Invertebrates, field-collected	Total µg/g	SEM µg/g	Filt µg/L	Nonfilt µg/L	Body					[15]	F
	679	569	<0.2	276	67 µg/g						
	113	62	1.2	120	11 µg/g						
	99	55	0.2	38	10 µg/g						
	86	50	0.3	35	32 µg/g						
	38	19	<0.2	9	4 µg/g						
	14	4	0.4	24	0.5 µg/g						
Tubificidae, Oligochaete worms	365 µg/g				16.5 mg/g					[14]	F
	138 µg/g				3.7 mg/g						
	375 µg/g				23.5 mg/g						
	297 µg/g				35.8 mg/g						
	283 µg/g				22.6 mg/g						
Nereis diversicolor, Polychaete worm	44 µg/g				5.9 µg/g					[10]	F
	154 µg/g				4.4 µg/g						
	35 µg/g				3.4 µg/g						
	21 µg/g				0.7 µg/g						
	299 µg/g				5.8 µg/g						
	287 µg/g				4.9 µg/g						
	359 µg/g				3.5 µg/g						
Dreissena polymorpha, Zebra mussel					200 mg/kg (whole body)[6]	Physiological, ED100				[21]	L; mussels stopped filtering
					200 mg/kg (whole body)[6]	Mortality, LOED				[21]	L; increased mortality
					30 mg/kg (whole body)[6]	Physiological, LOED				[21]	L; reduced filtration rate
					2 mg/kg (whole body)[6]	Mortality, NOED				[21]	L; no effect on mortality

Summary of Biological Effects Tissue Concentrations for Lead

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]**
			4 mg/kg (whole body)[6]	Mortality, NOED				[21]	L; no effect on mortality
			6 mg/kg (whole body)[6]	Mortality, NOED				[21]	L; no effect on mortality
			30 mg/kg (whole body)[6]	Mortality, NOED				[21]	L; no effect on mortality
			2 mg/kg (whole body)[6]	Physiological, NOED				[21]	L; no effect on filtration rate
			4 mg/kg (whole body)[6]	Physiological, NOED				[21]	L; no effect on filtration rate
			6 mg/kg (whole body)[6]	Physiological, NOED				[21]	L; no effect on filtration rate
Elliptio complanata, Freshwater mussel	<0.9-28.8 µg/g		ND[4] (foot) ND (muscle) 5.8 µg/g (visceral) 13.0 µg/g (hepatopancreas) 18.8 µg/g (gills) 13.9 µg/g (mantle)					[16]	F
	<0.9-97.5 µg/g		5.5 µg/g (foot) 3.8 µg/g (muscle) 6.9 µg/g (visceral) 14.3 µg/g (hepatopancreas) 36.0 µg/g (gills) 33.3 µg/g (mantle)						

Summary of Biological Effects Tissue Concentrations for Lead

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]**
	<0.9-100.0 µg/g		ND (foot) ND (muscle) 6.0 µg/g (visceral) 15.3 µg/g (hepatopancreas) 35.4 µg/g (gills) 35.6 µg/g (mantle)						
Balanus crenatus, Barnacle			90 mg/kg (whole body)[6]	Behavior, NOED				[23]	L; regulation of metals endpoint - summer experiment
Daphnia magna, Cladoceran			1,880 mg/kg (whole body)[6]	Reproduction, ED10				[12]	L; 10% reduction in number of offspring
			5,040 mg/kg (whole body)[6]	Mortality, ED50				[12]	L; lethal body burden after 21-day exposure
Hyallela azteca, Amphipod		3.3 µg/L	5.8 µg/g	60% survival				[11]	L
		2.6 µg/L	7.1 µg/g	65% survival					
		11.6 µg/L	15.8 µg/g	48% survival					
		8.8 µg/L	19.2 µg/g	31% survival					
		12.6 µg/L	30.0 µg/g	11% survival					
		24.0 µg/L	20.9 µg/g	4% survival					
	Total µg/g	SEM µg/g	Filt µg/L	Nonfilt µg/L	Body			[15]	F
	679	569	<0.2	276	7 µg/g				
	113	62	1.2	120	7 µg/g				
	99	55	0.2	38	6 µg/g				
	86	50	0.3	35	2 µg/g				
	38	19	<0.2	9	6 µg/g				
	14	4	0.4	24	0.4 µg/g				

Summary of Biological Effects Tissue Concentrations for Lead

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]**
Hyalella azteca, Freshwater amphipod			70 mg/kg (whole body)[6]	Mortality, ED50				[22]	L; lethal body burden
			160 mg/kg (whole body)[6]	Mortality, ED50				[22]	L; lethal body burden
			90 mg/kg (whole body)[6]	Mortality, ED50				[22]	L; lethal body burden
			115 mg/kg (whole body)[6]	Mortality, ED50				[22]	L; lethal body burden
Pontoporeia affiniss, Amphipod			4 mg/kg (whole body)[6]	Mortality, NOED				[24]	L; body burden estimated from graph
			4 mg/kg (whole body)[6]	Mortality, NOED				[24]	L; body burden estimated from graph
	Total SEM µg/g µg/g	Filt Nonfilt µg/L µg/L	Body					[15]	F
	679 569	<0.2 276	7 µg/g						
	113 62	1.2 120	7 µg/g						
	99 55	0.2 38	6 µg/g						
	86 50	0.3 35	2 µg/g						
	38 19	<0.2 9	6 µg/g						
	14 4	0.4 24	0.4 µg/g						
Chironomus riparius, Midge		0.728 mg/L	2650 µg/g		3.56			[9]	L
Chironomus gr. thummi, Midge	13.99 mg/kg		12.80 mg/kg 16.22 mg/kg	normal larvae deformed larvae				[13]	F

Summary of Biological Effects Tissue Concentrations for Lead

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]**
Chironomus gr. thummi, Midge			2.56 mg/kg (whole body)[6]	Morphology, NOED				[13]	L, 4th instar larvae
Fishes									
Salvelinus fontinalis, Brook trout			24 mg/kg (gill)	Behavior, LOED				[19]	L; hyperactivity, erratic swimming, loss of equilibrium
			30 mg/kg (kidney)	Behavior, LOED				[19]	L; hyperactivity, erratic swimming, loss of equilibrium
			20 mg/kg (liver)	Behavior, LOED				[19]	L; hyperactivity, erratic swimming, loss of equilibrium
			3.2 mg/kg (red blood cells)	Behavior, LOED				[19]	L; hyperactivity, erratic swimming, loss of equilibrium
			70 mg/kg (gill)	Development, LOED				[19]	L; spinal deformities
			30 mg/kg (kidney)	Development, LOED				[19]	L; spinal deformities
			25 mg/kg (liver)	Development, LOED				[19]	L; spinal deformities
			4.02 mg/kg (whole body)[6]	Development, LOED				[19]	L; reduced embryo hatchability
			4.02 mg/kg (whole body)[6]	Growth, LOED				[19]	L; reduced weight gain
			70 mg/kg (gill)	Morphology, LOED				[19]	L; darkening of caudal peduncle
			30 mg/kg (kidney)	Morphology, LOED				[19]	L; darkening of caudal peduncle

Summary of Biological Effects Tissue Concentrations for Lead

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]
			25 mg/kg (liver)	Morphology, LOED				[19]	L; darkening of caudal peduncle
			4.02 mg/kg (whole body)[6]	Morphology, LOED				[19]	L; deformed vertebral column
			2.55 mg/kg (whole body)[6]	Development, NOED				[19]	L; no effect on embryo hatchability
			1.6 mg/kg (whole body)[6]	Development, NOED				[19]	L; no effect on embryo hatchability
			38 mg/kg (gill)	Growth, NOED				[19]	L; no effect on length or weight
			70 mg/kg (gill)	Growth, NOED				[19]	L; no effect on growth
			60 mg/kg (gill)	Growth, NOED				[19]	L; no effect on length or weight of first generation fish
			20 mg/kg (gill)	Growth, NOED				[19]	L; no effect on length or weight of first generation fish
			6 mg/kg (gill)	Growth, NOED				[19]	L; no effect on length or weight of first generation fish
			3.2 mg/kg (gonad)	Growth, NOED				[19]	L; no effect on length or weight
			43 mg/kg (kidney)	Growth, NOED				[19]	L; no effect on length or weight
			30 mg/kg (kidney)	Growth, NOED				[19]	L; no effect on growth
			100 mg/kg (kidney)	Growth, NOED				[19]	L; no effect on length or weight of first generation fish
			40 mg/kg (kidney)	Growth, NOED				[19]	L; no effect on length or weight of first generation fish

Summary of Biological Effects Tissue Concentrations for Lead

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 mg/kg (kidney)	Growth, NOED				[19]	L; no effect on length or weight of first generation fish
			13.6 mg/kg (liver)	Growth, NOED				[19]	L; no effect on length or weight
			25 mg/kg (liver)	Growth, NOED				[19]	L; no effect on growth
			18 mg/kg (liver)	Growth, NOED				[19]	L; no effect on length or weight of first generation fish
			16 mg/kg (liver)	Growth, NOED				[19]	L; no effect on length or weight of first generation fish
			4 mg/kg (liver)	Growth, NOED				[19]	L; no effect on length or weight of first generation fish
			0.6 mg/kg (muscle)	Growth, NOED				[19]	L; no effect on length or weight
			1.5 mg/kg (red blood cells)	Growth, NOED				[19]	L; no effect on length or weight
			4 mg/kg (red blood cells)	Growth, NOED				[19]	L; no effect on length or weight of first generation fish
			0.5 mg/kg (red blood cells)	Growth, NOED				[19]	L; no effect on length or weight of first generation fish
			0.2 mg/kg (red blood cells)	Growth, NOED				[19]	L; no effect on length or weight of first generation fish
			6 mg/kg (spleen)	Growth, NOED				[19]	L; no effect on length or weight
			2.55 mg/kg (whole body)[6]	Growth, NOED				[19]	L; no effect on weight gain

Summary of Biological Effects Tissue Concentrations for Lead

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]
			1.6 mg/kg (whole body)[6]	Growth, NOED				[19]	L; no effect on weight gain
			2.55 mg/kg (whole body)[6]	Morphology, NOED				[19]	L; no effect on skeletal deformities
			1.6 mg/kg (whole body)[6]	Morphology, NOED				[19]	L; no effect on skeletal deformities
			38 mg/kg (gill)	Mortality, NOED				[19]	L; no effect on mortality
			70 mg/kg (gill)	Mortality, NOED				[19]	L; no effect on mortality
			60 mg/kg (gill)	Mortality, NOED				[19]	L; no effect on survival of first generation fish
			20 mg/kg (gill)	Mortality, NOED				[19]	L; no effect on survival of first generation fish
			6 mg/kg (gill)	Mortality, NOED				[19]	L; no effect on survival of first generation fish
			3.2 mg/kg (gonad)	Mortality, NOED				[19]	L; no effect on mortality
			43 mg/kg (kidney)	Mortality, NOED				[19]	L; no effect on mortality
			30 mg/kg (kidney)	Mortality, NOED				[19]	L; no effect on mortality
			100 mg/kg (kidney)	Mortality, NOED				[19]	L; no effect on survival of first generation fish
			40 mg/kg (kidney)	Mortality, NOED				[19]	L; no effect on survival of first generation fish
			8 mg/kg (kidney)	Mortality, NOED				[19]	L; no effect on survival of first generation fish
			13.6 mg/kg (liver)	Mortality, NOED				[19]	L; no effect on mortality
			25 mg/kg (liver)	Mortality, NOED				[19]	L; no effect on mortality
			18 mg/kg (liver)	Mortality, NOED				[19]	L; no effect on survival of first generation fish
			16 mg/kg (liver)	Mortality, NOED				[19]	L; no effect on survival of first generation fish
			4 mg/kg (liver)	Mortality, NOED				[19]	L; no effect on survival of first generation fish

Summary of Biological Effects Tissue Concentrations for Lead

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]
			0.6 mg/kg (muscle)	Mortality, NOED				[19]	L; no effect on mortality
			1.5 mg/kg (red blood cells)	Mortality, NOED				[19]	L; no effect on mortality
			4 mg/kg (red blood cells)	Mortality, NOED				[19]	L; no effect on survival of first generation fish
			0.5 mg/kg (red blood cells)	Mortality, NOED				[19]	L; no effect on survival of first generation fish
			0.2 mg/kg (red blood cells)	Mortality, NOED				[19]	L; no effect on survival of first generation fish
			6 mg/kg (spleen)	Mortality, NOED				[19]	L; no effect on mortality
			4.02 mg/kg (whole body)[6]	Mortality, NOED				[19]	L; no effect on mortality
			2.55 mg/kg (whole body)[6]	Mortality, NOED				[19]	L; no effect on mortality
			1.6 mg/kg (whole body)[6]	Mortality, NOED				[19]	L; no effect on mortality
			38 mg/kg (gill)	Reproduction, NOED				[19]	L; no effect on number of viable eggs produced
			70 mg/kg (gill)	Reproduction, NOED				[19]	L; no effect on number of viable eggs produced by second generation fish
			60 mg/kg (gill)	Reproduction, NOED				[19]	L; no effect on number of viable eggs produced
			20 mg/kg (gill)	Reproduction, NOED				[19]	L; no effect on number of viable eggs produced
			6 mg/kg (gill)	Reproduction, NOED				[19]	L; no effect on number of viable eggs produced
			3.2 mg/kg (gonad)	Reproduction, NOED				[19]	L; no effect on number of viable eggs produced
			43 mg/kg (kidney)	Reproduction, NOED				[19]	L; no effect on number of viable eggs produced

Summary of Biological Effects Tissue Concentrations for Lead

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]
			30 mg/kg (kidney)	Reproduction, NOED				[19]	L; no effect on number of viable eggs produced by second generation fish
			100 mg/kg (kidney)	Reproduction, NOED				[19]	L; no effect on number of viable eggs produced
			40 mg/kg (kidney)	Reproduction, NOED				[19]	L; no effect on number of viable eggs produced
			8 mg/kg (kidney)	Reproduction, NOED				[19]	L; no effect on number of viable eggs produced
			13.6 mg/kg (liver)	Reproduction, NOED				[19]	L; no effect on number of viable eggs produced
			25 mg/kg (liver)	Reproduction, NOED				[19]	L; no effect on number of viable eggs produced by second generation fish
			18 mg/kg (liver)	Reproduction, NOED				[19]	L; no effect on number of viable eggs produced
			16 mg/kg (liver)	Reproduction, NOED				[19]	L; no effect on number of viable eggs produced
			4 mg/kg (liver)	Reproduction, NOED				[19]	L; no effect on number of viable eggs produced
			0.6 mg/kg (muscle)	Reproduction, NOED				[19]	L; no effect on number of viable eggs produced
			1.5 mg/kg (red blood cells)	Reproduction, NOED				[19]	L; no effect on number of viable eggs produced
			4 mg/kg (red blood cells)	Reproduction, NOED				[19]	L; no effect on number of viable eggs produced
			0.5 mg/kg (red blood cells)	Reproduction, NOED				[19]	L; no effect on number of viable eggs produced
			0.2 mg/kg (red blood cells)	Reproduction, NOED				[19]	L; no effect on number of viable eggs produced
			6 mg/kg (spleen)	Reproduction, NOED				[19]	L; no effect on number of viable eggs produced

Summary of Biological Effects Tissue Concentrations for Lead

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	107 µg/g 365 µg/g 138 µg/g 241 µg/g 375 µg/g		10.5 mg/g 5.7 mg/g 0.8 mg/g 0.9 mg/g 20.0 mg/g					[17]	F
Pimephales promelas, Fathead minnow	508 µg/g 297 µg/g 377 µg/g 283 µg/g 286 µg/g		13.6 mg/g 11.9 mg/g 19.5 mg/g 15.1 mg/g 9.3 mg/g					[17]	F
Pimephales promelas, Fathead minnow			0.816 mg/kg (brain)	Behavior, LOED				[25]	L; significant reduction in feeding rate and number of ineffective feeding behaviors with 1-day-old *Daphnia*
			0.451 mg/kg (brain)	Behavior, LOED				[25]	L; significant reduction in number of ineffective feeding behaviors in lowest test concentration with 2-day-old *Daphnia*
			0.451 mg/kg (brain)	Behavior, LOED				[25]	L; significant reduction in feeding rate and number of ineffective feeding behaviors in lowest test concentration with 7-day-old *Daphnia*
			44.2 mg/kg (whole body)[6]	Behavior, LOED				[25]	L; significant reduction in feeding rate and number of ineffective feeding behaviors with 1-day-old *Daphnia*

Summary of Biological Effects Tissue Concentrations for Lead

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]
			26.2 mg/kg (whole body)[6]	Behavior, LOED				[25]	L; significant reduction in number of ineffective feeding behaviors in lowest test concentration with 2-day-old *Daphnia*
			26.2 mg/kg (whole body)[6]	Behavior, LOED				[25]	L; significant reduction in feeding rate and number of ineffective feeding behaviors in lowest test concentration with 7-day-old *Daphnia*
			0.816 mg/kg (brain)	Physiological, LOED				[25]	L; significant reduction norepinephrine and serotonin levels in brain
			44.2 mg/kg (whole body)[6]	Physiological, LOED				[25]	L; significant reduction norepinephrine and serotonin levels in brain
			0.451 mg/kg (brain)	Behavior, NOED				[25]	L; no significant reduction in feeding rate and number of ineffective feeding behaviors with 1-day-old *Daphnia*
			0.816 mg/kg (brain)	Behavior, NOED				[25]	L; no significant reduction in number of ineffective feeding behaviors with 2-day-old *Daphnia*
			26.2 mg/kg (whole body)[6]	Behavior, NOED				[25]	L; no significant reduction in feeding rate and number of ineffective feeding behaviors with 1-day-old *Daphnia*

Summary of Biological Effects Tissue Concentrations for Lead

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]**
			44.2 mg/kg (whole body)[6]	Behavior, NOED				[25]	L; no significant reduction in number of ineffective feeding behaviors with 2-day-old *Daphnia*
			0.451 mg/kg (brain)	Physiological, NOED				[25]	L; no significant reduction norepinephrine and serotonin levels in brain
			26.2 mg/kg (whole body)[6]	Physiological, NOED				[25]	L; No significant reduction norepinephrine and serotonin levels in brain
Wildlife									
Sterna hirundo, Common tern			247-389 ng/g (eggs) 912-1559 ng/g (feathers)					[18]	F
Sterna forsteri, Forster tern			174 ng/g (eggs) 1527 ng/g (feathers)					[18]	F
Sterna dougallii, Roseate tern			318 ng/g (eggs) 2213 ng/g (feathers)					[18]	F
Rynchops niger, Black skimmer			402-664 ng/g (eggs) 832-4091 ng/g (feathers)					[18]	F
Larus argentatus, Herring gull			1720-6743 ng/g (eggs) 1818-2101 ng/g (feathers)					[18]	F

Summary of Biological Effects Tissue Concentrations for Lead

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]**
Zenaida macroura, Mourning dove			58.35-214.7 mg/kg dry wt (liver alive) 267.3 mg/kg dry wt (liver dead) 346-1,297.6 mg/kg dry wt (kidney alive) 1,901 mg/kg dry wt (kidney dead)	Cellular abnormalities increased with increasing tissue concentrations				[26]	L; dosage was ingested lead shot pellets

[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] ND = not detected.
[5] CBR = critical body residue.
[6] 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: METAL

Chemical Name: METHYLMERCURY **CASRN:** 22967-92-6

Chemical Characteristics

Solubility in Water: No data [1] **Half-Life:** No data [2]

Log K_{ow}: – **Log K_{oc}:** –

Human Health

Oral RfD: 1 x 10^{-4} mg/kg-day [3] **Confidence:** Medium, uncertainty factor = 10

Critical Effect: Developmental neurologic abnormalities in infants

On May 1, 1995, IRIS was updated to include an oral RfD of 1 x 10^{-4} mg/kg/d based on developmental neurological effects in human infants. An oral RfD of 3 x 10^{-4} mg/kg/d for chronic systemic effects of methylmercury among the general adult population was available in IRIS until May 1, 1995; however, it was not listed in the IRIS update on that date. For the purposes of calculating an SV for methylmercury that is protective of developing fetuses and nursing infants, EPA's Office of Water has chosen to continue to use the general adult population RfD of 3 x 10^{-4} mg/kg/d for chronic systemic effects of methylmercury until a value is relisted in IRIS, and to reduce this value by a factor of 5 to derive an RfD of 6 x 10^{-5} mg/kg/d for developmental effects among fetuses and nursing infants. The protective factor of 5 is based on experimental results that suggest a possible 5-fold increase in fetal sensitivity to methylmercury exposure. This more protective approach recommended by the Office of Water was deemed to be most prudent at this time. This approach should be considered interim until such time as the Agency has reviewed new studies on the chronic and developmental effects of methylmercury.

Oral Slope Factor: – **Carcinogenic Classification:** C [3]

Wildlife

Partitioning Factors: Over 90 percent of methylmercury is absorbed from the gastrointestinal tract in animals, and following such absorption most accumulates in erythrocytes, giving red cell to plasma ratios of up to 300 to 1 [4]. This allows for efficient transport through the body and results in a generally uniform pattern of distribution in tissues and organs—blood, kidney, and brain concentrations are within a range of one to three by ratio [5]. There is an exceptional ability of methylmercury to pass the blood-brain barrier, and injury to the central nervous system then arises by strong binding of methylmercury to sulfhydryl residues and subsequent release of mercuric ions to binding sites in the central nervous system. The slow elimination of methylmercury from the body is a result of the high erythrocyte-plasma ratio [4]. Mercury will accumulate in both cerebellum and also cerebral cortex, where it will be tightly bound by sulfhydryl groups. Inside the cell, methylmercury will inhibit protein synthesis and RNA synthesis [6,7]. The effects are particularly important in the developing fetal and young brain of most animals. The ability

of methylmercury to penetrate the placental barrier leads to accumulation in the fetus. The rate of transport across the placental barrier is 10-fold higher than for inorganic mercury. It appears that fetal tissue has a greater binding ability for methylmercury than does the pregnant mother. Exposure via milk is also important for feeding babies. It does appear that pregnant animals may detoxify themselves by transferences to their fetuses [8].

Food Chain Multipliers: In birds, there is a tendency for mercury concentrations to be highest in species feeding on fish (or on other seabirds) [9]. However, when one compares mercury levels among predominantly fish-eating species, levels apparently do not show clear patterns or any evident association with diet composition [10]. Particularly high concentrations have been found in some species of procellariiforms [11]. There is an inverse relationship between total mercury and percent methylmercury in tissues of various avian species [12,13]. Overall, the form of mercury in seabirds is predominantly inorganic, suggesting that biotransformation of ingested methylmercury is an important mechanism by which long-lived and slow-moulting seabirds avoid the toxic effects of accumulating large quantities of methylmercury [14,15]. Among furbearers, mecury burdens are higher in fish-eating species than in herbivorous ones [16]. Mink and river otter accumulate about 10 times more mercury than predatory fishes from the same areas [17]. Nonmarine mammals with mercury concentrations in the liver and kidney in excess of approximately 30 mg/kg of wet weight were likely to suffer mercury intoxification. The results of laboratory studies support this value and indicate that a dietary methylmercury concentration of aproximately 2 to 6 mg/kg of wet weight produced mercury poisoning in feeding experiments using a range of mammalian species [18].

Aquatic Organisms

Partitioning Factors: Concentrations of total mercury in water are usually low, typically on the order of a few nanograms per liter. Elemental mercury adsorbs to sediments, where methylmercury can be produced and destroyed by microbial processes. This complex process is affected by environmental factors [1]. A significant fraction of the total mercury in water is found in the form of methylmercury, the species predominantly accumulated by aquatic organisms [19]. In the Onondaga Lake food web, the percent of total mercury occurring as methylmercury was determined as follows [20]:

Lake water 5%
Interstitial water 37%
Phytoplankton 24%
Zooplankton 40%
Benthic macroinvertebrates 26%
Fishes 96%

Bioconcentration factors (BCFs) for methylmercury are highly variable. Log BCFs for methylmercury in brook trout range from 4.84 to 5.80, depending on the tissue analyzed. Methylmercury concentrations and bioaccumulation factors (BAFs) increased with higher trophic levels in both the pelagic and benthic components of aquatic food webs [20].

Food Chain Multipliers: Fish bioconcentrate methylmercury directly from water by uptake across the gills [21,22,23] and piscivores, such as walleye, readily accumulate mercury from dietary sources [24,25]. Methylmercury accumulation from either source may be substantial, but the relative contribution of each

pathway may vary with fish species [26,27,28,29]. In addition, invertebrates generally have a lower percentage of methylmercury in their tissues than fish or marine mammals [30]. The percentage of methylmercury increases with age in both fish and invertebrates [30].

Mercury is accumulated by all trophic levels with biomagnification occurring up the food web. While sediment is usually the primary source of methylmercury in most aquatic systems, the food web is the main pathway for accumulation [24,25]. High concentrations of organic substances and reduced sulfur can complex free mercury ions in the sediment and reduce the availability to organisms [31,32]. Methylmercury can be accumulated directly from the water by uptake across the gills [21,22,23]. High-trophic-level species tend to accumulate the most methylmercury, with concentrations highest in fish-eating predators. Methylmercury concentrations in higher trophic species often do not correlate with concentrations in environmental media. Correlations have been made between sediment and lower trophic species that typically have a high percentage of inorganic mercury, and between mercury concentrations in higher trophic species and their prey items. The best measure of bioavailability of mercury in any system can be obtained through analysis of mercury concentrations in the biota at the specific site.

The transfer efficiency of mercury through the food web is affected by the form of mercury. Although inorganic mercury is the dominant form in the environment and easily accumulated, it is also depurated quickly. Methylmercury accumulates quickly, depurates very slowly, and therefore has a greater potential to biomagnify in higher-trophic-level species. Pharmacologic half-lives of total mercury in tissues of aquatic organisms have been estimated at approximately 2 months to 1 year in saltwater mussels, 1 to more than 3 years in fishes, and 1.4 to 2.7 years in pinnipeds and dolphins [33]. As the concentration of methylmercury increases in prey items, the transfer efficiency also increases [34]. Methylmercury accumulation from either the water column or food sources might be substantial, but the relative contribution of each pathway varies from species to species [26,27,28,29]. Invertebrates generally have a lower percentage of methylmercury in their tissues than fish or marine mammals, but the percentage can vary greatly, from 1 percent in deposit-feeding polychaetes to almost 100 percent in crabs.

The amount of methylmercury in animal tissues increases proportionately with the age of the organism, with the exception of marine mammals. Because marine mammals feed primarily on fish, they have the greatest potential for the highest tissue concentrations of methylmercury compared to other marine organisms. Contrary to other species or groups of animals, the tissue concentrations of methylmercury are higher in juvenile marine mammals than in adults because the adults can mineralize methylmercury into inorganic mercury [33].

Toxicity/Bioaccumulation Assessment Profile

Methylmercury is the most hazardous mercury species due to its high stability, its lipid solubility, and its ionic properties that lead to a high ability to penetrate the membranes of living organisms [35]. Because methylmercury is lipid-soluble, it can rapidly penetrate the blood-brain barrier [36,37,38,39,40]. Injury to the central nervous system arises by accumulation in the cerebellum and cerebral cortex, where methylmercury binds tightly to sulfhydryl groups, resulting in pathological changes [41]. Inside the cell, methylmercury inhibits protein synthesis and RNA synthesis [6,7].

The early developmental stages of organisms are the most sensitive to the toxic effects of mercury, with methylmercury being more toxic than inorganic mercury. Mercury adversely affects reproduction, growth, behavior, osmoregulation, and oxygen exchange in aquatic organisms. In birds and mammals, comparatively low concentrations of mercury have adverse effects on growth and development, behavior, motor coordination, vision, hearing, histology, and metabolism [33].

Toxicity of methylmercury is dependent on temperature [42], oxygen conditions [43], salinity [44], and the presence of other metals such as zinc and lead [45]. The complex behavior of methylmercury in sediments makes it difficult to predict toxicity from bulk sediment chemistry. Toxicity of mercury has been linked with bioaccumulation, but the situation is complicated by the fact that some animals exposed to low concentrations of mercury can build up a tolerance to this contaminant, as well as detoxify the free metal within their cells via the production of metallothioneins and other metal-binding proteins. Brown et al. [46] propose that toxic effects occur as the binding capacity of these metal-binding proteins becomes saturated.

Summary of Biological Effects Tissue Concentrations for Methylmercury

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									
Phytoplankton		Interstitial water: 0.003 µg/L Lake water: 0.0003 µg/L	32 µg/kg			5.00		[20]	F; estimated from chart; chart reported log BAF values
Crepidula fornicata, Slipper limpet			9.00045013427734 mg/kg (whole body)[5]	Growth, ED25				[62]	L; approximate 25% reduction in growth at lowest test concentration; algal food contained mercury at approximatley 2.9 µg/L in addition to water concentration
			15.0007495880126 mg/kg (whole body)[5]	Reproduction, LOED				[62]	L; significant effect on fecundity (number of gametes); exposure includes mercury in food at approximately 9.5 µg/L

Summary of Biological Effects Tissue Concentrations for Methylmercury

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]
			30.0014991760253 mg/kg (whole body)[5]	Development, NOED				[62]	L; no significant effect on number of live spat at peak settlement; exposure includes mercury in food at approximately 31 µg/L
			30.0014991760253 mg/kg (whole body)[5]	Reproduction, NOED				[62]	L; no significant effect on ability to produce gametes; exposure includes mercury in food at approximately 31 µg/L
			9.00045013427734 mg/kg (whole body)[5]	Reproduction, NOED				[62]	L; no significant effect on fecundity (number of gametes); exposure includes mercury in food at approximately 2.9 µg/L

Summary of Biological Effects Tissue Concentrations for Methylmercury

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]**
Elliptio complanata, Freshwater mussel			43 µg/kg	Relative to least contaminated station (17.9 mg/kg, dry total Hg in sediment vs. 0.07 mg/kg, dry), whole animal ww was reduced by 97 percent				[48]	F; 42 and 84 days exposure; probable effects at tissue concentrations >34 µg/kg, ww
Rangia cuneata, Marsh clam			12 mg/kg (whole body)[5]	Mortality, ED50				[54]	L; lethal to 50% of clams in 7 days
			28 mg/kg (whole body)[5]	Mortality, ED50				[54]	L; lethal to 50% of clams in 7 days
			73.1399993896484 mg/kg (whole body)[5]	Mortality, LOED				[54]	L; lethal body burden
			6 mg/kg (whole body)[5]	Mortality, NOED				[54]	L; no effect on mortality
Zooplankton, Cladocerans		Interstitial water: 0.003 µg/L Lake water: 0.0003 µg/L	260 µg/kg			5.94		[20]	F; estimated from chart; chart reported log BAF values

Summary of Biological Effects Tissue Concentrations for Methylmercury

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]**
Diaptomus oregonensis		Unfiltered water: total Hg = 0.43-4.79 ng/L MeHg = 0.04-2.20 ng/L	22-66 µg/kg (dw)		7.10			[47]	F; results were summarized for zooplankton and water samples taken from 12 lakes - ranges are given
Diaptomus minutus, Zooplankton		Filtered water: total Hg = 0.27-4.50 ng/L MeHg = 0.03-1.95 ng/L			4.04			[47]	F; results were summarized for zooplankton and water samples taken from 12 lakes - ranges are given
Holopedium gibberum, Zooplankton		Unfiltered water: total Hg = 0.43-4.79 ng/L MeHg = 0.04-2.20 ng/L Filtered water: total Hg = 0.27-4.50 ng/L MeHg = 0.03-1.95 ng/L	40-419 µg/kg (dw)					[47]	F; results were summarized for biota and water samples taken from 12 lakes - ranges are given

Summary of Biological Effects Tissue Concentrations for Methylmercury

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]**
Bosmina longirostris, Cladoceran		Unfiltered water: total Hg = 0.43-4.79 ng/L MeHg = 0.04-2.20 ng/L Filtered water: total Hg = 0.27-4.50 ng/L MeHg = 0.03-1.95 ng/L	479 µg/kg (dw)					[47]	F; results were summarized for biota and water samples taken from 12 lakes - ranges are given
Daphnia pulex *Daphnia galeatra mendotae* *Daphnia ambigua*, Cladocerans		Unfiltered water: total Hg = 0.43-4.79 ng/L MeHg[4] = 0.04-2.20 ng/L Filtered water: total Hg = 0.27-4.50 ng/L MeHg = 0.03-1.95 ng/L	1-211 µg/kg (dw)					[47]	F; results were summarized for biota and water samples taken from 12 lakes - ranges are given
Daphnia magna, Cladoceran			18.3999996185302 mg/kg (whole body)[5]	Mortality, ED25				[51]	L; 25% reduction in survival compared to controls in 21 days

Summary of Biological Effects Tissue Concentrations for Methylmercury

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]
			2.32800006866455 mg/kg (whole body)[5]	Reproduction, NA				[51]	L; 32% reduction in number of neonates produced in 21 days
			1.63999998569488 mg/kg (whole body)[5]	Reproduction, NA				[51]	L; 35% reduction in number of neonates produced in 21 days
			4.67000007629394 mg/kg (whole body)[5]	Reproduction, NA				[51]	L; 62% reduction in number of neonates produced in 21 days
			7.57000017166137 mg/kg (whole body)[5]	Reproduction, NA				[51]	L; 63% reduction in number of neonates produced in 21 days
			18.3999996185302 mg/kg (whole body)[5]	Reproduction, NA				[51]	L; 99% reduction in number of neonates produced in 21 days
			0.859000027179718 mg/kg (whole body)[5]	Mortality, NOED				[51]	L; no effect on mortality
			1.52600002288818 mg/kg (whole body)[5]	Mortality, NOED				[51]	L; no effect on mortality
			2.32800006866455 mg/kg (whole body)[5]	Mortality, NOED				[51]	L; no effect on mortality
			1.63999998569488 mg/kg (whole body)[5]	Mortality, NOED				[51]	L; no effect on mortality
			4.67000007629394 mg/kg (whole body)[5]	Mortality, NOED				[51]	L; no effect on mortality

Summary of Biological Effects Tissue Concentrations for Methylmercury

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]**
			7.57000017166137 mg/kg (whole body)[5]	Mortality, NOED				[51]	L; no effect on mortality
			0.859000027179718 mg/kg (whole body)[5]	Reproduction, NOED				[51]	L; no significant reproductive impairment
			1.52600002288818 mg/kg (whole body)[5]	Reproduction, NOED				[51]	L; no significant reproductive impairment
Daphnia magna, Cladoceran			0.790000021457672 mg/kg (whole body)[5]	Reproduction, ED10				[55]	L; 10% reduction in number of offspring
			91.3000030517578 mg/kg (whole body)[5]	Mortality, ED50				[55]	L; lethal body burden after 21 day exposure
Benthic invertebrates *Scientific names not given* (amphipods and chironomids)		Interstitial water: 0.003 µg/L Lake water: 0.0003 µg/L	25 µg/kg			$8.3x10^4$		[20]	F
Palaemonetes pugio, Grass shrimp			1.09399998188018 mg/kg (whole body)[5]	Behavior, LOED				[50]	L; decreased sensitivity to physical disturbance

Summary of Biological Effects Tissue Concentrations for Methylmercury

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]**
			2.12299990653991 mg/kg (whole body)[5]	Mortality, NOED				[50]	L; no statistically significant increase in mortality
Uca pugnax, Fiddler crab			12.329999923706 mg/kg (whole body)[5]	Development, LOED				[53]	L; inhibition of limb regeneration and molting in male crabs
			19.4200000762939 mg/kg (whole body)[5]	Development, LOED				[53]	L; inhibition of limb regeneration and molting in female crabs
Fishes									
Squalus acanthias, Spiny dogfish			0.0930000022053719 mg/kg (whole body)[5]	Mortality, NOED				[57]	L; no effect on mortality in 24 hours

Summary of Biological Effects Tissue Concentrations for Methylmercury

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		Exposure concentrations (CH_3HgCl): 4 µg/L	kidney = 74±30 (16-116 mg/kg) liver = 76±19 (32-114 mg/kg) spleen = 89±38 (32-118 mg/kg) brain = 19±8 (7-32 mg/kg) muscle = 31±12 (9-52 mg/kg) gill = 66±15 (42-93 mg/kg)	58.2 d±21.4				[49]	L; d = mean days to death ± SD; n = 20 fish per treatment.
		Exposure concentrations (CH_3HgCl): 9 µg/L	whole fish =11.2±6.1 (4.0-27.3 mg/kg)	24.2 d±5.6				[49]	
		Exposure concentrations (CH_3HgCl): 10 µg/L	kidney = 64±20 (40-116 mg/kg) liver = 47±10 (27-65 mg/kg) spleen = 72±22 (37-112 mg/kg) brain = 13±3 (7-19 mg/kg) muscle = 18±5 (9-27 mg/kg) gill = 51±12 (34-85 mg/kg)	21.7 d±6.0				[49]	L; n = 20 per treatment; d = days to death; n = 20 per treatment.

Summary of Biological Effects Tissue Concentrations for Methylmercury

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]
		Exposure concentrations (CH_3HgCl): 13 µg/L	kidney = 39±21 (19-91 mg/kg) liver = 42±27 (16-129 mg/kg) spleen = 51±38 (19-194 mg/kg) brain = 7.7±5.6 (2.3-22 mg/kg) muscle = 6.2±7.7 (1.2-26 mg/kg) gill = 64±15 (36-98 mg/kg)	7.6 d±5.1				[49]	L; n = 20 per treatment; d = days to death; n = 20 per treatment.
		Exposure concentrations (CH_3HgCl): 34 µg/L	kidney = 6.2±2.7 (2.3-10 mg/kg) liver = 7.2±2.8 (3.0-12 mg/kg) spleen = 6.4±3.2 (2.7-14 mg/kg) brain = 1.1±0.3 (0.6-1.5 mg/kg) muscle = 0.7±0.3 (2.7-14 mg/kg) gill = 56±12 (29-73 mg/kg)	1.0 d				[49]	L; d = mean days to death (no SD reported)
Oncorhynchus mykiss, Rainbow trout			1.60000002384185 mg/kg (blood)[5]	Growth, NOED				[52]	L; no effect on growth
			0.100000001490116 mg/kg (blood)[5]	Growth, NOED				[52]	L; no effect on growth

Summary of Biological Effects Tissue Concentrations for Methylmercury

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]
			0.5 mg/kg (brain)[5]	Growth, NOED				[52]	L; no effect on growth
			0.100000001490116 mg/kg (brain)[5]	Growth, NOED				[52]	L; no effect on growth
			0.400000005960464 mg/kg (gill)[5]	Growth, NOED				[52]	L; no effect on growth
			0.100000001490116 mg/kg (gill)[5]	Growth, NOED				[52]	L; no effect on growth
			1.60000002384185 mg/kg (kidney)[5]	Growth, NOED				[52]	L; no effect on growth
			0.200000002980232 mg/kg (kidney)[5]	Growth, NOED				[52]	L; no effect on growth
			1 mg/kg (liver)[5]	Growth, NOED				[52]	L; no effect on growth
			0.100000001490116 mg/kg (liver)[5]	Growth, NOED				[52]	L; no effect on growth
			0.5 mg/kg (muscle)[5]	Growth, NOED				[52]	L; no effect on growth
			0.100000001490116 mg/kg (muscle)[5]	Growth, NOED				[52]	L; no effect on growth
			1.60000002384185 mg/kg (posterior intestine)[5]	Growth, NOED				[52]	L; no effect on growth

Summary of Biological Effects Tissue Concentrations for Methylmercury

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]
			6 mg/kg (posterior intestine)[5]	Growth, NOED				[52]	L; no effect on growth
			1.29999995231628 mg/kg (spleen)[5]	Growth, NOED				[52]	L; no effect on growth
			0.300000011920929 mg/kg (spleen)[5]	Growth, NOED				[52]	L; no effect on growth
			0.140000000596046 mg/kg (whole body)[5]	Growth, NOED				[52]	L; no effect on growth
			0.469999998807907 mg/kg (whole body)[5]	Growth, NOED				[52]	L; no effect on growth
			1.60000002384185 mg/kg (blood)[5]	Mortality, NOED				[52]	L; no effect on mortality
			0.100000001490116 mg/kg (blood)[5]	Mortality, NOED				[52]	L; no effect on mortality
			0.5 mg/kg (brain)[5]	Mortality, NOED				[52]	L; no effect on mortality
			0.100000001490116 mg/kg (brain)[5]	Mortality, NOED				[52]	L; no effect on mortality
			0.400000005960464 mg/kg (gill)[5]	Mortality, NOED				[52]	L; no effect on mortality
			0.100000001490116 mg/kg (gill)[5]	Mortality, NOED				[52]	L; no effect on mortality
			1.60000002384185 mg/kg (kidney)[5]	Mortality, NOED				[52]	L; no effect on mortality

Summary of Biological Effects Tissue Concentrations for Methylmercury

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]
			0.200000002980232 mg/kg (kidney)[5]	Mortality, NOED				[52]	L; no effect on mortality
			1 mg/kg (liver)[5]	Mortality, NOED				[52]	L; no effect on mortality
			0.100000001490116 mg/kg (liver)[5]	Mortality, NOED				[52]	L; no effect on mortality
			0.5 mg/kg (muscle)[5]	Mortality, NOED				[52]	L; no effect on mortality
			0.100000001490116 mg/kg (muscle)[5]	Mortality, NOED				[52]	L; no effect on mortality
			1.60000002384185 mg/kg (posterior intestine)[5]	Mortality, NOED				[52]	L; no effect on mortality
			6 mg/kg (posterior intestine)[5]	Mortality, NOED				[52]	L; no effect on mortality
			1.29999995231628 mg/kg (spleen)[5]	Mortality, NOED				[52]	L; no effect on mortality
			0.300000011920929 mg/kg (spleen)[5]	Mortality, NOED				[52]	L; no effect on mortality
			0.140000000596046 mg/kg (whole body)[5]	Mortality, NOED				[52]	L; no effect on survival
			0.469999998807907 mg/kg (whole body)[5]	Mortality, NOED				[52]	L; no effect on survival

Summary of Biological Effects Tissue Concentrations for Methylmercury

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			15 mg/kg (whole body)[5]	Mortality, ED100				[58]	L; 100% mortality in 15 days
			20 mg/kg (whole body)[5]	Mortality, ED100				[58]	L; 100% mortality in 15 days
			6 mg/kg (whole body)[5]	Mortality, ED50				[58]	L; 50% mortality in 15 days
			4.76000022888183 mg/kg (whole body)[5]	Mortality, ED50				[58]	L; 30 day ED50 for brain
			5.69999980926513 mg/kg (whole body)[5]	Mortality, ED50				[58]	L; 15 day ED50 for single intraperitoneal injection
			3.91000008583068 mg/kg (whole body)[5]	Mortality, ED50				[58]	L; 30 day ED50 for muscle
			2.02999997138977 mg/kg (whole body)[5]	Mortality, ED50				[58]	L; 30 day ED50 for eye
			10 mg/kg (whole body)[5]	Mortality, LOED				[58]	L; 83% mortality in 15 days
			2 mg/kg (whole body)[5]	Mortality, LOED				[58]	L; 33% mortality in 15 days
			5 mg/kg (whole body)[5]	Mortality, LOED				[58]	L; 83% mortality in 15 days
			8 mg/kg (whole body)[5]	Mortality, LOED				[58]	L; 67% mortality in 15 days

Summary of Biological Effects Tissue Concentrations for Methylmercury

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]**
			4 mg/kg (whole body)[5]	Mortality, LOED				[58]	L; 13% mortality in 15 days
			2 mg/kg (whole body)[5]	Mortality, NOED				[58]	L; no mortality in 15 days
Salvelinus fontinalis, Brook trout			46.2000007629394 mg/kg (blood cells)[5]	Development, LOED				[38]	L; affected embryo development
			16.8999996185302 mg/kg (brain)[5]	Development, LOED				[38]	L; affected embryo development
			4.40000009536743 mg/kg (carcass)[5]	Development, LOED				[38]	L; affected embryo development
			22.2000007629394 mg/kg (gill)[5]	Development, LOED				[38]	L; affected embryo development
Salvelinus fontinalis, Brook trout			12.3000001907348 mg/kg (gonad)[5]	Development, LOED				[38]	L; affected embryo development
			26.8999996185302 mg/kg (kidney)[5]	Development, LOED				[38]	L; affected embryo development
			24.3999996185302 mg/kg (liver)[5]	Development, LOED				[38]	L; affected embryo development
			10.1999998092651 mg/kg (muscle)[5]	Development, LOED				[38]	L; affected embryo development
			38.7000007629394 mg/kg (spleen)[5]	Development, LOED				[38]	L; affected embryo development
			46.2000007629394 mg/kg (blood cells)[5]	Growth, LOED				[38]	L; decreased weight

Summary of Biological Effects Tissue Concentrations for Methylmercury

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]**
			16.8999996185302 mg/kg (brain)[5]	Growth, LOED				[38]	L; decreased weight
			4.40000009536743 mg/kg (carcass)[5]	Growth, LOED				[38]	L; decreased weight
			22.2000007629394 mg/kg (gill)[5]	Growth, LOED				[38]	L; decreased weight
			12.3000001907348 mg/kg (gonad)[5]	Growth, LOED				[38]	L; decreased weight
			26.8999996185302 mg/kg (kidney)[5]	Growth, LOED				[38]	L; decreased weight
			24.3999996185302 mg/kg (liver)[5]	Growth, LOED				[38]	L; decreased weight
Salvelinus fontinalis, Brook trout			10.1999998092651 mg/kg (muscle)[5]	Growth, LOED				[38]	L; decreased weight
			38.7000007629394 mg/kg (spleen)[5]	Growth, LOED				[38]	L; decreased weight
			9.39999961853027 mg/kg (whole body)[5]	Mortality, LOED				[38]	L; mortality of offspring
			46.2000007629394 mg/kg (blood cells)[5]	Reproduction, LOED				[38]	L; reduced reproduction
			16.8999996185302 mg/kg (brain)[5]	Reproduction, LOED				[38]	L; reduced reproduction
			4.40000009536743 mg/kg (carcass)[5]	Reproduction, LOED				[38]	L; reduced reproduction

Summary of Biological Effects Tissue Concentrations for Methylmercury

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]**
			22.2000007629394 mg/kg (gill)[5]	Reproduction, LOED				[38]	L; reduced reproduction
			12.3000001907348 mg/kg (gonad)[5]	Reproduction, LOED				[38]	L; reduced reproduction
			26.8999996185302 mg/kg (kidney)[5]	Reproduction, LOED				[38]	L; reduced reproduction
			24.3999996185302 mg/kg (liver)[5]	Reproduction, LOED				[38]	L; reduced reproduction
			10.1999998092651 mg/kg (muscle)[5]	Reproduction, LOED				[38]	L; reduced reproduction
			38.7000007629394 mg/kg (spleen)[5]	Reproduction, LOED				[38]	L; reduced reproduction
			3.40000009536743 mg/kg (whole body)[5]	Reproduction, LOED				[38]	L; reduction in reproduction
			2.70000004768371 mg/kg (whole body)[5]	Development, NOED				[38]	L; no physical abnormalities
Salvelinus fontinalis, Brook trout			21.3999996185302 mg/kg (blood cells)[5]	Growth, NOED				[38]	L; decreased weight
			5.19999980926513 mg/kg (blood cells)[5]	Growth, NOED				[38]	L; decreased weight
			2.29999995231628 mg/kg (blood cells)[5]	Growth, NOED				[38]	L; decreased weight
			5.30000019073486 mg/kg (brain)[5]	Growth, NOED				[38]	L; decreased weight

Summary of Biological Effects Tissue Concentrations for Methylmercury

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]**
			1.70000004768371 mg/kg (brain)[5]	Growth, NOED				[38]	L; decreased weight
			0.800000011920928 mg/kg (brain)[5]	Growth, NOED				[38]	L; decreased weight
			1.60000002384185 mg/kg (carcass)[5]	Growth, NOED				[38]	L; decreased weight
			0.589999973773956 mg/kg (carcass)[5]	Growth, NOED				[38]	L; decreased weight
			0.400000005960464 mg/kg (carcass)[5]	Growth, NOED				[38]	L; decreased weight
			6.19999980926513 mg/kg (gill)[5]	Growth, NOED				[38]	L; decreased weight
			1.60000002384185 mg/kg (gill)[5]	Growth, NOED				[38]	L; decreased weight
			0.699999988079071 mg/kg (gill)[5]	Growth, NOED				[38]	L; decreased weight
			2.90000009536743 mg/kg (gonad)[5]	Growth, NOED				[38]	L; decreased weight
			0.899999976158142 mg/kg (gonad)[5]	Growth, NOED				[38]	L; decreased weight
Salvelinus fontinalis, Brook trout			0.200000002980232 mg/kg (gonad)[5]	Growth, NOED				[38]	L; decreased weight
			8.89999961853027 mg/kg (kidney)[5]	Growth, NOED				[38]	L; decreased weight

Summary of Biological Effects Tissue Concentrations for Methylmercury

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]**
			2.5 mg/kg (kidney)[5]	Growth, NOED				[38]	L; decreased weight
			1.20000004768371 mg/kg (kidney)[5]	Growth, NOED				[38]	L; decreased weight
			8.30000019073486 mg/kg (liver)[5]	Growth, NOED				[38]	L; decreased weight
			2.20000004768371 mg/kg (liver)[5]	Growth, NOED				[38]	L; decreased weight
			0.699999988079071 mg/kg (liver)[5]	Growth, NOED				[38]	L; decreased weight
			4.90000009536743 mg/kg (muscle)[5]	Growth, NOED				[38]	L; decreased weight
			1.89999997615814 mg/kg (muscle)[5]	Growth, NOED				[38]	L; decreased weight
			1 mg/kg (muscle)[5]	Growth, NOED				[38]	L; decreased weight
			11.8000001907348 mg/kg (spleen)[5]	Growth, NOED				[38]	L; decreased weight
			3.20000004768371 mg/kg (spleen)[5]	Growth, NOED				[38]	L; decreased weight
			1.20000004768371 mg/kg (spleen)[5]	Growth, NOED				[38]	L; decreased weight
			2.70000004768371 mg/kg (whole body)[5]	Mortality, NOED				[38]	L; no effect on mortality
Salvelinus fontinalis, Brook trout			21.3999996185302 mg/kg (blood cells)[5]	Reproduction, NOED				[38]	L; reduced reproduction

Summary of Biological Effects Tissue Concentrations for Methylmercury

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]
			5.19999980926513 mg/kg (blood cells)[5]	Reproduction, NOED				[38]	L; reduced reproduction
			2.29999995231628 mg/kg (blood cells)[5]	Reproduction, NOED				[38]	L; reduced reproduction
			5.30000019073486 mg/kg (brain)[5]	Reproduction, NOED				[38]	L; reduced reproduction
			1.70000004768371 mg/kg (brain)[5]	Reproduction, NOED				[38]	L; reduced reproduction
			0.800000011920928 mg/kg (brain)[5]	Reproduction, NOED				[38]	L; reduced reproduction
			1.60000002384185 mg/kg (carcass)[5]	Reproduction, NOED				[38]	L; reduced reproduction
			0.589999973773956 mg/kg (carcass)[5]	Reproduction, NOED				[38]	L; reduced reproduction
			0.400000005960464 mg/kg (carcass)[5]	Reproduction, NOED				[38]	L; reduced reproduction
			6.19999980926513 mg/kg (gill)[5]	Reproduction, NOED				[38]	L; reduced reproduction
			1.60000002384185 mg/kg (gill)[5]	Reproduction, NOED				[38]	L; reduced reproduction
			0.699999988079071 mg/kg (gill)[5]	Reproduction, NOED				[38]	L; reduced reproduction
			2.90000009536743 mg/kg (gonad)[5]	Reproduction, NOED				[38]	L; reduced reproduction

Summary of Biological Effects Tissue Concentrations for Methylmercury

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]**
			0.899999976158142 mg/kg (gonad)[5]	Reproduction, NOED				[38]	L; reduced reproduction
Salvelinus fontinalis, Brook trout			0.200000002980232 mg/kg (gonad)[5]	Reproduction, NOED				[38]	L; reduced reproduction
			8.89999961853027 mg/kg (kidney)[5]	Reproduction, NOED				[38]	L; reduced reproduction
			2.5 mg/kg (kidney)[5]	Reproduction, NOED				[38]	L; reduced reproduction
			1.20000004768371 mg/kg (kidney)[5]	Reproduction, NOED				[38]	L; reduced reproduction
			8.30000019073486 mg/kg (liver)[5]	Reproduction, NOED				[38]	L; reduced reproduction
			2.20000004768371 mg/kg (liver)[5]	Reproduction, NOED				[38]	L; reduced reproduction
			0.699999988079071 mg/kg (liver)[5]	Reproduction, NOED				[38]	L; reduced reproduction
			4.90000009536743 mg/kg (muscle)[5]	Reproduction, NOED				[38]	L; reduced reproduction
			1.89999997615814 mg/kg (muscle)[5]	Reproduction, NOED				[38]	L; reduced reproduction
			1 mg/kg (muscle)[5]	Reproduction, NOED				[38]	L; reduced reproduction
			11.8000001907348 mg/kg (spleen)[5]	Reproduction, NOED				[38]	L; reduced reproduction

Summary of Biological Effects Tissue Concentrations for Methylmercury

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]**
			3.20000004768371 mg/kg (spleen)[5]	Reproduction, NOED				[38]	L; reduced reproduction
			1.20000004768371 mg/kg (spleen)[5]	Reproduction, NOED				[38]	L; reduced reproduction
Esox lucius, Northern pike			7 mg/kg (whole body)[5]	Physiological, LOED				[60]	F; lowered blood alkaline phosphatase, serum cortisol, emaciation
Planktivores: *Dorosoma cepedianum*, Gizzard shad		Interstitial water: 0.003 µg/L Lake water: 0.0003µg/L	680 µg/kg			6.40		[20]	F; mean methylmercury concentrations in whole bodies of fish were slightly lower than concentrations in fillets for 4 species evaluated (white perch, smallmouth bass, bluegill, and gizzard shad); differences were significant ($P \leq 0.05$, t-test) for bluegill only; BAF value estimated from chart as log BAF

Summary of Biological Effects Tissue Concentrations for Methylmercury

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]**
Benthivores: *Cyprinus carpio*; Carp; *Ictalurus punctatus*, Channel catfish; and *Lepomis macrochirus*, Bluegill		Interstitial water: 0.003 µg/L Lake water: 0.0003 µg/L	480 µg/kg			6.20		[20]	F; mean methylmercury concentrations in whole bodies of fish were slightly lower than concentrations in fillets for 4 species evaluated (white perch, smallmouth bass, bluegill, and gizzard shad); differences were significant ($P \leq 0.05$, t-test) for bluegill only; BAF value estimated from chart as log BAF
Oryzias latipes, Japanese medaka			54 mg/kg (whole body)[5]	Development, ED100				[59]	L; complete failure of eggs to hatch
			56 mg/kg (whole body)[5]	Development, ED100				[59]	L; complete failure of eggs to hatch
			54 mg/kg (whole body)[5]	Morphology, ED100				[59]	L; subcutaneous hemorrhage, deformed vertebrae
			56 mg/kg (whole body)[5]	Morphology, ED100				[59]	

Summary of Biological Effects Tissue Concentrations for Methylmercury

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]
			29 mg/kg (whole body)[5]	Behavior, LOED				[59]	L; hatchlings unable to control fin movement, loss of equilibrium
			29 mg/kg (whole body)[5]	Development, LOED				[59]	L; over 50% reduction in number of eggs which hatched
			29 mg/kg (whole body)[5]	Morphology, LOED				[59]	L; subcutaneous hemorrhage, deformed vertebrae
			16 mg/kg (whole body)[5]	Development, NOED				[59]	L; no effect on hatchability of eggs compared to controls
			16 mg/kg (whole body)[5]	Morphology, NOED				[59]	L; no observations of subcutaneous hemorrhage or deformed vertebrae

Summary of Biological Effects Tissue Concentrations for Methylmercury

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]**
Morone americana, White perch		Interstitial water: 0.003 µg/L Lake water: 0.0003 µg/L	680 µg/kg			6.40		[20]	F; mean methylmercury concentrations in whole bodies of fish were slightly lower than concentrations in fillets for 4 species evaluated (white perch, smallmouth bass, bluegill, and gizzard shad); differences were significant ($P \leq 0.05$, t-test) for bluegill only; BAF value estimated from chart as log BAF
Perca flavescens, Yellow perch			0.135000005364418 mg/kg (whole body)[5]	Growth, NOED				[63]	F, controlled field study; two years but only 1-year-old fish analyzed; basin treated by reducing pH from about 6 to 5.6

Summary of Biological Effects Tissue Concentrations for Methylmercury

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]**
Piscivores: *Microplerus dolomieui*, Smallmouth bass; and *Stizostedion vitreum*, Walleye		Interstitial water: 0.003 µg/L Lake water: 0.0003 µg/L	1,100 µg/kg			3.7×10^6		[20]	F; mean methylmercury concentrations in whole bodies of fish were slightly lower than concentrations in fillets for 4 species evaluated (white perch, smallmouth bass, bluegill, and gizzard shad); differences were significant ($P \leq 0.05$, t-test) for bluegill only; BAF value estimated from chart as log BAF
Stizostedion vitreum, Walleye			0.25 mg/kg (whole body)[5]	Cellular, LOED				[56]	L; multifocal cell atrophy, testicular atrophy
			2.36999988555908 mg/kg (whole body)[5]	Cellular, LOED				[56]	
			0.25 mg/kg (whole body)[5]	Development, LOED				[56]	L; decreased testicular development, lowered gonadosomatic index
			2.36999988555908 mg/kg (whole body)[5]	Development, LOED				[56]	

Summary of Biological Effects Tissue Concentrations for Methylmercury

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]**
			2.36999988555908 mg/kg (whole body)[5]	Growth, LOED				[56]	L; significant reduction in length and weight of males, but not females
			0.25 mg/kg (whole body)[5]	Physiological, LOED				[56]	L; reduced cortisol levels
			0.25 mg/kg (whole body)[5]	Growth, NOED				[56]	L; no effect on length or weight
			0.25 mg/kg (whole body)[5]	Mortality, NOED				[56]	L; no statistically significant increase in mortality
			2.36999988555908 mg/kg (whole body)[5]	Mortality, NOED				[56]	
			2.36999988555908 mg/kg (whole body)[5]	Physiological, NOED				[56]	L; no effect on cortisol levels
Pseudopleuronectes americanus, Winter flounder			5 mg/kg (whole body)[5]	Mortality, LOED				[61]	L; increased mortality
			2 mg/kg (whole body)[5]	Physiological, LOED				[61]	L; increased ornithine decarboxylase activity

Summary of Biological Effects Tissue Concentrations for Methylmercury

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]**
Wildlife									
Larus californicus, California gull			0.404000014066696 mg/kg (brain)[5]	Mortality, NA				[64]	L
			0.828999996185302 mg/kg (breast)[5]	Mortality, NA				[64]	L
			1.08000004291534 mg/kg (liver)[5]	Mortality, NA				[64]	L
Pelecanus occidentalis, Brown pelican			0.202999994158745 mg/kg (brain)[5]	Mortality, NA				[64]	L
			0.347499996423721 mg/kg (breast)[5]	Mortality, NA				[64]	L
			0.806500017642974 mg/kg (liver)[5]	Mortality, NA				[64]	L
Phalacrocorax penicillatus, Brandts cormorant			0.648999989032745 mg/kg (brain)[5]	Mortality, NA				[64]	L
			0.986000001430511 mg/kg (breast)[5]	Mortality, NA				[64]	L
			2.94000005722045 mg/kg (liver)[5]	Mortality, NA				[64]	L
			3.06999993324279 mg/kg (liver)[5]	Mortality, NA				[64]	L

[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] MeHg = methylmercury.
[5] 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: METAL

Chemical Name (Common Synonyms): NICKEL **ASRN:** 7440-02-0

Chemical Characteristics

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

Log K_{ow}: – **Log K_{oc}:** –

Human Health

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

Critical Effect: Decreased body and organ weights

Oral Slope Factor: Not available [2] **Carcinogenic Classification:** A [2]

Wildlife

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

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

Aquatic Organisms

Partitioning Factors: Nickel in the aquatic environment can partition to dissolved and particulate organic carbon. Also, the bioavailability of nickel can be influenced to some extent by the concentrations of calcium and magnesium in water. The bioavailability of nickel in sediments is controlled by the concentration of acid-volatile sulfides (AVS) [8].

Food Chain Multipliers: Little evidence exists to support the general occurrence of biomagnification of nickel in the aquatic environment [9 and 10].

Toxicity/Bioaccumulation Assessment Profile

Bioaccumulation of nickel is most pronounced in sediments when the ratio of simultaneously extracted metals to acid-volatile sulfide (SEM/AVS) is greater than 1. Although nickel concentrations in animals from sediments with SEM/AVS ratios >1 were approximately 2- to 10-fold greater than nickel concentrations in benthic organisms from sediments with SEM/AVS ratio <1, nickel uptake (tissue concentration) was proportional to the concentration in sediment. Ankley et al. [3] have shown that bioaccumulation of nickel from the sediment by *Lumbriculus variegatus* was not predictable based on total sediment metal concentration, but was related to the sediment SEM/AVS ratio.

Summary of Biological Effects Tissue Concentrations for Nickel

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									
Lumbriculus variegatus, Oligochaete worm		0.58 µmol/L	0.10 µmol/g					[3]	F
		16.44 µmol/L	5.00 µmol/g						
		38.24 µmol/L	3.32 µmol/g						
		31.40 µmol/L	0.87 µmol/g						
		4.53 µmol/L	0.07 µmol/g						
		32.77 µmol/L	0.33 µmol/g						
		8.58 µmol/L	1.88 µmol/g						
		14.43 µmol/L	0.97 µmol/g						
		3.72 µmol/L	3.59 µmol/g						
		17.96 µmol/L	2.77 µmol/g						
		0.52 µmol/L	0.10 µmol/g						
		2.75 µmol/L	0.29 µmol/g						
		0.50 µmol/L	1.41 µmol/g						
		3.51 µmol/L	1.91 µmol/g						
		16.67 µmol/L	7.79 µmol/g						
		17.20 µmol/L	0.95 µmol/g						
Tubificidae	51 µg/g		7.20 mg/g					[6]	L
	50 µg/g		3.19 mg/g						
	93 µg/g		6.96 mg/g						
	76 µg/g		12.04 mg/g						
	75 µg/g		9.45 mg/g						

Summary of Biological Effects Tissue Concentrations for Nickel

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]**
Neanthes arenaceodentata, Polychaete worm		<0.28 µmol/L	<0.002 µmol/g	3% mortality				[4]	F
		0.42 µmol/L	0.01 µmol/g	13% mortality					
		2.62 µmol/L	0.01 µmol/g	0% mortality					
		0.16 µmol/L	<0.002 µmol/g	3% mortality					
		<0.74 µmol/L	<0.001 µmol/g	7% mortality					
		3.72 µmol/L	0.01 µmol/g	13% mortality					
		0.37 µmol/L	0.02 µmol/g	0% mortality					
		0.80 µmol/L	<0.006 µmol/g	0% mortality					
		54.30 µmol/L	0.12 µmol/g	20% mortality					
		1.28 µmol/L	<0.002 µmol/g	7% mortality					
		64.8 µmol/L	0.05 µmol/g	10% mortality					
		67.4 µmol/L	0.06 µmol/g	0% mortality					
		36.4 µmol/L	0.12 µmol/g	3% mortality					
		0.86 µmol/L	0.02 µmol/g	0% mortality					
		73.1 µmol/L	0.10 µmol/g	0% mortality				[5]	F
		52.4 µmol/L	0.21 µmol/g	0% mortality					
		449 µmol/L	1.69 µmol/g	3% mortality					
Cerastoderma edule, Clam			56.6 mg/kg (whole body)[4]	Mortality, ED50				[12]	L; estimated body residue by regression from other data values, number of replicates is 2 to 5

Summary of Biological Effects Tissue Concentrations for Nickel

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]**
			128 mg/kg (adductor muscle)[4]	Physiological, NOED				[12]	L; no significant effect on respiration rate at 100 µg/L (highest test concentration at which body residues were measured), number of replicates is 2 to 5
			140 mg/kg (foot)[4]	Physiological, NOED				[12]	
			209 mg/kg (gill)[4]	Physiological, NOED				[12]	
			274 mg/kg (mantle)[4]	Physiological, NOED				[12]	
			138 mg/kg (visceral tissue)[4]	Physiological, NOED				[12]	
			167 mg/kg (whole body)[4]	Physiological,NOED				[12]	
Mytilus galloprovincialis, Mussel			1.1-1.4 mg/kg				0.22	[11]	F
Lamellidans marginalis, Freshwater mussel	110 mg/L		Day 4: 1456.1 µg/g (ctenidium) 432.7 µg/g (mantle) 468.3 µg/g (hepatopancreas) 328.4 µg/g (foot) 373.9 µg/g (adductor muscle)					[5]	L

Summary of Biological Effects Tissue Concentrations for Nickel

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]**
Lamellidans marginalis, Freshwater mussel	22 mg/L		Day 15: 569.8 µg/g (ctenidium) 277.1 µg/g (mantle) 327.1 µg/g (hepatopancreas) 218.6 µg/g (foot) 186.7 µg/g (adductor muscle)					[5]	L
Daphnia magna, Cladoceran			223 mg/kg (whole body)[4]	Mortality, ED50				[6]	L; lethal body burden after 21-day exposure
Fishes									
Cyprinus carpio, Carp		40 mg/L	Day 4: 202.8 mg/L (gill) 226.3 mg/L (kidney) 82.2 mg/L (liver) 97.1 mg/L (brain) 118.1 mg/L (white muscle)					[5]	L
		8 mg/L	Day 15: 103.0 mg/L (gill) 80.3 mg/L (kidney) 97.1 mg/L (liver) 40.6 mg/L (brain) 58.0 mg/L (white) muscle)						

Summary of Biological Effects Tissue Concentrations for Nickel

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	31 µg/g		8.69 mg/g					[7]	F
	51 µg/g		8.19 mg/g						
	50 µg/g		5.66 mg/g						
	57 µg/g		4.02 mg/g						
	93 µg/g		10.72 mg/g						
	73 µg/g		10.10 mg/g						
	76 µg/g		11.51 mg/g						
	60 µg/g		13.32 mg/g						
	75 µg/g		11.75 mg/g						
	53 µg/g		10.90 mg/g						

[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: PESTICIDE (CHLOROPHENOXY)

Chemical Name (Common Synonyms): OXYFLUORFEN **CASRN:** 42874-03-3

Chemical Characteristics

Solubility in Water: No data [1] **Half-Life:** No data [2]

Log K_{ow}: No data [3] **Log K_{oc}:** —

Human Health

Oral RfD: 3 x 10^{-3} mg/kg/day [4] **Confidence:** High, uncertainty factor = 100

Critical Effect: Increased absolute liver weight and nonneoplastic lesions in mice

Oral Slope Factor: 1.3 x 10^{-1} per (mg/kg)/day [5] **Carcinogenic Classification:** C [5]

Wildlife

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

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

Aquatic Organisms

Partitioning Factors: Partitioning factors for oxyfluorfen in aquatic organisms were not found in the literature.

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

Toxicity/Bioaccumulation Assessment Profile

A light activated herbicide, oxyfluorfen at 10^{-2} mM increased cell membrane permeability in *Lemna minor* [6]. The screening tissue value for fish for oxyfluorfen presented by the Chesapeake Bay Program is 800 ng/g [7].

Summary of Biological Effects Tissue Concentrations for Oxyfluorfen

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			**[NO DATA]**						
Fishes			**[NO DATA]**						
Wildlife			**[NO DATA]**						

[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: BIPHENYLS

Chemical Name (Common Synonyms): 2,4,4′-TRICHLOROBIPHENYL **CASRN:** 7012-37-5

Chemical Characteristics

Solubility in Water: No data [1], 160 µg/L [2]

Half-Life: No data [3,4]

Log K_{ow}: 5.60 [2]

Log K_{oc}: 5.51 L/kg organic carbon

Human Health

Oral RfD: No data [5]

Confidence: —

Critical Effect: —

Oral Slope Factor: No data [5]

Carcinogenic Classification: No data [5]

Wildlife

Partitioning Factors: No partitioning factors 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. Log biomagnification factors of 1.07 and 1.97 were determined for total PCBs from alewife to herring gull eggs and from alewife to whole body herring gull, respectively [11]. No specific food chain multipliers were identified for PCB 28.

Aquatic Organisms

Partitioning Factors: Biota-sediment accumulation factors (BSAFs) range from 1.5 to 18.2 for aquatic invertebrate species. The highest BSAF was provided for marine crustaceans.

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 PCB 28 or other trichlorobiphenyls.

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

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], whereas 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′-trachlorobiphenyl (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 Kow 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, 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 in females can be reduced during gametogenesis because of PCB transfer to the more lipophilic eggs. Therefore, the transferred 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 [33]. Toxic responses have been noted to occur at concentrations of 0.03 and 0.014 µg/L in marine and freshwater environments, respectively [33]. The LC50 for grass shrimp exposed to PCBs in marine waters for 4 days was 6.1 to 7.8 µg/L [33]. Chronic toxicity of PCBs presents a serious environmental concern because of their resistance to degradation [34], 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 [35].

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. [36] 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 [37] 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 PCB 28

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									
Microcystis aeruginosa, Daphnia longispina, Plankton	1.35 ± 0.9 ng/g dw (n = 11) (0-20 cm)		0.23 ± 0.22 ng/g (n=14)				3.3	[38]	F; Amsterdam; value is mean ± SD; mean sediment TOC = 9.7%; mean lipid = 0.65%
Plankton, Species not reported	1.949[4] (mean) SD = 0.7309 (n = 9) µg/kg dw	0.008[4] (mean) SD = 0.0031 (n = 3) ng/L	0.350[4] (mean) SD = 0.2353 (n = 5) µg/kg					[39]	F; collected in western Lake Erie (offshore Middle Sister Island); sediment TOC = 7.4% (SD = 1.78), lipid = 1.2% (mean) SD = 0.24
Dreissena polymorpha, Zebra mussel	1.949[4] (mean) SD = 0.7309 (n = 9) µg/kg dw	0.008[4] (mean) SD = 0.0031 (n = 3) ng/L	0.431[4] (mean) SD = 0.4642 (n = 20) µg/kg						lipid = 1.3% (mean) SD = 0.34
Dreissena polymorpha, Zebra mussel	1.35 ± 0.9 ng/g dw (n = 11) (0-20 cm)		0.52 ± 0.36 ng/g (n = 5)				2.8	[38]	F; Amsterdam; value is mean ± SD; mean sediment TOC = 9.7%; mean lipid = 1.74%
Corbicula fluminea, Bivalve	Station B6: 0.4[4] ng/g dw Station C10: 0.03[4] ng/g dw	1.1[4] ng/L <DL[4]	0.33[4] µg/g[5] of lipid (whole animal) 0.3 µg/g[5] of lipid					[40]	F; samples collected from the Rio de la Plata. Sediment depth samples was 0-5 cm. Water sample was filtered.

Summary of Biological Effects Tissue Concentrations for PCB 28

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]**
Crustaceans *Gammarus tigrinus, Assellus aquaticus, Orchestra carimana*	1.35 ± 0.9 ng/g dw (n = 11) (0-20 cm)		2.93 ± 1.41 ng/g (n = 7)				18.2	[38]	F; Amsterdam; value is mean ± SD; mean sediment TOC = 9.7%; mean lipid = 0.86%
Gammarus fasciatus, Amphipod	1.949[4] (mean) SD = 0.7309 (n = 9) µg/kg dw	0.008[4] (mean) SD = 0.0031 (n = 3) ng/L	0.666[4] (mean) SD = 0.2768 (n = 4) µg/kg						lipid = 2.1% (mean) SD = 1.04
Orconectes propinquus, Crayfish	1.949[4] (mean) SD = 0.7309 (n = 9) µg/kg dw	0.008[4] (mean) SD = 0.0031 (n = 3) ng/L	0.392[4] (mean) SD = 0.2407 (n = 5) µg/kg						lipid = 1.7% (mean) SD = 0.11
Hydropsyche alterans, Caddisfly larva	1.949[4] (mean) SD = 0.7309 (n = 9) µg/kg dw	0.008[4] (mean) SD = 0.0031 (n = 3) ng/L	0.369[4] (n = 1) µg/kg						lipid = 1.7% (mean)
Fishes									
Prochilodus platensis, Fish	Station F17: 0.08[4] ng/g dw	<DL[4]	0.9[4]µg/g[5] of lipid					[40]	F; samples collected from the Rio de la Plata.
Oligosarcus jenynsi, Fish	Station A1: 6[4] ng/g dw	0.7[4] ng/L	0.3[4] µg/g[5] of lipid					[40]	F; samples collected from the Rio Santiago.

Summary of Biological Effects Tissue Concentrations for PCB 28

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]**
Anguilla anguilla, Eel	1.35 ± 0.9 ng/g dw (n = 11) (0-20 cm)		3.98 ± 3.42 ng/g (n = 6)				1.5	[38]	F; Amsterdam; value is mean ± SD; mean sediment TOC = 9.7%; mean lipid = 14.9%

[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] Reported concentrations reflect both congeners 28 and 31.
[5] Not clear from reference if concentration is based on wet or dry weight.

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Chemical Category: POLYCHLORINATED BIPHENYLS

Chemical Name (Common Synonyms): 3,3′,4,4′-TETRACHLOROBIPHENYL **CASRN:** 32598-13-3

Chemical Characteristics

Solubility in Water: 0.18 mg/L [1]

Half-Life: No data [2,3]

Log K_{ow}: No data [4], 6.1 [5]

Log K_{oc}: —

Human Health

Oral RfD: No data [6]

Confidence: —

Critical Effect: —

Oral Slope Factor: No data [6]

Carcinogenic Classification: No data [6]

Wildlife

Partitioning Factors: Bioaccumulation factors (BAFs) were determined for mink. The mink had less PCB-77 in their tissues than was measured in their diet. BAF values ranged from 0.1 to 0.2.

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 [7]. 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 [8,9,10]. The results from Biddinger and Gloss [8] and USACE [10] generally agreed that highly water-insoluble compounds (including PCBs) have the potential to biomagnify in these types of food webs. Thomann's [11] model also indicated that highly water-insoluble compounds (log K_{ow} values 5 to 7) showed the greatest potential to biomagnify. Log biomagnification factors (BMFs) for tetrachlorobiphenyls from alewife to herring gulls ranged from 1.52 to 1.83, but were not measured specifically for PCB 77 [12]. A study of arctic marine food chains measured log biomagnification factors for tetrachlorobiphenyls that ranged from 0.08 to 0.40 for fish to seal, <−0.40 for seal to bear, and <−0.30 for fish to bear [13]. Log BMFs calculated for mink fed PCB 77-contaminated feed ranged from −1.00 to −0.70 [40].

Aquatic Organisms

Partitioning Factors: Log bioconcentration factors (BCFs) for blue mussels deployed in New Bedford Harbor, MA, were approximately 6.40 and 6.60 during two years of the study, as reported in the attached summary table [42].

Food Chain Multipliers: Polychlorinated biphenyls as a class have been demonstrated to biomagnify through the food web. Oliver and Niimi [14], 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. [15] 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 PCB 77 or other tetrachlorobiphenyls.

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 [16]. 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.

As a result of their stability and their general hydrophobic nature, PCBs released to the environment have dispersed widely throughout the ecosystem [16]. 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 [17]. 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 [17]. Solubilities and octanol-water partition coefficients for PCB congeners range over several orders of magnitude [18]. 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 [17] , whereas PCB mobility within and sorption to sediment are a function of chlorine substitution pattern and degree of chlorination [19]. The concentration of PCBs in sediments is a function of the physical characteristics of the sediment, such as grain size [20,21] and total organic carbon content [20,21,22,23]. Fine sediments typically contain higher concentrations of PCBs than coarser sediments because of more surface area [17]. Mobility of PCBs in sediment is generally quite low for the higher chlorinated biphenyls [19]. Therefore, it is common for the lower chlorinated PCBs to have a greater dispersion from the original point source [17]. Limited mobility and high rates of sedimentation could prevent some PCB congeners in the sediment from reaching the overlying water via diffusion [19].

The persistence of PCBs in the environment is a result of their general resistance to degradation [18]. The rate of degradation of PCB congeners by bacteria decreases with increasing degree of chlorination [24]; other structural characteristics of the individual PCBs can affect susceptibility to microbial degradation to a lesser extent [18]. 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 [23].

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 [25]. 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) [26]. 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) [27]. 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 [27,28]:

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 [29]. 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 [29]. Once taken up by an organism, PCBs partition primarily into lipid compartments [17]. Thus, differences in PCB concentration between species and between different tissues within the same species may reflect differences in lipid content [17]. PCB concentrations in polychaetes and fish have been strongly correlated to their lipid content [30]. 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 [31,32]. 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 [33,34]. 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 [33]. Consequently, fish and other aquatic organisms may accumulate more of the higher chlorinated PCB congeners than is found in the environment [18].

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 [35]. Toxic responses have been noted to occur at concentrations of 0.03 and 0.014 µg/L in marine and freshwater environments, respectively [35]. The LC50 for grass shrimp exposed to PCBs in marine waters for 4 days was 6.1 to 7.8 µg/L [35]. Chronic toxicity of PCBs presents a serious environmental concern because of their resistance to degradation [36], 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 [37].

A number of field and laboratory studies provide evidence of chronic sublethal effects on aquatic organisms at low tissue concentrations [18]. Field and Dexter [18] 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 [38] 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 [39] 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 [18].

Summary of Biological Effects Tissue Concentrations for PCB 77

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									
Mytilus edulis, Blue mussel		1993: Particulate 1.7 µg/L ±0.5 n = 9 Dissolved 1.0 µg/L ±0.1 n = 9			~6.60			[42]	F; New Bedford Harbor, MA; deployment study; tissue concentrations were only presented for 1994 samples; BCF and tissue concentrations are approximations (~) as data were taken from figure
Mytilus edulis, Blue mussel		1994: Particulate 2.3 µg/L ±0.9 n = 3 Dissolved 0.9 µg/L ±0.1 n = 3	-360 ng/g dw (whole body)		~6.40			[42]	Presented for 1994 samples; BCF and tissue concentrations are approximations (~) as data were taken from figure

Summary of Biological Effects Tissue Concentrations for PCB 77

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, Freshwater cladoceran		exposure water 0.1 µg/L	~6.5 ng/mg dw (n = 3)	No significant effect on survival or reproduction; increased biomass				[40]	L; 21-day static renewal tests; tissue concentrations are approximations (~), as data were taken from figures
		1.0 µg/L	~55 ng/mg dw (n = 3)	No significant effect on survival or reproduction; decreased biomass					
Mysis relicta, Epibenthic freshwater shrimp	118.47 µg/kg dw (TOC = 22.8%)		Screened mysids: 0.72 µg/kg					[41]	L; mysids exposed to field contaminated sediments from Lake Champlain, NY; 24-day exposure; screened mysids were screened from direct contact with sediments (% lipid = 5.94 ± 0.27) whole body;
			Unscreened mysids: 8.74 µg/kg						unscreened mysids were allowed to burrow into sediment.(% lipid = 5.80 ± 0.18) whole body

Summary of Biological Effects Tissue Concentrations for PCB 77

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]
Strongylocentrotus droebachiensis, Sea urchin	0.050 ng/g		0.087 ng/g					[43]	F; sediment and biota collected near or in Hamlet in Cambridge Bay, NW Territories, Canada.
Fishes									
Myoxocephalus quadricornis, Fourhorn sculpin	0.050 ng/g dw		0.056 ng/g (liver) 0.11ng/g (whole body)					[43]	F; sediment and biota collected near or in Hamlet in Cambridge Bay, NW Territories, Canada.
Salmonids							0.29	[47]	F
Wildlife									
Falco peregrinus, Peregrine falcon			1.5 ng/g (eggs) (n = 6)	11.4% eggshell thinning				[46]	F; Kola Peninsula, Russia
White leghorn chicken (embryo)			2.6 µg/kg (egg) 8.6 µg/kg (egg)	LD50 LD50				[44]	L; PCBs were injected into the air cell of eggs

Summary of Biological Effects Tissue Concentrations for PCB 77

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]
Mustela vison, Mink	Diet: 11 pg/g[4]		50 pg/g[4] (liver)	NOAEL		No BMF reported		[45]	L; BMF = lipid-normalized concentration in the liver divided by the lipid-normalized dietary concentration
	300 pg/g[4]		45 pg/g[4] (liver)	LOAEL; reduced kit body weights followed by reduced survival		Log BMF = −0.70			
	600 pg/g[4]		50 pg/g[4] (liver)	Reduced kit body weights followed by reduced survival		Log BMF = −1.00			
	1,100 pg/g[4]		90 pg/g[4] (liver)	Significant decrease in number of live kits whelped per female		Log BMF = −1.00			

[1] Concentration units expressed in 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] Not clear whether units are in dry or wet weight.

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Chemical Category: POLYCHLORINATED BIPHENYLS

Chemical Name (Common Synonyms): 3,4,4′,5-TETRACHLOROBIPHENYL **CASRN:** 70362-50-4

Chemical Characteristics

Solubility in Water: No data [1,2]

Half-Life: No data [2,3]

Log K_{ow}: No data [2,4]

Log K_{oc}: —

Human Health

Oral RfD: No data [5]

Confidence: —

Critical Effect: —

Oral Slope Factor: No data [5]

Carcinogenic Classification: No data [5]

Wildlife

Partitioning Factors: Bioaccumulation factors were determined for mink. At PCB 81 concentration ≥ 66 pg/g, the mink had less PCB 81 in their tissues (liver) than was measured in their diet. At low PCB 81 concentrations (e.g., 27 pg/g), there was an increase in the tissue burden. Log BAF values ranged from −0.10 to 0.23.

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. Log biomagnification factors for tetrachlorobiphenyls from alewife to herring gulls ranged from 1.52 to 1.83, but were not measured specifically for PCB 81 [11]. A study of arctic marine food chains measured log biomagnification factors for tetrachlorobiphenyls that ranged from 0.08 to 0.40 for fish to seal, <−0.4 for seal to bear, and <−0.3 for fish to bear [12]. No specific food chain multipliers were identified for PCB 81.

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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) [25]. 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) [26]. 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 [26,27]:

Congener Class	Recommended TEF
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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 [28]. 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 [28]. Once taken up by an organism, PCBs partition primarily into lipid compartments [16]. Thus, differences in PCB concentration between species and between different tissues within the same species may reflect differences in lipid content [16]. PCB concentrations in polychaetes and fish have been strongly correlated to their lipid content [29]. 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 [30, 31]. 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 [32,33]. 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 [32]. Consequently, fish and other aquatic organisms may accumulate more of the higher chlorinated PCB congeners than is found in the environment [17].

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A number of field and laboratory studies provide evidence of chronic sublethal effects on aquatic organisms at low tissue concentrations [17]. Field and Dexter [17] 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. [37] 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 [38] 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 [17].

Summary of Biological Effects Tissue Concentrations for PCB 81

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									
Tubifex sp, Oligochaetes .	0.0006 mg/kg (n = 1)		0.0003 mg/kg (one composite)					[39]	F; lower Detroit River
Fishes									
Cyprinus carpio, Carp	0.0006 mg/kg (n = 1)		0.021±0.012 mg/kg (n = 9)					[39]	F; lower Detroit River
Salmonids							0.67	[42]	F
Wildlife									
Bucephala clangula, Goldeneye	0.0006 mg/kg (n = 1)		0.017±0.0002 mg/kg (n = 3)					[39]	F; lower Detroit River
Aythya affinis, Lesser scaup	0.0006 mg/kg (n = 1)		0.31±0.017 mg/kg (n = 7)					[39]	F; lower Detroit River
Aythya marila, Greater scaup	0.0006 mg/kg (n = 1)		0.046±0.016 mg/kg (n = 3)					[39]	F; lower Detroit River
Falco peregrinus, Peregrine falcon			0.14 ng/g (eggs) (n = 6)	11.4% eggshell thinning				[40]	F; Kola Peninsula, Russia

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Taxa	**Sediment**	**Water**	**Tissue (Sample Type)**	**Effects**	**Log BCF**	**Log BAF**	**BSAF**	**Reference**	**Comments[3]**
Mustela vison, Mink	Diet: 2 pg/g[4]		50 pg/g[4] (liver)	NOAEL		No BMF reported		[41]	L; BMF = lipid-normalized concentration in the liver divided by the lipid-normalized dietary concentration
	27 pg/g[4]		45 pg/g[4] (liver)	LOAEL; reduced kit body weights followed by reduced survival		Log BMF = 0.23			
	66 pg/g[4]		50 pg/g[4] (liver)						
	150 pg/g[4]					Log BMF = −0.10			
			100 pg/g[4] (liver)	Reduced kit body weights followed by reduced survival		Log BMF = 0.00			
				Significant decrease in number of live kits whelped per female					

[1] Concentration units in 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] Not clear whether units are in dry or wet weight.

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Chemical Category: POLYCHLORINATED BIPHENYLS

Chemical Name (Common Synonyms): **CASRN:** 32598-14-4
2,3,3′,4,4′-PENTACHLOROBIPHENYL

Chemical Characteristics

Solubility in Water: No data [1], 0.0008 - 0.17 mg/L [2]

Half-Life: No data [2,3]

Log K_{ow}: 5.6 - 6.5 [2], No data [4]

Log K_{oc}: 5.51 - 6.39 L/kg organic carbon

Human Health

Oral RfD: No data [5]

Confidence: —

Critical Effect: —

Oral Slope Factor: No data [5]

Carcinogenic Classification: No data [5]

Wildlife

Partitioning Factors: One study reported biomagnification factors (BMFs) for mink exposed to PCB-contaminated food. The lipid-normalized BMFs ranged from 3.8 to 6.8 indicating an accumulation of this PCB congener in the liver.

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. The log biomagnification factor for PCB 105 from alewife to herring gulls in Lake Ontario was 2.01 [11]. A study of arctic marine food chains measured log biomagnification factors for pentachlorobiphenyls that ranged from 0.71 to 1.05 for fish to seal, 0.28 to 0.49 for seal to bear, and 1.14 for fish to bear [12].

Aquatic Organisms

Partitioning Factors: Two studies were found that reported laboratory-measured data to calculate non-normalized log bioaccumulation factors (BAFs) and biota-sediment accumulation factors (BSAFs). In the first study the log BAFs determined for marine clams ranged from 0.86 to 1.35 [41]. The BSAFs ranged from 1.63 to 3.85, with the highest BSAF value associated with the lowest BAF. In the second

study, only BSAF for marine clams were reported. These values ranged from 0.22 to 0.68 [42]. A BSAF of 4.49 was determined for salmonids [46].

Food Chain Multipliers: Polychlorinated biphenyls as a class have been demonstrated to biomagnify through the food web. Oliver and Niimi [13], 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. [14] 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 PCB 105 or other pentachlorobiphenyls.

Toxicity/Bioaccumulation Assessment Profile

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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 [34]. Toxic responses have been noted to occur at concentrations of 0.03 and 0.014 µg/L in marine and freshwater environments, respectively [34]. The LC50 for grass shrimp exposed to PCBs in marine waters for 4 days was 6.1 to 7.8 µg/L [34].

Chronic toxicity of PCBs presents a serious environmental concern because of their resistance to degradation [35], 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 [36].

A number of field and laboratory studies provide evidence of chronic sublethal effects on aquatic organisms at low tissue concentrations [17]. Field and Dexter [17] 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. [37] 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 [38] 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 [17].

Summary of Biological Effects Tissue Concentrations for PCB 105

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									
Plankton, Species not given	2.703 (mean) SD = 1.0659 (n = 9) fg/kg dw	Surface water: 0.003 (mean) SD = 0.0020 (n = 3) ng/L	0.666 (mean) SD = 0.1881 (n = 5) fg/kg					[39]	F; collected in western Lake Erie (offshore Middle Sister Island). Sediment TOC = 7.4% (SD-1.78); lipid = 1.2% (mean) SD-0.24
Plankton (a mixture of primarily phytoplankton and some zooplankton)	14 ± 5.1 ng/g dw (0-3 cm) (n = 38)	10 ± 8.4 pg/L (surface water) (n = 7)	0.8 ± 0.2 ng/g (n = 3)					[13]	F; Lake Ontario; value is mean ± SD; lipid content = 0.5%
Mainly *Tubifex tubifex* and *Limnodrilus hoffmeisteri,* Oligochaete	14 ± 5.1 ng/g dw (0-3 cm) (n = 38)	10 ± 8.4 pg/L (surface water) (n = 7)	2.6 ± 1.4 ng/g (n = 6)					[13]	F; Lake Ontario; value is mean ± SD; lipid content = 1%
Dreissena polymorpha, Zebra mussel	2.703 (mean) SD = 1.0659 (n = 9) fg/kg dw	0.003 (mean) SD = 0.0020 (n = 3) ng/L	1.627 (mean) SD = 1.6470 (n = 20) fg/kg						lipid = 1.3% (mean) SD = 0.34

Summary of Biological Effects Tissue Concentrations for PCB 105

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]**
Macoma nasuta, Bent-nose clam	52.6 ng/g dw (grain size < 1 mm)		1,046 ng/g dw (n = 30)			22.2 (dw)	1.63	[41]	L; steady state BAFs were calculated with average tissue residues and sediment concentra-tions from exposure days 42-119
	43.2 ng/g dw (grain size < 0.25 mm)		575 ng/g dw (n = 30)			14.5 (dw)	2.87		
	48.8 ng/g dw (grain size < 0.125 mm)		297 ng/g dw (n = 30)			7.3 (dw)	3.85		
Macoma nasuta, Bent-nose clam	ng/g dw:		ng/g dw:					[42]	L; value given is mean ± SE; sediment TOC ranged from 0.84% to 7.4%
	1.51 ±0.032		6.6±0.83				0.68		
	1.26		1.8±0.67				0.22		
	8.6±0.37		8.2±0.75				0.64		
	20±3.7		11.9±0.84				0.56		
	70±7.6		20.3±2.83				0.39		
Mysis relicta, Mysid	89.97 µg/kg dw (TOC = 22.8%)		Screened mysids: 1.46 µg/kg (whole body)					[40]	L; mysids exposed to field contam-inated sediments from Lake Champlain, NY; 24 day exposure; screened mysids were screened from direct contact with sediments (% lipid = 5.94±0.27); unscreened mysids were allowed to burrow into sediment.(% lipid = 5.80±0.18)
			Unscreened mysids: 9.85 µg/kg (whole body)						

Summary of Biological Effects Tissue Concentrations for PCB 105

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]**
Mysis relicta, Mysid	14 ± 5.1 ng/g dw (0-3 cm) (n = 38)	10 ±8.4 pg/L surface water (n = 7)	8.5 ± 3.5 ng/g (n = 2)					[13]	F; Lake Ontario; value is mean ± SD; lipid content = 3%
Gammarus fasciatus, Amphipod	2.703 (mean) SD = 1.0659 (n = 9) fg/kg dw	0.003 (mean) SD = 0.0020 (n = 3) ng/L	1.611 (mean) SD = 0.7505 (n = 4) fg/kg						lipid = 2.1% (mean) SD = 1.04
Pontoporeia affinis, Amphipod	14 ± 5.1 ng/g dw (0-3 cm) (n = 38)	10 ± 8.4 pg/L (surface water) (n = 7)	12 ± 8 ng/g (n = 6)					[13]	F; Lake Ontario; value is mean ± SD; lipid content = 3%
Orconectes propinquus, Crayfish	2.703 (mean) SD = 1.0659 (n = 9) fg/kg dw	0.003 (mean) SD = 0.0020 (n = 3) ng/L	0.606 (mean) 0.1101 (n = 5) fg/kg						lipid = 1.7% (mean) SD = 0.11
Hydropsyche alterans, Caddisfly larva	2.703 (mean) SD = 1.0659 (n = 9) fg/kg dw	0.003 (mean) SD = 0.0020 (n = 3) ng/L	1.109 (n = 1) fg/kg						lipid = 1.7% (mean)
Fishes									
Alosa pseudoharengus, Alewife	14 ± 5.1 ng/g dw (0-3 cm) (n = 38)	10 ±8.4 pg/L surface water (n = 7)	27 ng/g (one composite)					[13]	F; Lake Ontario; value is mean ± SD; lipid content = 7%

Summary of Biological Effects Tissue Concentrations for PCB 105

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]**
Cottus cognatus, Sculpin	14 ± 5.1 ng/g dw (0-3 cm) (n = 38)	10 ±8.4 pg/L surface water (n = 7)	39 ng/g (one composite)					[13]	F; Lake Ontario; value is mean ± SD; lipid content = 8%
Osmerus mordax, Small rainbow smelt	14 ± 5.1 ng/g dw (0-3 cm) (n = 38)	10 ±8.4 pg/L surface water (n = 7)	15 ± 2.0 ng/g (n = 4)					[13]	F; Lake Ontario; value is mean ± SD; lipid content = 4%
Osmerus mordax, Large rainbow smelt	14 ± 5.1 ng/g dw (0-3 cm) (n = 38)	10 ± 8.4 pg/L (surface water) (n = 7)	38 ng/g (one composite)					[13]	F; Lake Ontario; value is mean ± SD; lipid content = 4%
Salmonids: *Oncorhynchus kisutch,* Coho salmon;	10 ± 8.4 ng/g dw (0-3 cm) (n = 38)	14 ±5.1 pg/L surface water (n = 7)	110 ± 82 ng/g (n = 60)					[13]	F; Lake Ontario; value is mean ± SD; lipid content = 11%; wild fish.
Oncorhynchus mykiss (Salmo gairdner), Rainbow trout; *Salvelinus namaycush,* Lake trout; *Salmo trutta,* Brown trout							4.49	[46]	
Wildlife									
Falco peregrinus, Peregrine falcon			72 ng/g (eggs) (n = 6)	11.4% eggshell thinning				[44]	F; Kola Peninsula, Russia
White leghorn chicken embryo			2,200 µg/kg (egg)	LD50				[43]	L; PCBs were injected into the air cell of eggs

Summary of Biological Effects Tissue Concentrations for PCB 105

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]**
Mustela vison, Mink	Diet: 510 pg/g[4]		2,900 pg/g[4] (liver)	NOAEL		Log BMF = 0.58		[45]	L; BMF = lipid-normalized concentration in the liver divided by the lipid-normalized dietary concentra-tion
	12,000 pg/g[4]		54,000 pg/g[4] (liver)	LOAEL; reduced kit body weights followed by reduced survival		Log BMF = 0.68			
	23,000 pg/g[4]		105,000 pg/g[4] (liver)	Reduced kit body weights followed by reduced survival		Log BMF = 0.66			
	41,000 pg/g[4]		181,000 pg/g[4] (liver)	Significant decrease in number of live kits whelped per female		Log BMF = 0.83			

[1] Concentration units given in wet weight unless otherwise indicated.
[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] Not clear whether units are in dry or wet weight.

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Chemical Category: POLYCHLORINATED BIPHENYLS

Chemical Name (Common Synonyms): **CASRN:** 31508-00-6
2,3′,4,4′,5-PENTACHLOROBIPHENYL

Chemical Characteristics

Solubility in Water: No data [1]

Half-Life: No data [2,3]

Log K_{ow}: —

Log K_{oc}: 5.51 - 6.39 L/kg organic carbon

Human Health

Oral RfD: No data [5]

Confidence: —

Critical Effect: —

Oral Slope Factor: No data [5]

Carcinogenic Classification: No data [5]

Wildlife

Partitioning Factors: In a laboratory study with mink, the lipid-normalized ratios of PCB 118 in liver to food ranged from 3.4 to 5.9 (log BMF = 0.53 to 0.77) [49]. The ratio of PCB 118 in three species of duck to sediment in the lower Detroit River ranged from 21 to 35 [40].

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. The biomagnification factor for PCB 118 from alewife to herring gulls in Lake Ontario was 80 [11]. A study of arctic marine food chains measured log biomagnification factors for pentachlorobiphenyls that ranged from 0.71 to 1.05 for fish to seal, 0.28 to 0.49 for seal to bear, and 1.14 for fish to bear [12].

Aquatic Organisms

Partitioning Factors: Steady-state BSAFs for the bent-nose clam ranged from 0.59 to 4.7 in two laboratory studies. The ratio of PCB 118 in carp tissue to sediment from the lower Detroit River was 25.

Food Chain Multipliers: Polychlorinated biphenyls as a class have been demonstrated to biomagnify through the food web. Oliver and Niimi [13], 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. [14] 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 PCB 118 or other pentachlorobiphenyls.

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 [15]. 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.

As a result of their stability and their general hydrophobic nature, PCBs released to the environment have dispersed widely throughout the ecosystem [15]. 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 [16]. 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 [16]. Solubilities and octanol-water partition coefficients for PCB congeners range over several orders of magnitude [17]. 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 [16], whereas PCB mobility within and sorption to sediment are a function of chlorine substitution pattern and degree of chlorination [18]. The concentration of PCBs in sediments is a function of the physical characteristics of the sediment, such as grain size [19,20] and total organic carbon content [19,20,21,22]. Fine sediments typically contain higher concentrations of PCBs than coarser sediments because of more surface area [16]. Mobility of PCBs in sediment is generally quite low for the higher chlorinated biphenyls [18]. Therefore, it is common for the lower chlorinated PCBs to have a greater dispersion from the original point source [16]. Limited mobility and high rates of sedimentation could prevent some PCB congeners in the sediment from reaching the overlying water via diffusion [18].

The persistence of PCBs in the environment is a result of their general resistance to degradation [17]. The rate of degradation of PCB congeners by bacteria decreases with increasing degree of chlorination [23]; other structural characteristics of the individual PCBs can affect susceptibility to microbial degradation to a lesser extent [17]. 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 [22]. 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 [24]. 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) [25]. 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) [26]. 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 [26,27]:

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 [28]. 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 [28]. Once taken up by an organism, PCBs partition primarily into lipid compartments [16]. Thus, differences in PCB concentration between species and between different tissues within the same species may reflect differences in lipid content [16]. PCB concentrations in polychaetes and fish have been strongly correlated to their lipid content [29]. 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 [30,31]. 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 [32,33]. 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 [32]. Consequently, fish and other aquatic organisms may accumulate more of the higher chlorinated PCB congeners than is found in the environment [17].

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 [34]. Toxic responses have been noted to occur at concentrations of 0.03 and 0.014 µg/L in marine and freshwater environments, respectively [34]. The LC50 for grass shrimp exposed to PCBs in marine waters for 4 days was 6.1 to 7.8 µg/L [34]. Chronic toxicity of PCBs presents a serious environmental concern because of their resistance to degradation [35], 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 [36].

A number of field and laboratory studies provide evidence of chronic sublethal effects on aquatic organisms at low tissue concentrations [17]. Field and Dexter [17] 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. [37] 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 [38] 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 [17].

Summary of Biological Effects Tissue Concentrations for PCB 118

: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									
Plankton	4.514 (mean) SD = 1.8449 (n = 9) µg/kg dw	0.007 (mean) SD = 0.0044 (n = 3) ng/L	0.750 (mean) SD = 0.4919 (n = 5) fg/kg					[42]	F; collected in western Lake Erie (offshore Middle Sister Island); sediment TOC = 7.4%; SD = 1.78 lipid = 1.2% (mean) SD = 0.24
Tubifex sp., Oligochaetes	0.017 mg/kg		0.0069 mg/kg					[40]	F; lower Detroit River
Macoma nasuta, Bent-nose clam	ng/g dw: 2.93 ± 0.067 2.5 16.5 ± 1.42 45 ± 9.2 162 ± 16.5		ng/g dw: 20 ± 3.0 12.0 ± 1.89 28.9 ± 2.60 40.3 ± 2.64 66 ± 8.9				1.08 0.73 1.17 0.82 0.54	[43]	L; value given is mean ± SE; sediment TOC ranged from 0.84% to 7.4%
Macoma nasuta, Bent-nose clam	44.2 ng/g dw (grain size < 1 mm)		1,049 ng/g dw (n = 30)			30.3 (dw)	2.02	[41]	L; steady state BAFs were calculated with average tissue residues and sediment concentra-tions from exposure days 42-119.
	36.2 ng/g (grain size < 0.25 mm)		550 ng/g dw (n = 30)			18.5 (dw)	3.28		
	41.6 ng/g dw (grain size < 0.125 mm)		296 ng/g dw (n = 30)			8.4 (dw)	4.74		

Summary of Biological Effects Tissue Concentrations for PCB 118

: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]
Dreissena polymorpha, Zebra mussel	4.514 (mean) SD = 1.8449 (n = 9) µg/kg dw	0.007 (mean) SD = 0.0044 (n = 3) ng/L	2.156 (mean) SD = 0.8847 (n = 20) µg/kg						Lipid = 1.3% (mean) SD = 0.34
Mytilus edulis, Blue mussel		Water column: ~16.0 ng/L	Whole body: ~1,780 ng/g dw					[45]	F; New Bedford Harbor, MA; deployment study; ~ -read all values off figures
		~4.0 ng/L	~1,000 ng/g dw						
		~0.8 ng/L	~130 ng/g dw						
Daphnia magna, Freshwater cladoceran		0.1 fg/L	~3.5 ng/mg dw (n = 3)	No significant effect on survival, reproduction, or biomass				[39]	L; 21-day static renewal tests; tissue concentrations are approximations (~), as data were taken from figures
		1.0 fg/L	~130 ng/mg dw (n = 3)	No significant effect on survival, reproduction, or biomass					
Gammarus fasciatus, Amphipod	4.514 (mean) SD = 1.8449 (n = 9) µg/kg dw	0.007 (mean) SD = 0.0044 (n = 3) ng/L	3.113 (mean) SD = 1.7881 (n = 4) µg/kg						Lipid = 2.1% (mean) SD = 1.04

Summary of Biological Effects Tissue Concentrations for PCB 118

: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 propinquus, Crayfish	4.514 (mean) SD = 1.8449 (n = 9) µg/kg dw	0.007 (mean) SD = 0.0044 (n = 3) ng/L	2.242 (mean) SD = 0.3628 (n = 5) µg/kg						Lipid = 1.7% (mean) SD = 0.11
Hydropsyche alterans, Caddisfly larva	4.514 (mean) SD = 1.8449 (n = 9) µg/kg dw	0.007 (mean) SD = 0.0044 (n = 3) ng/L	4.780 (n = 1) µg/kg						Lipid = 1.7% (mean)
Mysis relicta, Mysid	135.73 µg/kg dw (TOC = 22.8%)		Screened mysids: 2.39 µg/kg (whole body) Unscreened mysids: 15.67 µg/kg (whole body)					[44]	L; mysids exposed to field contaminated sediments from Lake Champlain, NY; 24-day exposure screened mysids were screened from direct contact with sedi-ments (% lipid = 5.94 ± 0.27); unscreened mysids were allowed to burrow into sediment.(% lipid = 5.80 ± 0.18)

Summary of Biological Effects Tissue Concentrations for PCB 118

: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									
Salvelinus namaycush namaycush, Lake trout	0.87 ng/g ± 0.11 n = 4	0.20 ng/L ± 0.29 n = 11	290 ng/g lipid					[46, 47]	F; Siskiwit Lake, Isle Royale, Lake Superior; tissue concentrations are means of concentrations measured in several size classes; organic carbon content of sediment was not presented.
Coregonus culpeaformis neohantoniensus, Whitefish	0.87 ng/g ± 0.11 n = 4	0.20 ng/L ± 0.29 n = 11	280 ng/g lipid						
Salmonids						8.15	4.09	[13]	F; %lipid = 11; %sed OC = 2.7
							1.72	[50]	F
Cyprinus carpio, Carp	0.017 mg/kg (n = 1)		0.42 ± 0.26 mg/kg (n = 9)					[40]	F; lower Detroit River
Wildlife									
Bucephala clangula, Goldeneye	0.017 mg/kg (n = 1)		0.36 ± 0.041 mg/kg (n = 3)					[40]	F; lower Detroit River

Summary of Biological Effects Tissue Concentrations for PCB 118

: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]
Aythya affinis, Lesser scaup	0.017 mg/kg (n = 1)		0.52 ± 0.26 mg/kg (n = 7)					[40]	F; lower Detroit River
Aythya marila, Greater scaup	0.017 mg/kg (n = 1)		0.59 ± 0.10 mg/kg (n = 3)					[40]	F; lower Detroit River
Falco peregrinus, Peregrine falcon			450 ng/g (eggs) (n = 6)	11.4% eggshell thinning				[48]	F; Kola Peninsula, Russia
Mustela vison, Mink	Diet: 1,660 pg/g[4]		8,500 pg/g[4] (liver)	NOAEL		log BMF = 0.53		[49]	L; BMF = lipid-normalized concentration in the liver divided by the lipid-normalized dietary concentra-tion
	35,000 pg/g[4]		20,000 pg/g[4] (liver)	LOAEL; reduced kit body weights followed by reduced survival		log BMF = 0.56			
	68,000 pg/g[4]		284,000 pg/g[4] (liver)	Reduced kit body weights followed by reduced survival		log BMF = 0.63			
	125,000 pg/g[4]		478,000 pg/g[4] (liver)	Significant decrease in number of live kits whelped per female		log BMF = 0.77			

[1] Concentration units expressed in wet weight unless otherwise noted.
[2] BCF = bioconcentration factor, BAF = bioaccumulation factor, SAF = 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] Not clear whether units are in dry or wet weight.

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Chemical Category: POLYCHLORINATED BIPHENYLS

Chemical Name (Common Synonyms): **CASRN:** 57465-28-8
3,3′,4,4′,5-PENTACHLOROBIPHENYL

Chemical Characteristics

Solubility in Water: No data [1]
0.004 - 0.099 mg/L [2]

Half-Life: No data [2,3]

Log K_{ow}: 6.2 - 6.85 [2], No data [4]

Log K_{oc}: 6.09 - 6.73 L/kg organic carbon

Human Health

Oral RfD: No data [5]

Confidence: No data [5]

Critical Effect: —

Oral Slope Factor: No data [5]

Carcinogenic Classification: No data [5]

Wildlife

Partitioning Factors: Partitioning factors for PCB 126 in wildlife were not found.

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. The log biomagnification factor for pentachlorobiphenyls from alewife to herring gulls in Lake Ontario ranged from 1.18 to 2.00 [11]. A study of arctic marine food chains measured biomagnification factors for pentachlorobiphenyls that ranged from 0.71 to 1.05 for fish to seal, 0.28 to 0.49 for seal to bear, and 1.14 for fish to bear [12]. No specific food chain multipliers were identified for PCB 126.

Aquatic Organisms

Partitioning Factors: In an 83-day laboratory study with three-spined stickleback, the lipid-normalized ratio of PCB 126 in food to fish tissue ranged from 3.8 to 6.1. A log bioconcentration factor (BCF) for deployed mussels in New Bedford Harbor, MA, was approximately 6.90, as reported in the attached table.

Food Chain Multipliers: Polychlorinated biphenyls as a class have been demonstrated to biomagnify through the food web. Oliver and Niimi [13], 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. [14] 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 PCB 126 or other pentachlorobiphenyls.

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 [15]. 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.

As a result of their stability and their general hydrophobic nature, PCBs released to the environment have dispersed widely throughout the ecosystem [15]. 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 [16]. 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 [16]. Solubilities and octanol-water partition coefficients for PCB congeners range over several orders of magnitude [17]. 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 [16], whereas PCB mobility within and sorption to sediment are a function of chlorine substitution pattern and degree of chlorination [18]. The concentration of PCBs in sediments is a function of the physical characteristics of the sediment, such as grain size [19,20] and total organic carbon content [19,20,21,22]. Fine sediments typically contain higher concentrations of PCBs than coarser sediments because of more surface area [16]. Mobility of PCBs in sediment is generally quite low for the higher chlorinated biphenyls [18]. Therefore, it is common for the lower chlorinated PCBs to have a greater dispersion from the original point source [16]. Limited mobility and high rates of sedimentation could prevent some PCB congeners in the sediment from reaching the overlying water via diffusion [18].

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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 [24]. 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) [25]. 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) [26]. 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 [26,27]:

Congener Class	Recommended TEF
3,3′,4,4′,5-PentaCB	0.1
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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 [28]. 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 [28]. Once taken up by an organism, PCBs partition primarily into lipid compartments [16]. Thus, differences in PCB concentration between species and between different tissues within the same species may reflect differences in lipid content [16]. PCB concentrations in polychaetes and fish have been strongly correlated to their lipid content [29]. 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 [30,31]. 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 [32,33]. 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 [32]. Consequently, fish and other aquatic organisms may accumulate more of the higher chlorinated PCB congeners than is found in the environment [17].

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 [34]. Toxic responses have been noted to occur at concentrations of 0.03 and 0.014 µg/L in marine and freshwater environments, respectively [34]. The LC50 for grass shrimp exposed to PCBs in marine waters for 4 days was 6.1 to 7.8 µg/L [34]. Chronic toxicity of PCBs presents a serious environmental concern because of their resistance to degradation [35], 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 [36].

A number of field and laboratory studies provide evidence of chronic sublethal effects on aquatic organisms at low tissue concentrations [17]. Field and Dexter [17] 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. [37] 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 [38] 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 [17].

Summary of Biological Effects Tissue Concentrations for PCB 126

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BAF	Log BAF	BSAF	Reference	Comments[3]
Invertebrates									
Mytilus edulis, Blue mussel		1993: particulate 0.2 µg/L ±0.1 n = 9 dissolved 0.02 µg/L ±0.01 n = 9						[39]	F; New Bedford Harbor, MA; deployment study; tissue concentrations were only presented for 1994 samples; BCF and tissue concentrations read from figures (~)
		1994: particulate 0.2 µg/L ±0.1 n = 3 dissolved 0.03 µg/L ±0.01 n = 3	~20 ng/g dw (whole body)		6.90				
Fishes									
Gasterosteus aculeatus, Three-spined stickleback						0.78 (male) 0.58 (female)		[41]	L; 83-day dosing study; BAF = lipid-normalized concent-ration in fish divided by the lipid-normalized concentration in food
Myoxocephalus quadricornis, Four-horn sculpin	0.013 ng/g dw		0.035 ng/g (liver) 0.068 ng/g (whole body)					[40]	F; collected in or near Hamlet in Cambridge Bay, NW Territories, Canada
Salmonids							3.21	[45]	F

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Taxa	Sediment	Water	Tissue (Sample Type)	Effects	Log BAF	Log BAF	BSAF	Reference	Comments[3]
Wildlife									
Sterna hirundo, Common tern (embryo)			45 µg/kg[4] (egg)	35% embryo mortality (through hatching)				[42]	L; PCBs were injected into the air cell of eggs
Falco peregrinus, Peregrine falcon			1.3 ng/g (eggs) (n = 6)	11.4% eggshell thinning				[44]	F; Kola Peninsula, Russia
Falco sparverius, American kestrel (embryo)			65 µg/kg[4] (egg)	LD50 (through hatching)				[42]	L; PCBs were injected into the air cell of eggs
Falco sparverius, American kestrel (nestling)			156 µg/kg[4] (liver)	Histopathology of liver, thyroid, and spleen				[42]	L
Colinus virginianus, Bobwhite (embryo)			24 µg/kg[4] (egg)	LD50 (through hatching)				[42]	L; PCBs were injected into the air cell of eggs
White leghorn chicken (embryo)			0.4 µg/kg (egg)	LD50				[42]	L; PCBs were injected into the air cell of eggs from day 4 of incubation through hatching
White leghorn chicken (embryo)			3.1 µg/kg (egg)	LD50				[43]	L; PCBs were injected into the air cell of eggs from day 7 through day 10 of incubation

[1] Concentration units expressed in wet weight unless otherwise indicated.
[2] BCF = bioconcentration factor, BAF = bioaccumulation factor, BSAF = biota-sediment accumulation factor.
[2] L = laboratory study, spiked sediment, single chemical; F = field study, multiple chemical exposure; other unusual study conditions or observations noted.
[4] Not clear from reference if concentration is based on wet or dry weight.

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Chemical Category: POLYCHLORINATED BIPHENYLS

Chemical Name (Common Synonyms): **CASRN:** 38380-08-4
2,3,3′,4,4′,5-HEXACHLOROBIPHENYL

Chemical Characteristics

Solubility in Water: No data [1], 0.004 - 0.038 mg/L [2] **Half-Life:** No data [2,3]

Log K_{ow}: 6.7 - 7.3 [2] **Log K_{oc}:** 6.59 - 7.18 L/kg organic carbon

Human Health

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

Critical Effect: —

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

Wildlife

Partitioning Factors: In a laboratory study with mink, the lipid-normalized ratios of PCB 156 in liver to food ranged from 5.5 to 11.6. The ratio of PCB 156 in tissues of three species of duck to sediment in the lower Detroit River ranged from 27 to 41.

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. The log biomagnification factors for hexachlorobiphenyls from alewife to herring gulls in Lake Ontario ranged from 1.30 to 2.14 [11]. A study of arctic marine food chains measured log biomagnification factors for hexachlorobiphenyls that ranged from 0.99 to 1.36 for fish to seal, 0.97 to 1.26 for seal to bear, and 2.23 for fish to bear [12]. No specific food chain multipliers were identified for PCB 156.

Aquatic Organisms

Partitioning Factors: In Lake Ontario, ratios of PCB-156 in tissue (wet weight) to sediment (dry weight) for plankton, oligochaetes, mysids, and amphipods were 0.10, 0.14, 0.57, and 1.9 respectively; ratios in sculpin, alewife, rainbow smelt, and salmonids were 6.7, 3.0, 2.9, and 16, respectively. In carp from the lower Detroit River the tissue to sediment ratio (wet weight) was 25. BSAFs for clam in a laboratory study ranged from 0.16 to 0.67.

Food Chain Multipliers: Polychlorinated biphenyls as a class have been demonstrated to biomagnify through the food web. Oliver and Niimi [13], 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. [14] 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 PCB 156 or other hexachlorobiphenyls.

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 [15]. 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.

As a result of their stability and their general hydrophobic nature, PCBs released to the environment have dispersed widely throughout the ecosystem [15]. 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 [16]. 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 [16]. Solubilities and octanol-water partition coefficients for PCB congeners range over several orders of magnitude [17]. Octanol-water partition coefficients, which are often used as estimators of the potential for bioconcentration, are highest for the most chlorinated PCB congeners.

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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 [34]. Toxic responses have been noted to occur at concentrations of 0.03 and 0.014 µg/L in marine and freshwater environments, respectively [34]. The LC50 for grass shrimp exposed to PCBs in marine waters for 4 days was 6.1 to 7.8 µg/L [34]. Chronic toxicity of PCBs presents a serious environmental concern because of their resistance to degradation [35], 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 [36].

A number of field and laboratory studies provide evidence of chronic sublethal effects on aquatic organisms at low tissue concentrations [17]. Field and Dexter [17] 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. [37] 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 [38] 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 [17].

Summary of Biological Effects Tissue Concentrations for PCB 156

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									
Plankton (a mixture of primarily phytoplankton and some zooplankton)	2.1 ± 1.4 ng/g dw (0-3 cm) (n = 38)	Not detected in surface water (n = 7)	0.2 ± 0.1 ng/g (n = 3)					[13]	F; Lake Ontario; value is mean ± SD; lipid content = 0.5%
Mainly *Tubifex tubifex* and *Limnodrilus hoffmeisteri,* Oligochaete	2.1 ± 1.4 ng/g dw (0-3 cm) (n = 38)	Not detected in surface water (n = 7)	0.3 ± 0.4 ng/g (n = 6)					[13]	F; Lake Ontario; value is mean ± SD; lipid content = 1%
Tubifex sp, Oligochaetes	0.0024 mg/kg (n = 1)		0.0016 mg/kg (n = 1)					[39]	F; lower Detroit River
Macoma nasuta, Bent-nose clam	ng/g dw:		ng/g dw:					[40]	L; values given are mean ± SE; sediment TOC ranged from 0.84% to 7.4%. *Macoma* were exposed to 5 sediments containing different PCB concentra-tions; NA means number was not legible.
	0.60 ± 0.019		2.6 ± 0.59				0.67		
	0.48		1.93 ± 0.284				0.61		
	NA		2.61 ± 0.192				0.51		
	11.6 ± 2.29		2.89 ± 0.215				0.23		
	34 ± 5.3		4.1 ± 0.77				0.16		
Pontoporeia affinis, Amphipods	2.1 ± 1.4 ng/g dw (0-3 cm) (n = 38)	Not detected in surface water (n = 7)	3.9 ± 2.3 ng/g (n = 6)					[13]	F; Lake Ontario; value is mean ± SD; lipid content = 3%

Summary of Biological Effects Tissue Concentrations for PCB 156

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]
Mysis relicta, Mysids	2.1 ± 1.4 ng/g dw (0-3 cm) (n = 38)	Not detected in surface water (n = 7)	1.2 ± 0.1 ng/g (n = 2)					[13]	F; Lake Ontario; value is mean ± SD; lipid content = 3%
Fishes									
Salmonids: *Oncorhynchus velinus namaycush,* Coho salmon; *Oncorhynchus mykiss* (*Salmo gairdneri*), Rainbow trout; *Salvelinus namaycush,* Lake trout; *Salmo trutta,* Brown trout	2.1 ± 1.4 ng/g dw (0-3 cm) (n = 38)	Not detected in surface water (n = 7)	34 ± 27 ng/g (n = 60)				3.97	[13]	F; Lake Ontario; value is mean ± SD; lipid content = 11%
Cyprinus carpio, Carp	0.0024 mg/kg (n = 1)		0.061±0.024 mg/kg (n = 9)					[39]	F; lower Detroit River
Cottus cognatus, Sculpin	2.1 ± 1.4 ng/g dw (0-3 cm) (n = 38)	Not detected in surface water (n = 7)	14 ng/g (one composite)					[13]	F; Lake Ontario; value is mean ± SD; lipid content = 8%
Alewife	2.1 ± 1.4 ng/g dw (0-3 cm) (n = 38)	Not detected in surface water (n = 7)	6.3 ng/g (one composite)					[13]	F; Lake Ontario; value is mean ± SD; lipid content = 7%
Osmerus mordax, Small rainbow smelt	2.1 ± 1.4 ng/g dw (0-3 cm) (n = 38)	Not detected in surface water (n = 7)	2.7 ± 1.9 ng/g (n = 4)					[13]	F; Lake Ontario; value is mean ± SD; lipid content = 4%

Summary of Biological Effects Tissue Concentrations for PCB 156

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]
Osmerus mordax, Large rainbow smelt	2.1 ± 1.4 ng/g dw (0-3 cm) (n = 38)	Not detected in surface water (n = 7)	6.1 ng/g (one composite)					[13]	F; Lake Ontario; value is mean ± SD; lipid content = 4%
Wildlife									
Bucephala clangula, Goldeneye	0.0024 mg/kg (n = 1)		0.064±0.018 mg/kg (n = 3)					[39]	F; lower Detroit River
Aythya affinis, Lesser scaup	0.0024 mg/kg (n = 1)		0.090±0.044 mg/kg (n = 7)					[39]	F; lower Detroit River
Aythya marila, Greater scaup	0.0024 mg/kg (n = 1)		0.098±0.0091 mg/kg (n = 3)					[39]	F; lower Detroit River
Falco peregrinus, Peregrine falcon			82 ng/g (eggs) (n = 6)	11.4% eggshell thinning				[41]	F; Kola Peninsula, Russia

Summary of Biological Effects Tissue Concentrations for PCB 156

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]**
Mustela vison, Mink	Diet: 110 pg/g[4]		920 pg/g[4] (liver)	NOAEL		Log BMF = 0.74		[42]	L; BMF = lipid-normalized concentration in the liver divided by the lipid-normalized dietary concentra-tion
	1,300 pg/g[4]		12,000 pg/g[4] (liver)	LOAEL; reduced kit body weights followed by reduced survival		Log BMF = 0.96			
	2,800 pg/g[4]		23,000 pg/g[4] (liver)	reduced kit body weights followed by reduced survival		Log BMF = 0.91			
	5,000 pg/g[4]		37,100 pg/g[4] (liver)	Significant decrease in number of live kits whelped per female		Log BMF = 1.06			

[1] Concentration units expressed in 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] Not clear whether units are in dry or wet weight.

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Chemical Category: POLYCHLORINATED BIPHENYLS

Chemical Name (Common Synonyms): **CASRN:** 32774-16-6
3,3′,4,4′,5,5′-HEXACHLOROBIPHENYL

Chemical Characteristics

Solubility in Water: No data [1], 0.5 mg/L [2] **Half-Life:** No data [2,3]

Log K_{ow}: 7.4 [5] **Log K_{oc}:** 7.27 L/kg organic carbon

Human Health

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

Critical Effect: —

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

Wildlife

Partitioning Factors: In a laboratory study with mink, the lipid-normalized ratios of PCB 169 in liver to food ranged from 12.4 to 21.4.

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. The log biomagnification factors for hexachlorobiphenyls from alewife to herring gulls in Lake Ontario ranged from 1.30 to 2.14 [11]. A study of arctic marine food chains measured log biomagnification factors for hexachlorobiphenyls that ranged from 0.99 to 1.36 for fish to seal, 0.99 to 1.26 for seal to bear, and 2.23 for fish to bear [12]. Log BMFs ranged from 1.09 to 1.33 for mink fed PCB 169 in the diet [40].

Aquatic Organisms

Partitioning Factors: In an 83-day laboratory study with three-spined stickleback, the lipid-normalized ratio of PCB 169 in food to fish tissue (log BAF) ranged from 0.50 to 0.79.

Food Chain Multipliers: Polychlorinated biphenyls as a class have been demonstrated to biomagnify through the food web. Oliver and Niimi [13], 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. [14] 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 PCB 169 or other hexachlorobiphenyls.

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 [15]. 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.

As a result of their stability and their general hydrophobic nature, PCBs released to the environment have dispersed widely throughout the ecosystem [15]. 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 [16]. 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 [16]. Solubilities and octanol-water partition coefficients for PCB congeners range over several orders of magnitude [17]. 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 [18], whereas PCB mobility within and sorption to sediment are a function of chlorine substitution pattern and degree of chlorination [18]. The concentration of PCBs in sediments is a function of the physical characteristics of the sediment, such as grain size [19,20] and total organic carbon content [19,20,21,22]. Fine sediments typically contain higher concentrations of PCBs than coarser sediments because of more surface area [16]. Mobility of PCBs in sediment is generally quite low for the higher chlorinated biphenyls [18]. Therefore, it is common for the lower chlorinated PCBs to have a greater dispersion from the original point source [16]. Limited mobility and high rates of sedimentation could prevent some PCB congeners in the sediment from reaching the overlying water via diffusion [18].

The persistence of PCBs in the environment is a result of their general resistance to degradation [19]. The rate of degradation of PCB congeners by bacteria decreases with increasing degree of chlorination [23]; other structural characteristics of the individual PCBs can affect susceptibility to microbial degradation to a lesser extent [17]. 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 [22].

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 [24]. 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) [25]. 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) [26]. 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 [26,27]:

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 [28]. 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 [28]. Once taken up by an organism, PCBs partition primarily into lipid compartments [16]. Thus, differences in PCB concentration between species and between different tissues within the same species may reflect differences in lipid content [16]. PCB concentrations in polychaetes and fish have been strongly correlated to their lipid content [29]. 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 [30,31]. 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 [32,33]. 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 [32]. Consequently, fish and other aquatic organisms may accumulate more of the higher chlorinated PCB congeners than is found in the environment [17].

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 [34]. Toxic responses have been noted to occur at concentrations of 0.03 and 0.014 µg/L in marine and freshwater environments, respectively [34]. The LC50 for grass shrimp exposed to PCBs in marine waters for 4 days was 6.1 to 7.8 µg/L [34]. Chronic toxicity of PCBs presents a serious environmental concern because of their resistance to degradation [35], 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 [36].

A number of field and laboratory studies provide evidence of chronic sublethal effects on aquatic organisms at low tissue concentrations [17]. Field and Dexter [17] 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. [37] 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 [38] 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 [17].

Summary of Biological Effects Tissue Concentrations for PCB 169

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									
Gasterosteus aculeatus, Three-spined stickleback						0.79 (male) 0.50 (female)		[39]	L; 83-day dosing study; BAF = lipid-normalized concentration in fish divided by the lipid-normalized concentration in food
Wildlife									
Mustela vison, Mink	Diet:								
	2 pg/g[4]		65 pg/g[4] (liver)	NOAEL		Log BMF = 1.33		[40]	L; BMF = lipid-normalized concentration in the liver divided by the lipid-normalized dietary concentration
	5 pg/g[4]		65 pg/g[4] (liver)	LOAEL; reduced kit body weights followed by reduced survival		Log BMF = 1.10			
	10 pg/g[4]		120 pg/g[4] (liver)	Reduced kit body weights followed by reduced survival		Log BMF = 1.09			
	20 pg/g[4]		205 pg/g[4] (liver)	Significant decrease in number of live kits whelped per female		Log BMF = 1.20			

[1] Concentration units expressed as 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] Not clear from reference if concentration is based on wet or dry weight.

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Chemical Category: SUBSTITUTED PHENOLS

Chemical Name (Common Synonyms): PENTACHLOROPHENOL (PCP) **CASRN:** 87-86-5

Chemical Characteristics

Solubility in Water: 14 mg/L at 20°C [1]

Half-Life: 23 - 178 days, sediment grab sample, estimated unacclimated aqueous aerobic biodegradation [2]

Log K_{ow}: 5.09 [3]

Log K_{oc}: 5.00 L/kg organic carbon

Human Health

Oral RfD: 3×10^{-2} mg/kg/day [4]

Confidence: Medium, uncertainty factor = 100

Critical Effect: Liver and kidney pathology

Oral Slope Factor: 1.2×10^{-1} per (mg/kg)/day [4]

Carcinogenic Classification: B2 [4]

Wildlife

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

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

Aquatic Organisms

Partitioning Factors: Partitioning factors for pentachlorophenol in aquatic organisms were not found in the literature.

Food Chain Multipliers: Food chain multipliers for pentachlorophenol in aquatic organims were not found in the literature.

Toxicity/Bioaccumulation Assessment Profile

Technical PCP has been reported to contain chlorodiphenylethers, chlorodibenzo-*p*-dioxins, chlorodibezofurans, and hydroxychlorodiphenylethers, whereas commercial PCP contains significant quantities of tetrachlorophenol [5]. These impurities contribute to PCP toxicity, especially sublethal effects at low concentrations of PCP. PCP undergoes rapid degradation (by chemical, microbiological, or photochemical processes) in the environment.

PCP affects energy metabolism by increasing oxygen consumption and altering the activities of several glycolytic and citric acid cycle enzymes and by increasing the consumption rate of stored lipid [6]. PCP toxicity ranged from 3 to 100 µg/L for invertebrates and 1 to 68 µg/L for fish. In oral doses PCP was fatal to birds at 380 to 580 mg/kg. Adverse sublethal effects in birds were observed in a diet containing 1 mg/kg of PCP [5].

Residues above 11 mg/kg in bird tissues were associated with acute toxicity. Studies with birds showed that PCP killed various species at single oral doses of 380 to 504 mg/kg at dietary concentration of 3,850 mg/kg, fed over a 5-day period. Residues of PCP in dead birds were 11 mg/kg in brain, 20 mg/kg in kidney, and 46 mg/kg in liver [7]. Chickens fed 1 mg/kg PCP over an 8-week period accumulated substantial amounts of PCP: 2 mg/kg in muscle, 80 mg/kg in kidney, 25 mg/kg in liver [8]. Residues of PCP in dead organisms after treatment in rice fields were 8.1 mg/kg in frogs and 36.8 mg/kg in snails, and the residues ranged from 31.2 to 59.5 mg/kg in three fish species [7].

Accumulation of PCP is pH-dependent; at pH 4, PCP is completely protonated and therefore highly lipophilic. At this pH, PCP has the greatest accumulation potential. Conversely, PCP is completely ionized at pH 9. Early studies estimated the lethal body burden or critical body residue for goldfish was 0.36 mmol PCP/kg [12] and 0.75 mmol PCP/kg for brown trout [13] (these were prior to 1985 and are not included in the following table). Experiments with rainbow trout [9] showed that neither the twofold difference in body weight nor the 3-percent difference in body lipid content gave fish resistance to the toxicity of PCP. Mean lethal body residues (= critical body residue) ranged from 0.08 to 0.15 mmol/kg. The PCP accumulation by medaka (*Oryzias latipes*) acclimated in freshwater and saltwater decreased with increased salinity [10]. However, the amount of PCP accumulated by killifish acclimated to freshwater was greater than that accumulated by killifish acclimated to saltwater. The growth rate of bluegill was reduced by 75 percent during the 22-day subchronic exposure to 173 µg/L of PCP [11]. The critical body residue for chlorophenols for fathead minnows ranged from 1.1 to 1.7 mmol/kg [14].

PCP is rapidly accumulated and rapidly excreted, and it has no tendency to persist in living organisms. However, PCP tends to accumulate in mammalian tissues unless it is efficiently conjugated into a readily excretable form [15]. Humans eliminate 75 percent of all PCP in the urine. Rats (*Rattus* sp.) and mice can eliminate PCP in the urine very efficiently; however, rhesus monkeys (*Macaca mulatta*) are unable to excrete PCP efficiently.

Summary of Biological Effects Tissue Concentrations for Pentachlorophenol

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									
Glycera dibranchiata, Polychaete			6.64 mg/kg (whole body)[4]	Cellular, LOED				[20]	L; reduced ability of amebocytes to recognize foreign material
			1.55 mg/kg (whole body)[4]	Physiological, LOED				[20]	L; reduced antibacterial activity
Neanthes virens, Polychaete - sandworm			28 mg/kg (whole body)[4]	Physiological, LOED				[23]	L; significant reduction in coelomic fluid glucose level, number of replicates is 8 to 10
			112 mg/kg (whole body)[4]	Physiological, LOED				[23]	L; decrease in tissue glycogen
			13.8 mg/kg (whole body)[4]	Mortality, ED100				[32]	L; lethal body burden
			469 mg/kg (extractable lipid)[4]	Mortality, ED50				[9]	L; median survival time with fish fed low fat diet for 11 weeks then PCP exposure
			471 mg/kg (extractable lipid)[4]	Mortality, ED50				[9]	L; median survival time with fish fed high fat diet for 11 weeks then PCP exposure

Summary of Biological Effects Tissue Concentrations for Pentachlorophenol

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]
			29.8 mg/kg (whole body)[4]	Mortality, ED50				[9]	L; median survival time with fish fed low fat diet for 11 weeks then PCP exposure
			39.4 mg/kg (whole body)[4]	Mortality, ED50				[9]	L; median survival time with fish fed high fat diet for 11 weeks then PCP exposure

Summary of Biological Effects Tissue Concentrations for Pentachlorophenol

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]
Eisenia fetida, Earthworm,	6.75 mmol/kg 3.75 mmol/kg 2.10 mmol/kg 1.20 mmol/kg 0.68 mmol/kg 0.38 mmol/kg 0.21 mmol/kg 0.12 mmol/kg 0.068 mmol/kg 0.038 mmol/kg		1.39-2.65 mmol/kg 0.74-1.19 mmol/kg 0.62-1.35 mmol/kg 0.56-1.16 mmol/kg 0.59-1.58 mmol/kg 0.51-0.80 mmol/kg 0.33-0.84 mmol/kg 0.79-1.16 mmol/kg 0.44-1.29 mmol/kg 0.21 mmol/kg					[19]	L
Physa sp., Snail			0.33 mg/kg (whole body)[4]	Mortality, NOED				[28]	L; no effect on survivorship in 24 hours

Summary of Biological Effects Tissue Concentrations for Pentachlorophenol

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]
Anodonta anatina, Duck mussel			3.1 mg/kg (whole body)[4]	Behavior, LOED				[30]	L; behavioral changes, distended foot could not be retracted
			1.5 mg/kg (whole body)[4]	Behavior, NOED				[30]	L; no effect on behavior
			3.1 mg/kg (whole body)[4]	Mortality, NOED				[30]	L; no effect on mortality
Mytilus edulis, Blue mussel	5 µg/kg		32-244 µg/kg					[16]	F
Mytilus edulis, Mussel			2.34 mg/kg (whole body)[4]	Physiological, LOED				[34]	L; significant increase in anoxic heat dissipation (j/h/g)at test concentration
			2.34 mg/kg (whole body)[4]	Physiological, NA				[34]	L; 10% reduction in anoxia tolerance as percent of controls
			9.9 mg/kg (whole body)[4]	Physiological, NA				[34]	L; 36% reduction in anoxia tolerance as percent of controls
			29.4 mg/kg (whole body)[4]	Physiological, NA				[34]	L; 54% reduction in anoxia tolerance as percent of controls

Summary of Biological Effects Tissue Concentrations for Pentachlorophenol

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]
Mercenaria mercenaria, Quahog clam			0.498 mg/kg (whole body)[4]	Physiological, LOED				[21]	L; impaired ability to clear flavobacterium
			0.498 mg/kg (whole body)[4]	Mortality, NOED				[21]	L; no effect on mortality
Daphnia magna, Cladoceran			0.45 mg/kg (whole body)[4]	Mortality, NOED				[28]	L; no effect on survivorship in 24 hours
Pontoporeia hoyi, Amphipod			48.6 mg/kg (whole body)[4]	Survival, ED50				[27]	L
		200 mmol/L	3.8 mmol/kg	lethal				[17]	L
		300 mmol/L	5.6 mmol/kg	lethal					
		430 mmol/L	7.6 mmol/kg	lethal					
			CBR = 0.33 to1.1 mmol/kg						
Chironomus riparius, Midge			1.1 mg/kg (whole body)[4]	Behavior, NOED				[29]	L; no effect on swimming behavior
			0.87 mg/kg (whole body)[4]	Behavior, NOED				[29]	L; no effect on swimming behavior
			0.38 mg/kg (whole body)[4]	Behavior, NOED				[29]	L; no effect on swimming behavior

Summary of Biological Effects Tissue Concentrations for Pentachlorophenol

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]
Strongylocentrotus purpuratus, Purple sea urchin			95 mg/kg (whole body)[4]	Development, LOED				[22]	L; increase in number of abnormal embryos
			927 mg/kg (whole body)[4]	Development, LOED				[22]	L; genotoxicity, anaphase aberrations
			662 mg/kg (whole body)[4]	Reproduction, LOED				[22]	L; reduced fertilization of embryos
Fishes									
Oncorhynchus kisutch, Coho salmon		1.3 µg/L	21 µg/kg					[17]	L
Oncorhynchus mykiss, Rainbow trout		1.3 µg/L	24 µg/kg						

Summary of Biological Effects Tissue Concentrations for Pentachlorophenol

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]
		1100 mmol/L	3.8 mmol/kg	lethal				[17]	L
		1150 mmol/L	4.0 mmol/kg	lethal					
		1300 mmol/L	4.3 mmol/kg	lethal					
		1400 mmol/L	4.4 mmol/kg	lethal					
		1600 mmol/L	5.2 mmol/kg	lethal					
		1700 mmol/L	6.0 mmol/kg	lethal					
		2300 mmol/L	8.0 mmol/kg	lethal					
			CBR = 0.08 to 0.15 mmol/kg						
Salmo trutta, Brown trout		0.2 mg/l	200 mg/kg (whole body)[4]	Mortality, ED50				[13]	L; lethal body burden
Salvelinus namaycush, Lake trout		1.3 µg/L	11 µg/kg					[17]	L
Carassius auratus, Goldfish			82 mg/kg (whole body)[4]	Mortality, ED100				[25]	L; lethal body burden
			97 mg/kg (whole body)[4]	Mortality, ED100				[25]	L; lethal body burden
			89 mg/kg (whole body)[4]	Mortality, ED100				[25]	L; lethal body burden

Summary of Biological Effects Tissue Concentrations for Pentachlorophenol

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]
			88 mg/kg (whole body)[4]	Mortality, ED100				[25]	L; lethal body burden
			97 mg/kg (whole body)[4]	Mortality, ED100				[25]	L; lethal body burden
			99 mg/kg (whole body)[4]	Mortality, ED100				[25]	L; lethal body burden
			87 mg/kg (whole body)[4]	Mortality, ED100				[25]	L; lethal body burden
			86 mg/kg (whole body)[4]	Mortality, ED100				[25]	L; lethal body burden
			82 mg/kg (whole body)[4]	Mortality, ED100				[25]	L; lethal body burden
			107 mg/kg (whole body)[4]	Mortality, ED100				[25]	L; lethal body burden
			92 mg/kg (whole body)[4]	Mortality, ED100				[25]	L; lethal body burden
			89 mg/kg (whole body)[4]	Mortality, ED100				[25]	L; lethal body burden
			100 mg/kg (whole body)[4]	Mortality, ED100				[25]	L; lethal body burden
			82 mg/kg (whole body)[4]	Mortality, ED100				[25]	L; lethal body burden

Summary of Biological Effects Tissue Concentrations for Pentachlorophenol

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]**
			99 mg/kg (whole body)[4]	Mortality, ED100				[25]	L; lethal body burden
			86 mg/kg (whole body)[4]	Mortality, ED100				[25]	L; lethal body burden
			95 mg/kg (whole body)[4]	Mortality, ED50				[26]	L; mortality
Pimephales promelas, Fathead minnow			CBR = 1.1-1.7 mmol/kg	50% mortality				[14]	L
Pimephales promelas, Fathead minnow			69 mg/kg (whole body)[4]	Growth, LOED				[33]	L; pH was 8.5
			22.1 mg/kg (whole body)[4]	Growth, LOED				[33]	L; pH was 8.0
			25.1 mg/kg (whole body)[4]	Growth, LOED				[33]	L; pH was 7.5
			43.8 mg/kg (whole body)[4]	Morphology, LOED				[33]	L; pH was 8.0
			69 mg/kg (whole body)[4]	Morphology, LOED				[33]	L; pH was 8.5

Summary of Biological Effects Tissue Concentrations for Pentachlorophenol

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]
			35.1 mg/kg (whole body)[4]	Mortality, LOED				[33]	L; pH was 8.5
			45.9 mg/kg (whole body)[4]	Mortality, LOED				[33]	L; pH was 6.5
			45.9 mg/kg (whole body)[4]	Mortality, LOED				[33]	L; pH was 6.5
			43.8 mg/kg (whole body)[4]	Mortality, LOED				[33]	L; pH was 8.0
			12.6 mg/kg (whole body)[4]	Growth, NOED				[33]	L; pH was 8.0
			12.3 mg/kg (whole body)[4]	Growth, NOED				[33]	L; pH was 7.5
			45.9 mg/kg (whole body)[4]	Growth, NOED				[33]	L; pH was 6.5
			35.1 mg/kg (whole body)[4]	Growth, NOED				[33]	L; pH was 8.5
			35.1 mg/kg (whole body)[4]	Morphology, NOED				[33]	L; pH was 8.5
			22.1 mg/kg (whole body)[4]	Morphology, NOED				[33]	L; pH was 8.0
			21.5 mg/kg (whole body)[4]	Morphology, NOED				[33]	L; pH was 6.5

Summary of Biological Effects Tissue Concentrations for Pentachlorophenol

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]
			25.1 mg/kg (whole body)[4]	Morphology, NOED				[33]	L; pH was 7.5
			17.8 mg/kg (whole body)[4]	Mortality, NOED				[33]	L; pH was 8.5
			22.1 mg/kg (whole body)[4]	Mortality, NOED				[33]	L; pH was 8.0
			25.1 mg/kg (whole body)[4]	Mortality, NOED				[33]	L; pH was 7.5
			21.5 mg/kg (whole body)[4]	Mortality, NOED				[33]	L; pH was 6.5
			25.1 mg/kg (whole body)[4]	Reproduction, NOED				[33]	L; pH was 7.5
			45.9 mg/kg (whole body)[4]	Reproduction, NOED				[33]	L; pH was 6.5
			69 mg/kg (whole body)[4]	Reproduction, NOED				[33]	L; pH was 8.5
			43.8 mg/kg (whole body)[4]	Reproduction, NOED				[33]	L; pH was 8.0
Ictalurus nebulosus, Brown bullhead		5.7 µg/L	260 µg/kg					[18]	F

Summary of Biological Effects Tissue Concentrations for Pentachlorophenol

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]
Oryzias latipes, Medaka		100 µg/L	41.02 µg/g 38.02 µg/g 37.50 µg/g					[10]	L
Gambusia affinis, Mosquito fish			0.8 mg/kg (whole body)[4]	Mortality, NOED				[28]	L; no effect on survivorship in 24 hours
Osmerus mordax, Rainbow smelt		1.3 µg/L	6 µg/kg					[17]	L
Leuciscus idus, Golden ide			13 mg/kg (whole body)[4]	Mortality, NOED				[24]	L; no effect on survivorship in 3 days
Micropterus salmoides, Largemouth bass			9.6 mg/kg (whole body)[4]	Behavior, LOED				[31]	L; reduced success rate of prey capture
			9.6 mg/kg (whole body)[4]	Growth, LOED				[31]	L; reduction in growth
			9.6 mg/kg (whole body)[4]	Physiological, LOED				[31]	L; reduced food conversion efficiency, condition factor
			10.8 mg/kg (whole body)[4]	Mortality, NOED				[31]	L; no effect on mortality

Summary of Biological Effects Tissue Concentrations for Pentachlorophenol

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]
Perca flavescens, Yellow perch		5.7 µg/L	260 µg/kg					[18]	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 (low molecular weight)

Chemical Name (Common Synonyms): PHENANTHRENE **CASRN:** 85-01-8

Chemical Characteristics

Solubility in Water: 0.6 ± 0.1 mg/L, 22°C [1]

Half-Life: 16-200 days, aerobic soil die-away test [2]

Log K_{ow}: 4.55 [3]

Log K_{oc}: 4.47 L/kg organic carbon

Human Health

Oral RfD: No data [4]

Confidence: ___

Critical Effect: ___

Oral Slope Factor: No data [4]

Carcinogenic Classification: D [4]

Wildlife

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

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

Aquatic Organisms

Partitioning Factors: The water quality criterion tissue level (WQCTL) for phenanthrene, which is calculated by multiplying the water quality chronic value (4.6 µg/L) by the BCF (1380.38), is 6,350 µg/kg [5]. The partitioning between interstitial water and sediment particles increases with sediment aging [6]. The increasing partitioning suggests that phenanthrene becomes more tightly bound with increased contact time. A log BCF of 2.51 was reported for *Daphnia magna* [16].

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

Toxicity/Bioaccumulation Assessment Profile

PAHs are readily metabolized and excreted by fish and invertebrates [11], affecting bioaccumulation kinetics and equilibrium tissue residues. The bioconcentration of phenanthrene by *Hexagenia* was related

to the weight of the mayflies [12]. The bioaccumulation of phenanthrene by three amphipod species was much higher (up to 24 times) for the water-only exposure than for uptake from the sediment [13].

According to Landrum et al. [7], accumulation of sediment-associated PAHs (including phenanthrene) by the amphipod *Diporeia* spp. was limited by both the desorption rate to the interstitial water and the rate of accumulation through ingestion. Because of these limitations the concentration required to produce biological effects (mortality) was approximately 20 times greater than would be predicted using an equilibrium-partitioning approach. Amphipods exposed to 0.08, 0.18, 0.45, and 0.62 µmol/g of phenanthrene accumulated up to 5.8 µmol/g. The highest concentration (0.62 µmol/g of phenanthrene) was slightly toxic to the amphipods (12% mortality in highest concentration). According to the authors the amphipods never reached 6.1 µmol/g in their tissues, the concentration that was required (according to equilibrium-partitioning) to produce toxicity. The results reported by Swartz et al. [8] suggest that phenanthrene at a concentration more than two orders of magnitude higher than the acute concentration measured in the laboratory was not toxic to amphipods. The toxic level of phenanthrene established in the laboratory for the amphipod *Rhepoxynius abronius* was 3.68 mg/kg [9] (10-day LC50 value), while exposure of amphipods to 2,000 mg/kg of phenanthrene in sediment from Eagle Harbor did not produce acute responses. According to McCarty et al. [10], the toxic (critical) body residue of individual PAHs in tissues ranged from 513 to 4,248 mg/kg.

Summary of Biological Effects Tissue Concentrations for Phenanthrene

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,	0.001		0.094					[14]	F
	0.004		0.035						
	0.006		0.007						
	0.023		0.029						
	0.023		0.063						
	0.028		0.046						
	0.042		0.340						
	0.051		0.039						
Crassostrea virginica, Eastern oyster	0.00001		0.00003					[15]	F
	0.00001		0.00013						
	0.00001		0.00017						
	0.00002		0.00015						
	0.00002		0.00010						
	0.00004		0.00020						
	0.00004		0.00018						
	0.00005		0.00029						
	0.00005		0.00022						
	0.00005		0.00025						
	0.00007		0.00022						
	0.00007		0.00009						
	0.00007		0.00022						
	0.00007		0.00018						

Summary of Biological Effects Tissue Concentrations for Phenanthrene

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]**
Crassostrea virginica, Eastern oyster	0.00008		0.0001						
	0.00008		0.0002						
	0.00008		0.0001						
	0.00008		BDL[4]						
	0.00010		0.0003						
	0.00010		0.0006						
	0.00010		0.0002						
	0.00010		0.0001						
	0.00010		0.0001						
	0.00010		0.0001						
	0.00010		0.0002						
	0.00010		0.0001						
	0.0002		0.0002						
	0.0002		0.0002						
	0.0003		0.0004						
	0.0005		0.0001						
	0.0009		0.0001						
Mytilus edulis, Mussel			30.7 mg/kg (whole body)[5]	Physiological, ED50				[22]	L; 50% reduction in feeding rate
Macoma balthica, Baltic macoma	0.001		0.216						
	0.004		0.062						
	0.006		--						
	0.023		0.026						
	0.023		0.027						
	0.028		0.110						
	0.042		0.396						
	0.051								
Daphnia magna, Cladoceran		0.225	73 nM/G		2.51			[16]	L

Summary of Biological Effects Tissue Concentrations for Phenanthrene

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]**
Diporeia sp., Amphipod	0.08		Day 1: 0.2 Day 3: 0.4 Day 8: 0.2 Day 16: 0.1 Day 32: 0.1					[7]	L
	0.18		Day 1: 0.2 Day 3: 0.4 Day 8: 0.2 Day 16: 0.1 Day 32: 0.1						
	0.45		Day 1: 3.2 Day 3: 3.8 Day 8: 1.4 Day 16: 0.6 Day 32: 0.4						
Diporeia sp., Amphipod	0.62		Day 1: 2.2 Day 3: 5.8 Day 8: 2.8 Day 16: 1.2 Day 32: 0.4					[7]	L
Diporeia spp., Amphipod			71 mg/kg (whole body)[5]	Mortality, LOED				[21]	L; 12% mortality

Summary of Biological Effects Tissue Concentrations for Phenanthrene

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]**
Eohaustorius estuarius, Amphipod	0.208	0.014 overlying water	0.225, lipid 5.899, total					[13]	L
		0.14 overlying water	0.719, lipid 17.191, total						
Grandidierella japonica, Amphipod	0.208	0.0140 overlying water	0.096, lipid 1.011, total					[13]	L
		0.140 overlying water	0.938, lipid 10.169, total						
Leptocheirus plumulosus, Amphipod	0.208	0.0140 overlying water	0.073, lipid 0.899, total					[13]	L
		0.140 overlying water	0.360, lipid 3.427, total						
Pontoporeia hoyi, Amphipod	0.0004 0.004	0.006 0.008	0.004 0.007					[18]	L
Fishes									

Summary of Biological Effects Tissue Concentrations for Phenanthrene

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]**
Oncorhynchus mykiss, Rainbow trout			30 mg/kg (whole body)[5]	Physiological, LOED				[24]	L; induction of hepatic mixed function oxidases
Brachydanio rerio, Zebrafish	0.013	0.004	0.013 -24 hours 0.0007 - 240 hours					[20]	L
Leuciscus idus, Golden ide			88 mg/kg (whole body)[5]	Mortality, NOED				[23]	L; no effect on survivorship In 3 days
Pleuronectes vetulus, English sole	0.0009-1.07		0.0005 (liver) <0.00001 (muscle)					[19]	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.
[5] 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): PYRENE **CASRN:** 129-00-0

Chemical Characteristics

Solubility in Water: 0.135 mg/L at 25°C [1]

Half-Life: 210 days - 5.2 yrs based on aerobic soil die-away test data at 10-30°C [2]

Log K_{ow}: 5.11 [3]

Log K_{oc}: 5.02 L/kg organic carbon

Human Health

Oral RfD: 3 x 10^{-2} mg/kg/day [4]

Confidence: Low, uncertainty factor = 3000

Critical Effect: Kidney effects (renal tubular pathology, decreased kidney weights)

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

Carcinogenic Classification: No data [4]

Wildlife

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

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

Aquatic Organisms

Partitioning Factors: Log BCFs for pyrene ranged from 2.85 for midges [6] to 4.05 for guppies [17]. Log BAFs ranged from −0.43 for clams to 4.65 for amphipods [16].

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

Toxicity/Bioaccumulation Assessment Profile

The acute toxicity of hydrocarbons, including pyrene, to both fresh and saltwater 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]. Bioconcentration and depuration of pyrene and its biotransformation products display a clear pH-dependency both in rate and bioconcentration [6]. Decreasing ambient pH leads to decreasing

bioconcentration rates, depuration rates, bioconcentration factors. The accumulation kinetics of pyrene suggest that uptake occurs largely via the sediment interstitial water and is controlled by desorption from sediment particles and dissolved organic matter [7].

Bioavailability of sediment-associated PAHs has been observed to decline with increased contact time [8]. The concentration of pyrene declined significantly over the course of the exposures for all aging durations. Increases in the length of contact between the sediment and pyrene reduced its bioavailability compared to 3 days of aging, but after 60 days, the bioavailability appeared to stabilize. Pyrene exhibited increased partitioning between interstitial water and sediment particles as aging increased [8]. The increasing partitioning suggests that the compounds are becoming more tightly bound with increased contact time.

The results from the laboratory experiments performed by Harkey et al. [9] indicated that accumulation of pyrene from pore-water exposures was lower than accumulation from whole sediment. The concentrations of pyrene in whole sediment and pore water were 0.14-0.87 ng/g and 0.001-0.016 mg/mL, respectively. Harkey et al. [9] concluded that aqueous extracts of whole sediment did not accurately represent the exposure observed in whole sediment. The aqueous extracts of whole sediment underexposed organisms compared to whole sediment, even after adjusting accumulation to the fraction of organic carbon contained in the test media. While the total pyrene concentration in the sediment stayed constant, total concentration decreased appreciably in pore water and elutriate over the course of the exposure, and it is likely that the bioavailability concentrations in these media also decreased. The dissolved organic material in the interstitial waters interfered with the direct uptake of PAHs, e.g., pyrene, in a manner similar to that observed with humic material [10]. Unlike the Aldrich humics that showed a very close relationship between log K_{ow} and log K_b, sorption by dissolved organic carbon from interstitial waters would not necessarily be predicted from K_{ow}. Pyrene was quickly accumulated by *Lumbriculus variegatus* and achieved apparent steady state within 48 to 168 hours [11].

The relative pyrene distribution among sediment particle size revealed 44 percent of pyrene within 43-63 µm particle size [12]. In general, most of pyrene was found in the smallest-sized particles. The narcotic effect for *Diporeia* exposed to pyrene depends on attaining a certain molar concentration in the organism [12]. Using equilibrium-partitioning theory, the BCF value, and critical body residue (LD50), Landrum et al. [12] calculated the sediment concentration that would produce 50 percent amphipod mortality. Based on these assumptions, the pyrene concentration of 14.2 µg/g in sediment should produce 50 percent mortality. The LC50 based on laboratory exposure was estimated to be between 147 and 223 µg/g pyrene. The comparison of the calculated values with the estimated LC50 value (147 to 223 µg/g) from the laboratory experiments suggested that the equilibrium-partitioning approach overestimated the toxicity of sediment-associated pyrene by a factor of 10 at minimum.

Summary of Biological Effects Tissue Concentrations for Pyrene

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 virens, Polychaete	0.006	0.008	0.023-0.031 in 4 days		3.33			[14]	L
Lumbriculus variegatus, Oligochate	0.003	17.327	0.004					[16]	L
		0.000001	0.0002 in 2 days, 0.0003 in 25 days, 0.0004 in 58 days,					[11]	L
		0.0003	0.0015 in 96 h, 0.0014 in 168 h,					[11]	L
		0.0007	0.0019 in 96 h, 0.0020 in 168 h,					[11]	L
		0.001	0.0019 in 96 h, 0.0020 in 168 h,					[11]	L
		0.0013	0.0023 in 96 h, 0.0016 in 168 h,					[11]	L
Dreissena polymorpha, Zebra mussel					4.65			[13]	L; not lipid normalized
Mytilus edulis, Mussel	0.006	0.008	0.022-0.031 in 4 days		3.70			[14]	L

Summary of Biological Effects Tissue Concentrations for Pyrene

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]
Mytilus edulis, Mussel			189 mg/kg (whole body)[4]	Physiological, ED50				[20]	L;50% reduction in feeding rate, exp_conc = >0.04
Macoma nasuta, Clam	0.00006		0.0002			−0.28		[15]	F
	0.00006		0.0002			−0.36		[15]	F
	0.0005		0.0003			−0.43		[15]	F
	0.0006		0.0004			−0.30		[15]	F
	0.0018		0.0009			−0.33		[15]	F
	0.0025		0.0008			−0.37		[15]	F
Diporeia spp., Amphipod			1270 mg/kg (whole body)[4]	Mortality, ED50				[12]	L; 50% mortality
Diporeia spp., Amphipod	0.52		6.8 in 28d, 2.8 in 14d,					[12]	L
	0.86		7.4 in 28d, 4.6 in 14d,	LD50 (critical body residue) was 6.3 and 9.4 µmol/g				[12]	L
	1.11		6.6 in 28d, 4.6 in 14d,	LC50 was between 147 and 223 µg/g (0.72-1.1 µmol/g)				[12]	L

Summary of Biological Effects Tissue Concentrations for Pyrene

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]**
Pontoporeia hoyi, Amphipod	0.0006		0.005					[7]	L
	0.0002	0.02	0.007			4.65		[16]	L
Pontoporeia hoyi, Amphipod	0.0014	0.014	0.015					[16]	L
Chironomus riparius					2.85			[6] [6] [6]	L; at pH of 4 L; at pH of 6 L; at pH of 8
Crangon separenaria, Shrimp	0.006	0.008	0.010-0.011 in 4 days		2.95			[14]	L
Fishes									
Oncorhynchus mykiss, Rainbow trout			30 mg/kg (whole body)[4]	Physiological, LOED				[21]	L; increased hepatic concentration of cytochrome P450

Summary of Biological Effects Tissue Concentrations for Pyrene

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]**
Cyprinus carpio, Common carp			28.7 mg/kg (liver)[4]	Physiological, NA				[19]	L; significant increrase in EROD enzyme and P450 1a protein content
Brachydanio rerio, Zebrafish	0.011	0.088	0.008 -24 hours 0.001 - 240 hours					[18]	L
Poecilia reticulata, Guppy		0.6	0.742		4.05			[17]	L

[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: METAL

Chemical Name (Common Synonyms): SELENIUM **CASRN:** 7782-49-2

Chemical Characteristics

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

Log K_{ow}: – **Log K_{oc}:** –

Human Health

Oral RfD: 5 x 10^{-3} mg/kg/day [2] **Confidence:** High, uncertainty factor = 3

Critical Effect: Clinical selenosis (hair or nail loss, morphological changes of the nails, skin lesions, central nervous system abnormalities including peripheral anesthesia, acroparesthesia, and pain in the extremities, and liver dysfunction indicated by prolongation of blood clotting time and reduced serum glutathione titer)

Oral Slope Factor: Inadequate data [2] **Carcinogenic Classification:** D, selenium sulfide B2 [2]

Wildlife

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

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

Aquatic Organisms

Partitioning Factors: Most of the selenium in sediments is bound to humic and fulvic acids. Microorganisms are closely involved with the selenium cycle and are capable of oxidizing elemental selenium to selenite [6].

Food Chain Multipliers: The results of several studies showed that selenium can biomagnify within the aquatic system [7,8].

Toxicity/Bioaccumulation Assessment Profile

Selenium is an element normally found at low levels in aquatic ecosystems. Although the literature values for acute (600 to 35,000 µg/L) or chronic (30 to 60 µg/L) toxicity via water exposure for fish are a few orders of magnitude higher than its concentration in surface waters, a dietary uptake at relatively low

levels (5 to 10 µg/L) can be toxic to fish. The dietary toxicity was confirmed by Schultz and Hermanutz [1] and Woock et al. [2]. They demonstrated that fish fed with invertebrates containing high levels of selenium developed signs of selenosis and some of them died. Female fish transferred selenium to their progeny, and embryos showed an increased incidence of edema and lordosis. Monitoring concentrations of selenium in sediment and benthic fauna is essential since selenium can biomagnify sufficiently to cause acute toxicity to fishes.

Three species, *Chlorella vulgaris*, *Brachionus calyciflorus*, and *Pimephales promelas* were exposed to selenate for 25 days in a three-trophic level system [10]. Selenium as selenate reduced larval fathead minnow biomass and impared both the algal and rotifer population growth rates at 108.1 µg/L. The results of Dobbs et. al [10] supported the work of earlier researchers [7,8] who found that selenium had a negative impact on aquatic biota at concentrations above 100 µg/L.

Summary of Biological Effects Tissue Concentrations for Selenium

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]**
Plants									
Chlorella vulgaris, Green algae			7.4 mg/kg (whole body)[5]	Growth, LOED				[10]	L; reduced growth
Invertebrates									
Brachionus calyciflorus, Rotifer			15 mg/kg (whole body)[5]	Mortality, ED100				[10]	L; lethal body burden
			6.5 mg/kg (whole body)[5]	Growth, LOED				[10]	L; reduction in population biomass
Daphnia magna, Cladoceran			3 mg/kg (whole body)[5]	Growth, LOED				[15]	L; increased biomass over controls
			25 mg/kg (whole body)[5]	Mortality, LOED				[15]	L; mortality
			2.94 mg/kg (whole body)[5]	Growth, LOED				[16]	L; reduced growth
			10.2 mg/kg (whole body)[5]	Physiological, LOED				[16]	L; decreased whole body chloride concentration
			2.94 mg/kg (whole body)[5]	Physiological, LOED				[16]	L; increased whole body calcium content

Summary of Biological Effects Tissue Concentrations for Selenium

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]
			6.34 mg/kg (whole body)[5]	Reproduction, LOED				[16]	L; delayed time to first brood, decreased intrinsic rate of natural increase
			10.2 mg/kg (whole body)[5]	Growth, NA				[16]	L; reduced growth
			6.34 mg/kg (whole body)[5]	Growth, NA				[16]	L; reduced growth
			6.34 mg/kg (whole body)[5]	Physiological, NA				[16]	L; increased whole body calcium content
			10.2 mg/kg (whole body)[5]	Reproduction, NA				[16]	L; delayed time to first brood, decreased intrinsic rate of natural increase
			4.22 mg/kg (whole body)[5]	Growth, NOED				[16]	L; no effect on growth
			0.26 mg/kg (whole body)[5]	Growth, NOED				[16]	L; no effect on growth
			10.2 mg/kg (whole body)[5]	Mortality, NOED				[16]	L; no effect on mortality
			6.34 mg/kg (whole body)[5]	Mortality, NOED				[16]	L; no effect on mortality
			2.94 mg/kg (whole body)[5]	Mortality, NOED				[16]	L; no effect on mortality

Summary of Biological Effects Tissue Concentrations for Selenium

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]**
			4.22 mg/kg (whole body)[5]	Mortality, NOED				[16]	L; no effect on mortality
			0.26 mg/kg (whole body)[5]	Mortality, NOED				[16]	L; no effect on mortality
			4.22 mg/kg (whole body)[5]	Physiological, NOED				[16]	L; no effect on whole body ions
			0.26 mg/kg (whole body)[5]	Physiological, NOED				[16]	L; no effect on whole body ions
			2.94 mg/kg (whole body)[5]	Reproduction, NOED				[16]	L; no effect on reproduction
			4.22 mg/kg (whole body)[5]	Reproduction, NOED				[16]	L; no effect on reproduction
			0.26 mg/kg (whole body)[5]	Reproduction, NOED				[16]	L; no effect on reproduction
Chironomus decorus, Midge			2 mg/kg (whole body)[5]	Growth, LOED				[12]	L; reduced growth, exp_conc = <1.0
			12.6 mg/kg (whole body)[5]	Mortality, ED50				[19]	L; lethal to 50% of animals in 48 hours
			17 mg/kg (whole body)[5]	Mortality, ED50				[19]	L; lethal to 50% of animals in 48 hours
			0.51 mg/kg (whole body)[5]	Growth, LOED				[20]	L; reduction in growth

Summary of Biological Effects Tissue Concentrations for Selenium

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									
Oncorhynchus tshawytscha, Chinook salmon			0.68 mg/kg (whole body)[5]	Growth, LOED				[5]	L; diet exposure, reduced weight and length gain in 30 days
			0.66 mg/kg (whole body)[5]	Growth, LOED				[5]	L; diet exposure, reduced weight and length gain in 60 days
			2.88 mg/kg (whole body)[5]	Growth, LOED				[5]	L; diet exposure, reduced length after 120 days
			2.01 mg/kg (whole body)[5]	Growth, LOED				[5]	L; diet exposure, reduced weight and length gain in 60 days
			2.16 mg/kg (whole body)[5]	Growth, LOED				[5]	L; diet exposure, reduced weight and length gain in 60 days
			1.6 mg/kg (whole body)[5]	Growth, LOED				[5]	L; diet exposure, reduced weight gain after 120 days
			4.64 mg/kg (whole body)[5]	Growth, LOED				[5]	L; diet exposure, reduced weight and length gain in 120 days in salt water

Summary of Biological Effects Tissue Concentrations for Selenium

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]**
			5.88 mg/kg (whole body)[5]	Mortality, LOED				[5]	L; diet exposure, reduced survival in 60 days
			1.3 mg/kg (whole body)[5]	Mortality, LOED				[5]	L; diet exposure, reduced survival in 90 days
			2.08 mg/kg (whole body)[5]	Mortality, LOED				[5]	L; diet exposure, no effect on survival in 60 days
			0.52 mg/kg (whole body)[5]	Mortality, LOED				[5]	L; diet exposure, no effect on survival in 90 days
			4.68 mg/kg (whole body)[5]	Mortality, LOED				[5]	L; diet exposure, reduced survival in 60 days
			1.08 mg/kg (whole body)[5]	Mortality, LOED				[5]	L; diet exposure, reduced survival in 90 days
			1.02 mg/kg (whole body)[5]	Growth, NOED				[5]	L; diet exposure, no effect on weight or length gain in 30 days
			1.06 mg/kg (whole body)[5]	Growth, NOED				[5]	L; diet exposure, no effect on weight or length gain in 60 days

Summary of Biological Effects Tissue Concentrations for Selenium

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]**
			0.54 mg/kg (whole body)[5]	Growth, NOED				[5]	L; diet exposure, no effect on weight or length gain in 90 days
			1.6 mg/kg (whole body)[5]	Growth, NOED				[5]	L; diet exposure, no effect on length after 120 days
			1.08 mg/kg (whole body)[5]	Growth, NOED				[5]	L; diet exposure, no effect on weight or length gain in 90 days
			0.72 mg/kg (whole body)[5]	Growth, NOED				[5]	L; diet exposure, no effect on weight gain after 120 days
			2.52 mg/kg (whole body)[5]	Growth, NOED				[5]	L; diet exposure, no effect on lenght and weight gain in salt water
			2.66 mg/kg (whole body)[5]	Mortality, NOED				[5]	L; diet exposure, no effect on survival in 60 days
			0.8 mg/kg (whole body)[5]	Mortality, NOED				[5]	L; diet exposure, no effect on survival in 90 days
			5.76 mg/kg (whole body)[5]	Mortality, NOED				[5]	L; diet exposure, no effect on survival in 120 days

Summary of Biological Effects Tissue Concentrations for Selenium

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]**
			4.64 mg/kg (whole body)[5]	Mortality, NOED				[5]	L; diet exposure, no effect on survival in 120 days
Pimephales promelas, Fathead minnow			12.2 mg/kg (whole body)[5]	Growth, LOED				[16]	L; reduction in size and growth of larvae
			10.3 mg/kg (whole body)[5]	Growth, NOED				[14]	L; no effect on larval growth
			12.2 mg/kg (whole body)[5]	Mortality, NOED				[14]	L; no effect on mortality
			15.2 mg/kg (whole body)[5]	Growth, LOED				[10]	L; reduced growth of larvae
			17.8 mg/kg (whole body)[5]	Mortality, LOED				[10]	L; mortality, loss of weight
Lepomis macrochirus, Bluegill			2.4 mg/kg (whole body)[5]	Mortality, NOED				[13]	L; no effect on mortality
			15.8 mg/kg (liver)[5]	Mortality, LOED				[7]	L
			2.8 mg/kg (skeletal muscle)[5]	Mortality, LOED				[7]	L
			6.3 mg/kg (testis)[5]	Mortality, LOED				[7]	L

Summary of Biological Effects Tissue Concentrations for Selenium

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]**
			4.6 mg/kg (whole body)[5]	Mortality, LOED				[7]	L
			4.6 mg/kg (whole body)[5]	Growth, NA				[7]	L
			4.6 mg/kg (whole body)[5]	Reproduction, NA				[7]	L; measurable but not statistically significant reduced survival of embryos and larvae
			0.4 mg/kg (brain)[5]	Mortality, NOED				[17]	L; no effect on survivorship
			8.3 mg/kg (gill)[5]	Mortality, NOED				[17]	L; no effect on survivorship
			1.8 mg/kg (gonad)[5]	Mortality, NOED				[17]	L; no effect on survivorship
			13.7 mg/kg (heart)[5]	Mortality, NOED				[17]	L; no effect on survivorship
			2.2 mg/kg (intestine)[5]	Mortality, NOED				[17]	L; no effect on survivorship
			10.2 mg/kg (kidney)[5]	Mortality, NOED				[17]	L; no effect on survivorship
			11.4 mg/kg (liver)[5]	Mortality, NOED				[17]	L; no effect on survivorship
			2.4 mg/kg (plasma)[5]	Mortality, NOED				[17]	L; no effect on survivorship

Summary of Biological Effects Tissue Concentrations for Selenium

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]**
			7.2 mg/kg (red blood cells)[5]	Mortality, NOED				[17]	L; no effect on survivorship
			17.7 mg/kg (spleen)[5]	Mortality, NOED				[17]	L; no effect on survivorship
			1 mg/kg (stomach)[5]	Mortality, NOED				[17]	L; no effect on survivorship
			2.6 mg/kg (white muscle)[5]	Mortality, NOED				[17]	L; no effect on survivorship
			4.3 mg/kg (whole body)[5]	Mortality, NOED				[17]	L; no effect on survivorship
			1.6 mg/kg (whole body)[5]	Cellular, LOED				[18]	L; structural changes in gill tissue
			1.6 mg/kg (whole body)[5]	Mortality, LOED				[18]	L; 35% reduction in survival after 180 days
			1.6 mg/kg (whole body)[5]	Physiological, LOED				[18]	L; increased respiratory demands, lipid depletion
			1.6 mg/kg (whole body)[5]	Behavior, NOED				[18]	L; no effect on feeding behavior

Summary of Biological Effects Tissue Concentrations for Selenium

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		0.16 mg/L	Day 30:	Mortality:				[6]	L
		0.33 mg/L	3.0 µg/g	10%					
		0.64 mg/L	3.5 µg/g	20%					
		1.12 mg/L	4.0 µg/g	40%					
		2.80 mg/L	7.0 µg/g	55%					
			14.3 µg/g	88%					
		0.16 mg/L	Day 60:						
		0.33 mg/L	2.8 µg/g	10%					
		0.64 mg/L	4.1 µg/g	22%					
		1.12 mg/L	5.0 µg/g	52%					
		2.80 mg/L	9.7 µg/g	70%					
			–	98%					
			Day 258:					[7]	L
		10 µg/L	9.3 µg/g (liver)						
			4.4 µg/g (ovaries)						
			3.0 µg/g (testes)						
			1.8 µg/g (muscles)						
			Day 356:						
			7.3 µg/g (liver)						
			4.5 µg/g (ovaries)						
			7.6 µg/g (testes)						
			4.2 µg/g (muscles)						

Summary of Biological Effects Tissue Concentrations for Selenium

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			8.4 µg/L water, 0.8 µg/g diet: 3 µg/g 1 µg/g					[8]	L
			10.5 µg/L water, 4.6 µg/g diet: 3 µg/g						
			10.5 µg/L water, 8.4 µg/g diet: 5 µg/g						
			10.1 µg/L water, 16.8 µg/g diet: 10 µg/g						
			11.0 µg/L water, 33.3 µg/g diet: 19 µg/g						
Micropterus salmoides, Largemouth bass			0.4 mg/kg (brain)[5]	Mortality, NOED				[17]	L; no effect on survivorship
			6.2 mg/kg (gill)[5]	Mortality, NOED				[17]	L; no effect on survivorship
			1.7 mg/kg (gonad)[5]	Mortality, NOED				[17]	L; no effect on survivorship
			12 mg/kg (heart)[5]	Mortality, NOED				[17]	L; no effect on survivorship
			2.1 mg/kg (intestine)[5]	Mortality, NOED				[17]	L; no effect on survivorship

Summary of Biological Effects Tissue Concentrations for Selenium

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]**
			8.6 mg/kg (kidney)[5]	Mortality, NOED				[17]	L; no effect on survivorship
			10 mg/kg (liver)[5]	Mortality, NOED				[17]	L; no effect on survivorship
			3.2 mg/kg (plasma)[5]	Mortality, NOED				[17]	L; no effect on survivorship
			8 mg/kg (red blood cells)[5]	Mortality, NOED				[17]	L; no effect on survivorship
			16.4 mg/kg (spleen)[5]	Mortality, NOED				[17]	L; no effect on survivorship
			1.3 mg/kg (stomach)[5]	Mortality, NOED				[17]	L; no effect on survivorship
			1.4 mg/kg (white muscle)[5]	Mortality, NOED				[17]	L; no effect on survivorship
			3 mg/kg (whole body)[5]	Mortality, NOED				[17]	L; no effect on survivorship

Summary of Biological Effects Tissue Concentrations for Selenium

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]**
Wildlife									
Anas platyrhynochos, Mallard			0 ppm diet: 2.5 ppm (liver) 15 ppm diet: 2.0 ppm (liver) 0/100 ppm diet: 35.0 ppm (liver) 15/100 ppm diet: 53.0 ppm (liver)					[9]	L
			0 ppm diet: 0.88 ppm (liver) females 0.69 ppm, males 1.1 ppm, 3.5 ppm diet: 3.7 ppm (liver) females 3.2 ppm, males 4.3 ppm, 7.0 ppm diet: 6.2 ppm (liver) females 5.1 ppm, males 7.3 ppm					[11]	L

[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.
[5] 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: METAL

Chemical Name (Common Synonyms): SILVER **CASRN:** 7440-22-4

Chemical Characteristics

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

Log K_{ow}: – **Log K_{oc}:** –

Human Health

Oral RfD: 5 x 10^{-3} mg/kg/day [2] **Confidence:** Low, uncertainty factor = 3

Critical Effect: Argyria—permanent, but benign, bluish-gray discoloration of the skin

Oral Slope Factor: No data [2] **Carcinogenic Classification:** D [2]

Wildlife

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

Food Chain Multipliers: Food chain mulitpliers for silver in wildlife were not found in the literature.

Aquatic Organisms

Partitioning Factors: Silver in the water column can partition to dissolved and particulate organic carbon. Important issues related to water column concentrations of silver are water hardness (i.e., calcium concentration), pH, and metal speciation, since the monovalent form of silver is believed to be responsible for observed biological effects. In addition, silver is known to form a variety of relatively insoluble (i.e., nonbioavailable) complexes, including silver sulfides formed with acid volatile sulfides, that can be important in controlling the toxicity and bioaccumulation of silver in sediments [8 and 9].

Food Chain Multipliers: Little evidence exists to support the general occurrence of biomagnification of silver within marine or freshwater food webs [3]. Silver uptake by aquatic organisms appears to be almost entirely from the dissolved form. When silver was bound to algal cell membranes, it could not be dislodged by either mechanical disruption or leaching at low pH; therefore, silver bound to algal cells is likely unassimilable by higher organisms [3].

Toxicity/Bioaccumulation Assessment Profile

Silver does not appear to be a highly mobile element under typical conditions in most aquatic habitats. Tissue residue-toxicity relationships can also vary because organisms may sequester metal in different forms that might be analytically measurable as tissue residue, but might actually be stored in unavailable forms within the organism as a form of detoxification [4]. Whole-body residues also might not be indicative of effects concentrations at the organ level because concentrations in target organs, such as the kidneys and liver, can be 20 times greater than whole body residues [5]. The application of "clean" chemical analytical and sample preparation techniques is also critical in the measurement of metal tissue residues [6]. Exposure of rainbow trout to three different silver salts revealed that silver, introduced as silver nitrate, was 15,000 and 11,000 times more toxic than silver chloride and silver thiosulfate [11]. However, all three forms of dissolved silver were taken up by rainbow trout and accumulated in the tissue. Interestingly, extremely high levels of silver were found in livers of fish exposed to silver as silver chloride and silver thiosulfate. Hogstrand et al. [11] attributed low toxicity to these two forms to production of metallothionein, a small cysteine-rich, intracellular protein that avidly binds most metals.

Summary of Biological Effects Tissue Concentrations for Silver

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									
Busycotypus canaliculatum, Channeled whelk		0.1-0.5 µg/L	1.1 µg/g					[7]	F
Corbicula fluminea, Asiatic clam			1,650 mg/kg (whole body)[4]	Growth, LOED				[8]	L; reduction in growth
			800 mg/kg (whole body)[4]	Growth, NOED				[8]	L; no effect on growth
			2,510 mg/kg (whole body)[4]	Mortality, LOED				[8]	L; reduced survival
			1,650 mg/kg (whole body)[4]	Mortality, NOED				[8]	L; no effect on survival
Mytilus edulis, Mussel			3.7 mg/kg (whole body)[4]	Physiological, LOED				[12]	L; significantly increased oxygen consumption at lowest test concentration at 25 ppt salinity, number of replicates is 12 to 20
Crassostrea virginica, Eastern oyster		2 µg/L 5 µg/L 7 µg/L	2.6 µg/g 6.5 µg/g 4.8 µg/g					[9]	L

Summary of Biological Effects Tissue Concentrations for Silver

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]**
Crassostrea virginica, Oyster			38 mg/kg (gill)[4]	Physiological, LOED				[12]	L; Significantly increased oxygen consumption at lowest test concentration at 25 ppt salinity, number of replicates is 12 to 20
			12.4 mg/kg (whole body)[4]	Physiological, LOED				[12]	
Mercenaria mercenaria, Quahog clam			7.6 mg/kg (gill)[4]	Physiological, LOED				[12]	L; significantly increased oxygen consumption at lowest test concentration at 25 ppt salinity, number of replicates is 12 to 20
			0.8 mg/kg (whole body)[4]	Physiological, LOED				[12]	
Mya arenaria, Soft shell clam			10.4 mg/kg (whole body)[4]	Physiological, LOED				[12]	L; significantly increased oxygen consumption at lowest test concentration at 25 ppt salinity, number of replicates is 12 to 20

Summary of Biological Effects Tissue Concentrations for Silver

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]**
Homarus americanus, American lobster		0.1-0.5 µg/L	2.3 µg/g					[7]	F
Fishes									
Oncorhyncus mykiss, Rainbow trout		4.3 µg/L	16 µg/g (liver), 4 µg/g (gills)					[11]	L
		7.2 µg/L	13 µg/g (liver) 4 µg/g (gills)						
		9.3 µg/L	20 µg/g (liver) 4.8 µg/g (gills)						
Salmo trutta, Brown trout			1343 Bq/g in food, Day 7: 17.6 Bq/g 269 Bq/g in food, Day 13: 18.5 Bq/g 296 Bq/g in food, Day 20: 21.7 Bq/g						
Salmo trutta, Brown trout			289 Bq/g in food, Day 26: 26.6 Bq/g 273 Bq/g in food, Day 33: 27.4 Bq/g					[10]	L

Summary of Biological Effects Tissue Concentrations for Silver

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]
			623.8 Bq/g (liver) 24.9 Bq/g (kidneys) 25.5 Bq/g (viscera) 5.5 Bq/g (gills) 23.9 Bq/g (digestive tract) 3.2 Bq/g (muscle) 4.4 Bq/g (bone) 2.9 Bq/g (head) 7.2 Bq/g (skin)					[10]	L

[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: METAL

Chemical Name (Common Synonyms): TRIBUTYLTIN **CASRN:** 688-73-3

Tributyltin compounds, such as those used in antifouling paints, consist of a tin (Sn) atom covalently bonded to three butyl (C_4H_9-) moieties and an associated anion (X). A number of organotin compounds have been used as ingredients in paints, pesticides, and preservatives, including trialkyltins (e.g., bis(tributyltin) oxide (TBTO), bis(tributyltin) sulfide, tributyltin acetate, tributyltin fluoride, tributyltin naphthenate, and tributyltin resinate), triaryltins (e.g., triphenyltin hydroxide), dialkyltins (e.g., (TBTFl) dibutyltin dilaurte, dibutyltin isooctylmercaptonacetate, and dibutyltin maleate), and monooctyltins (e.g., monooctyltin tris isooctyl mercaptoacetate). In aquatic systems, the distribution of TBT species is dependent on pH and salinity. In seawater, the hydrated TBT cation, tributyltin chloride, (TBTCl) bis(tributyltin carbonate), and tributyltin hydroxide are in equilibrium. It is widely accepted that tributyltin toxicity is ascribed to the cation (TBT^{++}) and not to which anion is associated with the biocide in the neutral compound. Researchers have been inconsistent and at times ambiguous in reporting concentrations of organotins and in their use of units in the literature [1]. The following discussion is based on the tributyltin cation (TBT^{++}) and not the various species. The table summarizing biological effects contains data for the tributyltin cation, as well as for tributyltin chloride, tributyltin fluoride, tributyltin oxide, and tin. The table identifies the chemical species measured, if the information was available in the original document reviewed.

Chemical Characteristics

Solubility in Water: <1 to >200 mg/L [2]

Half-Life: Sediments: >20 months [3]

Log K_{ow}: 2.2 - 4.4 [2]

Log K_{oc}: 4.36 - 5.02 [4]

Human Health

Oral RfD: 3×10^{-5} mg/kg/day [5]

Confidence: Low, uncertainty factor = 1000

Critical Effect: Immunotoxicity in rats

Oral Slope Factor (Reference): No data [5]

Carcinogenic Classification: No data [5]

Wildlife

Partitioning Factors: Laboratory studies have demonstrated accumulation of TBT in mice and rats, and butyltin residues were detected recently in the blubber of a number of marine mammal species [2]. However, accurate determination of partitioning factors for TBT in wildlife is difficult because this compound is rapidly metabolized once it has been taken up by vertebrates. No partitioning factors were identified for wildlife in the studies reviewed.

Food Chain Multipliers: Biomagnification of butyltins in aquatic systems does not occur, or if it does, only to a minor extent [2].

Aquatic Organisms

Partitioning Factors: Uptake of TBT from sediment to tissues is a complex, non-linear process, and may be better approximated by a power function [6]. Uptake and elimination rates vary considerably by species [4] and the bioavailability of sediment-associated TBT is controlled by a wide range of parameters (eg., chemical speciation, pH, organic content), further moderating uptake rates [2,6]. Attempts to derive BSAFs with wide-ranging utility are also hampered by the fact that tissues burdens in aquatic animals have traditionally been correlated with TBT concentrations in the water column, rather than sediment concentrations.

Once TBT has been incorporated, it tends to partition into multiple tissue compartments. Log BCFs ranged from 2.70 in carp muscle [7] to 2.32-2.74 in whole rainbow trout [8] and 3.26 in muscle tissue, 3.66 in viscera, and 3.41 in whole body residues of sheepshead minnow [9]. Tsuda et al. [7] found that BCFs for carp were highest in kidney, followed by gall bladder, liver, and muscle, in that order. In rainbow trout, BCFs for TBT were highest for peritoneal fat, followed by kidney, liver, and gall bladder. As with wildlife, TBT can be rapidly metabolized by many aquatic organisms. The rapid metabolism of TBT possibly explains why apparent uptake rates in bivalves, whose enzyme systems metabolize butyltins at a much slower rate, are typically higher than in other organisms [2]. Seasonal variability has been reported for the eastern oyster *Crassostrea gigas*. The lowest proportion of TBT in tissues was found in the summer months and associated with either higher biodegradation rates of TBT in the water column or higher biotransformation rates in oyster tissues [10]. In the studies reviewed, Log BCF's for marine bivalves range from 4.09 to 5.10 The highest log BCF identified was for the zebra mussel (*Dreissena polymorpha*) at 5.95 Reported log BCFs for polychaete worms are approximately 3.85.

Food Chain Multipliers: Biomagnification of TBT does not appear to be significant in aquatic systems. Although TBT is accumulated or concentrated to a very high degree in lower trophic level organisms, dietary uptake in higher trophic level organisms appears to be counteracted by biotransformation in the liver [2].

Toxicity/Bioaccumulation Assessment Profile

Tri-substituted organotins (such as tributyltin) are most commonly used as pesticides in commercial and agricultural applications. Tributyltin (TBT) is widely used as a preservative for timber and wood, textiles, paper, and leather [2]. The use of marine paints containing TBT compounds as toxic additives has been found to be very effective in eliminating fouling problems [11]. TBT-based antifouling paints typically contain up to 20 percent by weight of a suitable tributyl or triphenyltin toxicant which is slowly leached into the surrounding water in the immediate vicinity of the hull. The active lifetime of these paints is usually 1-2 years, after which time the vessel must be repainted [12].

The toxicity of organotins increases with progressive introduction of organic groups at the tin atom [2]. Thus, the high toxicity of TBT led to its use as a fungicide, bactericide, and algicide. TBT-containing antifouling paints were recognized as up to 100 times more effective than copper-based antifouling paints [10]. In fact, studies have demonstrated that TBT is deleterious at concentrations far lower than those indicated for other marine pollutants [13]. Consequently TBT has been used in antifouling paints since the early 1960s and gained widespread application on all types of vessels in the 1970s and 1980s [2]. Shell thickening in oysters (*Crassostrea gigas*) has been reported in some areas of France since

the outset of its introduction in that country in 1968 [14]. TBT leaching from the ship hulls into the water appeared to be the major pathway of entry into the aquatic environment [2]. Other sources of TBT in the aqueous environment include releases of fugitive paint and paint chips from vessel repair and dry-dock facilities [15]. TBT is likely to partition between suspended particles in the water column and sediments, although up to 99 percent of the TBT may reside in the sediments. TBT-contaminated sediments can represent a substantial source of organotin to aquatic receptors [16]. TBT has a significant lipid solubility and thus a high affinity for bioaccumulation [17]. Some organisms, including fishes, crustaceans, bivalves, and microorganisms, have the ability to bioconcentrate TBT to concentrations which are orders of magnitude higher than the exposure concentration [13].

Acute effects of TBT have been observed in the water column at TBT concentrations of 1 ng/L. This concentration has been associated with reduced reproduction in snails [17]. Histological alterations were observed in young European minnows exposed to an aqueous TBT concentration of 0.8 µg/L [17]. Reduced growth was noted in long-term exposures of rainbow trout yolk sac fry to 0.2 µg/L TBT, resulting in an estimated NOEC of 0.04 µg/L [17]. Immunotoxic effects were observed in the guppy at 0.32 µg/L TBT. In studies of *Acartia tonsa*, reductions in survival in acute tests were observed at 0.029 µg/L; NOECs and LOECs for survival during chronic tests were 0.024 and 0.017 µg/L, respectively [18].

As a group, molluscs are among the most sensitive to TBT. Gastropod snails exhibit anatomical abnormalities referred to as imposex, the superimposition of male characteristics onto a normal female reproductive system [19]. Growth in oyster spat is inhibited at aqueous concentrations of 0.15 µg/L and shell thickening has been reported at 0.2 µg/L. Other effects in oysters include abnormal veliger development, malformation of trocophores, larval anomalies, perturbation in food assimilation, and high mortality [20]. Some freshwater and marine bivalves are able to tolerate short-term TBT exposure due to their ability to isolate themselves from the irritating environment by closing their valves.

TBT concentrations in sediments can be from one to several thousand times higher than concentrations found in the overlying water [21]. Bivalve populations can be completely eliminated when sediment TBT concentrations exceed 0.8 µg/g [17]. No sediment criteria exist for TBT, and ER-L and ER-M ranges are unavailable. However, studies indicate that mollusks respond to sediment concentrations of TBT as low as 10 ng/g, while some copepod crustaceans, echinoderms, polychaetes, tunicates, phytoplankton, and fish respond to sediment TBT concentrations between 10 and 100 ng/g [21].

Summary of Biological Effects Tissue Concentrations for Tributyltin

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	**Sediment**	**Water**	**Tissue (SampleType)**	**Effects**	**Log BCF**	**Log BAF**	**BSAF**	**Reference**	**Comments[3]**
Invertebrates									
Nereis diversicolor, Polychaete	445 ± 83 ng/g dw (n=5)	68.2 ± 40.6 ng/L[3] (n=8)	479 ± 249 ng/g dw[†] (pooled, whole body) (n=5)		3.85			[21]	F
Neanthes arenaceodentata, Polychaete		100 ng/L[3]	6.27µg/g dw TBT[++] (whole body)	Reduced growth and reproduction				[23]	L[5]
		50 ng/L[3]	<3.0 µg/g dw TBT[++] (whole body)	No significant effect on survival, growth, or reproduction				[23]	L[5]
		500 ng/L[4]	16.81 µg/g dw TBT[++] (whole body)	Significant effect on survival				[23]	L[5]
Littorina littorea, Gastropod mollusk (Common winkle)	445±83 ng dw (n = 5)	68.2± 40.6 ng/L (n = 8)	1,009±428 ng/g dw (pooled, soft tissue) (n = 4)		4.17			[21]	F

Summary of Biological Effects Tissue Concentrations for Tributyltin

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	**Sediment**	**Water**	**Tissue (SampleType)**	**Effects**	**Log BCF**	**Log BAF**	**BSAF**	**Reference**	**Comments[3]**
Littorina littorea, Periwinkle			0.1 mg/kg TBTCl (whole body)[8]	Reproduction, NOED				[46]	L and F combined; imposex - intersex response (prostate length, isi); estimated wet weight
Marisa cornuarietis, Freshwater gastropod (Ramshorn snail)		50 ng Sn/L[4]	≈800 µg Sn/g dw (soft tissue)	VDS index constant at stage 1	4.23[4]			[32]	L; equilibrium reached after 3 to 4 months; females accumulate more than males
Marisa cornuarietis, Freshwater gastropod (Ramshorn snail)		200 ng Sn/L[4]	≈1600 µg Sn/g dw (soft tissue)	VDS index increased from stage 1 to stage 3	4.96[4]			[32]	L; equilibrium reached after 3 to 4 months; females accumulate more than males
Ilyanassa obsoleta, Mud snail		20 ng/L[4]	620 ng/g dw[†] (soft tissue)	100% occurrence of imposex in females				[35]	F
Nucella lapillus, Dog welk		18.7 ng/L[4]	♂: 1,475 ng Sn/g dw ♀: 1,864 ng Sn/g dw (soft tissue)	Imposex	♂: 77,900 ♀: 99,700			[36]	F

Summary of Biological Effects Tissue Concentrations for Tributyltin

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (SampleType)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
		107 ng/L[4]	♂: 2,436 ng Sn/g dw ♀: 3,498 ng Sn/g dw (soft tissue)	Sterilization (♀)	♂: 22,800 ♀: 32,700			[36]	F
			0.1 mg/kg (whole body)	Induction of imposex				[37]	L
			0.1 µg Sn/g dw (soft tissue)	Normal breeding occurs				[38]	L
		0.25 ng Sn/L[4]	0.025 µg Sn/g dw (soft tissue)	Stage 1 (infolding of pallian cavity floor) Imposex				[38]	L
		1-2 ng Sn/L[4]	0.238 - 0.239 µg Sn/g dw (soft tissue)	Relative Penis Size (RPS) = 48%; Vas Deferens Sequence (VDS) = Stage 4.4 (breeding not impaired)				[38]	L
		3-5 ng Sn/L[4]	0.602 - 0.569 µg Sn/g dw (soft tissue)	RPS = 96.6%; VDS = Stage 5.1 (breeding impaired)				[38]	L
		20 ng Sn/L[4]	1.464 - 1.696 µg Sn/g dw (soft tissue)	RPS = 109%; VDS = Stage 5.0 (breeding impaired)				[38]	L

Summary of Biological Effects Tissue Concentrations for Tributyltin

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	**Sediment**	**Water**	**Tissue (SampleType)**	**Effects**	**Log BCF**	**Log BAF**	**BSAF**	**Reference**	**Comments[3]**
Nucella lapillus, Dog welk		100 ng Sn/L[4]	2.520 - 3.164 µg Sn/g dw (soft tissue)	RPS = 90.4%; VDS = Stage 5.0 (breeding impaired)				[38]	L
		<0.5 ng Sn/L[4]	0.039 - 0.092 µg Sn/g dw (soft tissue)	RPS = 3.7%; VDS = Stage 3.2 (breeding not impaired)				[38]	F
Nucella lapillus, Dog whelk			2 mg/kg TBT ion and DBT ion (whole body)[8]	Development, NA				[36]	L and F combined; paint on shell; female penis length increased; body burden as tin not TBT or DBT
			1.97 mg/kg TBT ion and DBT ion (whole body)[8]	Development, NOED				[36]	L and F combined; paint on shell; no effect in male penis length; body burden as tin not TBT or DBT

Summary of Biological Effects Tissue Concentrations for Tributyltin

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (SampleType)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
			0.0413 mg/kg TBTCl (whole body)[8]	Development, LOED				[36]	L and F combined; relative penis size significantly decreased (female/male penis length); body burden as tin not TBT or DBT
			1.17 mg/kg TBTCl (whole body)[8]	Reproduction, LOED				[36]	L and F combined; sterility in females; body burden as tin not TBT or DBT
			0.733 mg/kg TBTCl (whole body)[8]	Reproduction, LOED				[36]	L and F combined; sterility in females; body burden as tin not TBT or DBT
			1.82 mg/kg TBTCl (whole body)[8]	Development, NA				[36]	L and F combined; paint on shell; no effect in male penis length; body burden as tin not TBT or DBT

Summary of Biological Effects Tissue Concentrations for Tributyltin

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	**Sediment**	**Water**	**Tissue (SampleType)**	**Effects**	**Log BCF**	**Log BAF**	**BSAF**	**Reference**	**Comments[3]**
			1.33 mg/kg TBTCl (whole body)[8]	Development, NA				[36]	L and F combined; paint on shell; female penis length increased; body burden as tin not TBT or DBT
			0.909 mg/kg TBTCl (whole body)[8]	Development, NOED				[36]	L and F combined; paint on shell; no effect in male penis length; body burden as tin not TBT or DBT
			0.0666 mg/kg TBTCl (whole body)[8]	Development, NOED				[36]	L and F combined; no increase in penis length; equals 0.5 ug/g TBT+DBT; body burden as tin not TBT or DBT
Thais clavigera, Whelk			0.013 mg/kg TBTCl (whole body)[8]	Reproduction, LOED				[50]	L; degradation products present
Mytilus edulis, Blue mussel			0.019 - 0.047 µg/g[†] (pooled, soft tissue)	Reduced growth				[24]	F; 82-day exposure

Summary of Biological Effects Tissue Concentrations for Tributyltin

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	**Sediment**	**Water**	**Tissue (SampleType)**	**Effects**	**Log BCF**	**Log BAF**	**BSAF**	**Reference**	**Comments[3]**
			2.0 µg/g dw† (soft tissue)	Threshold for reduced scope for growth				[25]	F; other contaminants present
			4 µg/g dw† (soft tissue)	Severe inhibition of growth, significantly reduced feeding rate, threshold concentration				[26]	L[5]
			1.5 mg/kg† (soft tissue)	Threshold for growth rate inhibition				[27]	F; 84-day exposure
			2.20 mg/kg TBTO (soft tissue)	Reduced growth in spat				[28]	L; 45-day exposure
		200 ng/L[4]	1.5 µg/g† (whole body)	Reduced growth				[29]	F[6]
	0.08 µg/g dw	15 ±8 ng/L[4]	0.64 µg/g † (soft tissue)					[30]	F
Mytilus edulis, Blue mussel	0.03 µg/g dw	33 ±27 ng/L[4]	0.75 µg/g† (soft tissue)					[30]	F
	0.02 µg/g dw	21 ±8 ng/L[4]	0.34 µg/g† (soft tissue)					[30]	F
	0.10 µg/g dw	13 ng/L[4]	0.16 µg/g† (soft tissue)					[30]	F
	0.15 µg/g dw	22 ±12 ng/L[4]	0.66 µg/g† (soft tissue)					[30]	F
	0.04 µg/g dw	17 ±12 ng/L[4]	0.44 µg/g† (soft tissue)					[30]	F

Summary of Biological Effects Tissue Concentrations for Tributyltin

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	**Sediment**	**Water**	**Tissue (SampleType)**	**Effects**	**Log BCF**	**Log BAF**	**BSAF**	**Reference**	**Comments[3]**
	0.08 µg/g dw	13 ng/L[4]	0.30 µg/g† (soft tissue)					[30]	F
	0.05 µg/g dw	8 ng/L[4]	0.15 µg/g† (soft tissue)					[30]	F
	0.04 µg/g dw	35 ±17 ng/L[4]	1.01 µg/g† (soft tissue)					[30]	F
	0.11 µg/g dw	17 ±9 ng/L[4]	0.61 µg/g† (soft tissue)					[30]	F
	0.07 µg/g dw	22 ±14 ng/L[4]	0.46 µg/g† (soft tissue)					[30]	F
	0.04 µg/g dw	8 ±2 ng/L[4]	0.29 µg/g† (soft tissue)					[30]	F
	0.36 µg/g dw	45 ±17 ng/L[4]	0.98 µg/g† (soft tissue)					[30]	F
	0.15 µg/g dw	31 ±18 ng/L[4]	1.04 µg/g† (soft tissue)					[30]	F
	0.31 µg/g dw	23 ±18 ng/L[4]	0.38 µg/g† (soft tissue)					[30]	F
Mytilus edulis, Blue mussel	0.10 µg/g dw	11 ±4 ng/L[4]	0.29 µg/g† (soft tissue)					[30]	F
	0.07 µg/g dw	26 ±9 ng/L[4]	0.75 µg/g† (soft tissue)					[30]	F
	0.05 µg/g dw	22 ±15 ng/L[4]	0.47 µg/g† (soft tissue)					[30]	F
	0.27 µg/g dw	13 ±5 ng/L[4]	0.27 µg/g† (soft tissue)					[30]	F
	0.07 µg/g dw	8 ng/L[4]	0.17 µg/g† (soft tissue)					[30]	F

Summary of Biological Effects Tissue Concentrations for Tributyltin

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	**Sediment**	**Water**	**Tissue (SampleType)**	**Effects**	**Log BCF**	**Log BAF**	**BSAF**	**Reference**	**Comments[3]**
	0.05 µg/g dw	26 ±12 ng/L[4]	0.45 µg/g† (soft tissue)					[30]	F
	0.02 µg/g dw	18 ±13 ng/L[4]	0.41 µg/g† (soft tissue)					[30]	F
	0.02 µg/g dw	15 ±12 ng/L[4]	0.19 µg/g† (soft tissue)					[30]	F
	0.04 µg/g dw	8 ±2 ng/L[4]	0.12 µg/g† (soft tissue)					[30]	F
	<0.01 µg/g dw	11 ng/L[4]	0.30 µg/g† (soft tissue)					[30]	F
	0.01 µg/g dw	23 ±23 ng/L[4]	0.35 µg/g† (soft tissue)					[30]	F
	0.02 µg/g dw	3 ±2 ng/L[4]	0.07 µg/g† (soft tissue)					[30]	F
	0.01 µg/g dw	16 ng/L[4]	0.17 µg/g† (soft tissue)					[30]	F
	0.01 µg/g dw	6 ±5 ng/L[4]	0.11 µg/g† (soft tissue)					[30]	F
Mytilus edulis, Blue mussel	0.05 µg/g dw	6 ±5 ng/L[4]	0.11 µg/g† (soft tissue)					[30]	F
	<0.01 µg/g dw	2 ng/L[4]	0.05 µg/g† (soft tissue)					[30]	F
	0.66 µg/g dw	38 ±21 ng/L[4]	1.06 µg/g† (soft tissue)					[30]	F
	0.26 µg/g dw	366 ±29 ng/L[4]	0.82 µg/g† (soft tissue)					[30]	F
	0.15 µg/g dw	76 ±43 ng/L[4]	0.32 µg/g† (soft tissue)					[30]	F

Summary of Biological Effects Tissue Concentrations for Tributyltin

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	**Sediment**	**Water**	**Tissue (SampleType)**	**Effects**	**Log BCF**	**Log BAF**	**BSAF**	**Reference**	**Comments[3]**
	0.53 µg/g dw	25 ng/L[4]	0.35 µg/g† (soft tissue)					[30]	F
	0.08 µg/g dw	38 ±33 ng/L[4]	0.60 µg/g† (soft tissue)					[30]	F
	0.19 µg/g dw	13 ±4 ng/L[4]	0.41 µg/g† (soft tissue)					[30]	F
	0.17 µg/g dw	13 ±5 ng/L[4]	0.20 µg/g† (soft tissue)					[30]	F
	0.07 µg/g dw	8 ±3 ng/L[4]	0.11 µg/g† (soft tissue)					[30]	F
	0.06 µg/g dw	15 ±6 ng/L[4]	0.93 µg/g† (soft tissue)					[30]	F
	0.03 µg/g dw	11 ±6 ng/L[4]	0.58 µg/g† (soft tissue)					[30]	F
	0.04 µg/g dw	5 ±2 ng/L[4]	0.10 µg/g† (soft tissue)					[30]	F
	0.05 µg/g dw	12 ±10 ng/L[4]	0.33 µg/g† (soft tissue)					[30]	F
Mytilus edulis, Blue mussel	0.02 µg/g dw	7 ±6 ng/L[4]	0.23 µg/g† (soft tissue)					[30]	F
	0.03 µg/g dw	6 ±2 ng/L[4]	0.09 µg/g† (soft tissue)					[30]	F
	0.02 µg/g dw	6 ±4 ng/L[4]	0.07 µg/g† (soft tissue)					[30]	F
	4.6 µg/g dw	93 ±45 ng/L[4]	2.57 µg/g† (soft tissue)					[30]	F
	10.8 µg/g dw	1090 ±1850 ng/L[4]	3.22 µg/g† (soft tissue)					[30]	F

Summary of Biological Effects Tissue Concentrations for Tributyltin

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	**Sediment**	**Water**	**Tissue (SampleType)**	**Effects**	**Log BCF**	**Log BAF**	**BSAF**	**Reference**	**Comments[3]**
	0.23 µg/g dw	25 ±7 ng/L[4]	0.81 µg/g[†] (soft tissue)					[30]	F
Mytilus edulis, Mussel			2.58 mg/kg TBTCl (whole body)[8]	Physiological, LOED				[53]	L; significant increase in anoxic heat dissipation (j/h/g) at test concentration
			2.58 mg/kg TBTCl (whole body)[8]	Physiological, NA				[53]	L; 35% reduction in anoxia tolerance as percent of controls

Summary of Biological Effects Tissue Concentrations for Tributyltin

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	**Sediment**	**Water**	**Tissue (SampleType)**	**Effects**	**Log BCF**	**Log BAF**	**BSAF**	**Reference**	**Comments[3]**
Mytilus edulis, Mussel			0.556 mg/kg TBTCl (whole body)[8]	Physiological, ED5				[26]	L; 50% increase in respiration as compared to controls calculated from formula in text; exposure concentrations variable because of rapid uptake by test organisms so not measured or reported
			1.8 mg/kg TBTCl (whole body)[8]	Physiological, ED5				[26]	L; 50%reduction in clearance rate (feeding rate) as compared to controls; exposure concentrations variable because of rapid uptake by test organisms so not measured or reported

Summary of Biological Effects Tissue Concentrations for Tributyltin

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (SampleType)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
			1.08 mg/kg TBTCl (whole body)[8]	Physiological, LOED				[26]	L; significant decrease in clearance rate (feeding); exposure concentrations variable because of rapid uptake by test organisms so not measured or reported
			1.08 mg/kg TBTCl (whole body)[8]	Physiological, LOED				[26]	L; significant decrease in scope for growth; exposure concentrations variable because of rapid uptake by test organisms so not measured or reported
			0.8 mg/kg TBTCl (whole body)[8]	Physiological, NOED				[26]	L; no significant decrease in clearance rate (feeding); exposure concentrations variable because of rapid uptake by test organisms so not measured or reported

Summary of Biological Effects Tissue Concentrations for Tributyltin

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	**Sediment**	**Water**	**Tissue (SampleType)**	**Effects**	**Log BCF**	**Log BAF**	**BSAF**	**Reference**	**Comments[3]**
			0.8 mg/kg TBTCl (whole body)[8]	Physiological, NOED				[26]	L; no significant decrease in scope for growth; exposure concentrations variable because of rapid uptake by test organisms so not measured or reported
			2 mg/kg TBTCl (whole body)[8]	Physiological, NOED				[26]	L; no significant change in food absorption eficiency; exposure concentrations variable because of rapid uptake by test organisms so not measured or reported
Arca zebra, Mussel			1.11 µg/g dw[†] (soft tissue)	35 % reduction in scope for growth				[25]	F

Summary of Biological Effects Tissue Concentrations for Tributyltin

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	**Sediment**	**Water**	**Tissue (SampleType)**	**Effects**	**Log BCF**	**Log BAF**	**BSAF**	**Reference**	**Comments[3]**
Dreissena polymorpha, Zebra mussel		70 ng/L[4] ($\bar{x}$; n = 2)	73.13 µg/g dw TBT cation (soft tissue) ($\bar{x}$; n = 2)	Reduction in growth after 105 days exposure and transfer to clean site	5.95			[31]	F; steady-state reached after 35 days; 105-day uptake and depuration phases
Dreissena polymorpha, Zebra mussel			12.7 mg/kg TBTCl (whole body)[8]	Growth, NOED				[31]	F; concentration of TBT in tissues and water; field study at marina with exposure to TBT and DBT likely; mean values provided; no significant impact on growth
			1.66 mg/kg TBTCl (whole body)[8]	Growth, NOED				[31]	F; concentration of DBT in tissues and TBT in water; field study at marina with exposure to TBT and DBT likely; mean values provided; no significant impact on growth

Summary of Biological Effects Tissue Concentrations for Tributyltin

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	**Sediment**	**Water**	**Tissue (SampleType)**	**Effects**	**Log BCF**	**Log BAF**	**BSAF**	**Reference**	**Comments[3]**
Crassostrea gigas, Pacific oyster			0.75 mg/kg† (whole body)	Reduction in condition factor and growth				[33]	F
			0.27 µg/g† (soft tissue)	Reduced tissue growth; shell thickening				[34]	F
			2.38 mg/kg TBTO (soft tissue)	Reduced growth in spat				[28]	L; 45-day exposure
	0.08 µg/g dw	15 ±8 ng/L[4]	1.61 µg/g† (soft tissue)	Shell thickness index = 4.39; $\bar{x}$ tissue weight = 1.29				[30]	F
	0.03 µg/g dw	33 ±27 ng/L[4]	1.64 µg/g† (soft tissue)	Shell thickness index = 4.85; $\bar{x}$ tissue weight = 0.70				[30]	F
	0.02 µg/g dw	21 ±8 ng/L[4]	0.62 µg/g† (soft tissue)	Shell thickness index = 6.85; $\bar{x}$ tissue weight = 2.59				[30]	F
	0.10 µg/g dw	13 ng/L[4]	0.36 µg/g† (soft tissue)	Shell thickness index = 9.82; $\bar{x}$ tissue weight = 5.66				[30]	F

Summary of Biological Effects Tissue Concentrations for Tributyltin

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	**Sediment**	**Water**	**Tissue (SampleType)**	**Effects**	**Log BCF**	**Log BAF**	**BSAF**	**Reference**	**Comments[3]**
Crassostrea gigas, Pacific oyster	0.15 µg/g dw	22 ±12 ng/L[4]	1.20 µg/g† (soft tissue)	Shell thickness index = 4.78; $\bar{x}$ tissue weight = 1.26				[30]	F
	0.04 µg/g dw	17 ±12 ng/L[4]	1.46 µg/g† (soft tissue)	Shell thickness index = 4.67; $\bar{x}$ tissue weight = 0.67				[30]	F
	0.08 µg/g dw	13 ng/L[4]	0.44 µg/g† (soft tissue)	Shell thickness index = 8.10; $\bar{x}$ tissue weight = 2.62				[30]	F
	0.05 µg/g dw	8 ng/L[4]	0.33 µg/g† (soft tissue)	Shell thickness index = 10.2; $\bar{x}$ tissue weight = 2.84				[30]	F
	0.04 µg/g dw	35 ±17 ng/L[4]	1.49 µg/g† (soft tissue)	Shell thickness index = 5.14; $\bar{x}$ tissue weight = 0.97				[30]	F
	0.11 µg/g dw	17 ±9 ng/L[4]	1.73 µg/g† (soft tissue)	Shell thickness index = 5.29; $\bar{x}$ tissue weight = 0.84				[30]	F

Summary of Biological Effects Tissue Concentrations for Tributyltin

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	**Sediment**	**Water**	**Tissue (SampleType)**	**Effects**	**Log BCF**	**Log BAF**	**BSAF**	**Reference**	**Comments[3]**
	0.07 µg/g dw	22 ±14 ng/L[4]	0.61 µg/g † (soft tissue)	Shell thickness index = 8.07; $\bar{x}$ tissue weight = 2.23				[30]	F
	0.04 µg/g dw	8 ±2 ng/L[4]	0.38 µg/g† (soft tissue)	Shell thickness index = 9.83; $\bar{x}$ tissue weight = 2.90				[30]	F
	0.36 µg/g dw	45 ±17 ng/L[4]	1.24 µg/g† (soft tissue)	Shell thickness index = 4.95; $\bar{x}$ tissue weight = 0.77				[30]	F
	0.15 µg/g dw	31 ±18 ng/L[4]	1.57 µg/g† (soft tissue)	Shell thickness index = 5.04; $\bar{x}$ tissue weight = 0.49				[30]	F
Crassostrea gigas, Pacific oyster	0.31 µg/g dw	23 ±18 ng/L[4]	0.50	e weight = 2.39				[30]	F
	0.10 µg/g dw	11 ±4 ng/L[4]	0.45 µg/g† (soft tissue)	Shell thickness index = 10.2; $\bar{x}$ tissue weight = 2.77				[30]	F

Summary of Biological Effects Tissue Concentrations for Tributyltin

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (SampleType)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
	0.07 µg/g dw	26 ±9 ng/L[4]	0.74 µg/g† (soft tissue)	Shell thickness index = 5.06; $\bar{x}$ tissue weight = 1.24				[30]	F
	0.05 µg/g dw	22 ±15 ng/L[4]	1.26 µg/g† (soft tissue)	Shell thickness index = 5.24; $\bar{x}$ tissue weight = 1.24				[30]	F
	0.27 µg/g dw	13 ±5 ng/L[4]	0.34 µg/g† (soft tissue)	Shell thickness index = 9.83; $\bar{x}$ tissue weight = 2.44				[30]	F
	0.07 µg/g dw	8 ng/L[4]	0.31 µg/g† (soft tissue)	Shell thickness index = not sampled; $\bar{x}$ tissue weight = not sampled				[30]	F
	0.05 µg/g dw	26 ±12 ng/L[4]	0.80 µg/g† (soft tissue)	Shell thickness index = 5.39; $\bar{x}$ tissue weight = 1.18				[30]	F
	0.02 µg/g dw	18 ±13 ng/L[4]	0.98 µg/g† (soft tissue)	Shell thickness index = 5.29; $\bar{x}$ tissue weight = 1.14				[30]	F

Summary of Biological Effects Tissue Concentrations for Tributyltin

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	**Sediment**	**Water**	**Tissue (SampleType)**	**Effects**	**Log BCF**	**Log BAF**	**BSAF**	**Reference**	**Comments[3]**
	0.02 µg/g dw	15 ±12 ng/L[4]	0.24 µg/g† (soft tissue)	Shell thickness index = 9.00; $\bar{x}$ tissue weight = 2.50				[30]	F
Crassostrea gigas, Pacific oyster	0.04 µg/g dw	8 ±2 ng/L[4]	0.27 µg/g† (soft tissue)	Shell thickness index = 8.62; $\bar{x}$ tissue weight = 5.92				[30]	F
	<0.01 µg/g dw	11 ng/L[4]	0.37 µg/g† (soft tissue)	Shell thickness index = 9.63; $\bar{x}$ tissue weight = 1.39				[30]	F
	0.01 µg/g dw	23 ±23 ng/L[4]	0.56 µg/g† (soft tissue)	Shell thickness index = 6.48; $\bar{x}$ tissue weight = 1.44				[30]	F
	0.02 µg/g dw	10 ± ng/L[4]	0.17 µg/g† (soft tissue)	Shell thickness index = not sampled; $\bar{x}$ tissue weight = not sampled				[30]	F
	0.02 µg/g dw	3 ±2 ng/L[4]	0.11 µg/g† (soft tissue)	Shell thickness index = 19.8; $\bar{x}$ tissue weight = 5.96				[30]	F

Summary of Biological Effects Tissue Concentrations for Tributyltin

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	**Sediment**	**Water**	**Tissue (SampleType)**	**Effects**	**Log BCF**	**Log BAF**	**BSAF**	**Reference**	**Comments[3]**
	0.01 µg/g dw	16 ng/L[4]	0.18 µg/g† (soft tissue)	Shell thickness index = 12.4; $\bar{x}$ tissue weight = 2.72				[30]	F
	0.01 µg/g dw	6 ±5 ng/L[4]	0.28 µg/g† (soft tissue)	Shell thickness index = 9.64; $\bar{x}$ tissue weight = 2.06				[30]	F
	0.05 µg/g dw	6 ±5 ng/L[4]	0.08 µg/g† (soft tissue)	Shell thickness index = 23.3; $\bar{x}$ tissue weight = 4.11				[30]	F
	<0.01 µg/g dw	2 ng/L[4]	0.08 µg/g† (soft tissue)	Shell thickness index = 21.0; $\bar{x}$ tissue weight = 9.28				[30]	F
Crassostrea gigas, Pacific oyster	0.66 µg/g dw	38 ±21 ng/L[4]	2.26 µg/g† (soft tissue)	Shell thickness index = 4.95; $\bar{x}$ tissue weight = 1.00				[30]	F
	0.26 µg/g dw	366 ±29 ng/L[4]	2.18 µg/g† (soft tissue)	Shell thickness index = 3.96; $\bar{x}$ tissue weight = 0.97				[30]	F

Summary of Biological Effects Tissue Concentrations for Tributyltin

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (SampleType)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
	0.15 µg/g dw	76 ±43 ng/L[4]	1.34 µg/g† (soft tissue)	Shell thickness index = 6.87; $\bar{x}$ tissue weight = 2.53				[30]	F
	0.53 µg/g dw	25 ng/L[4]	0.65 µg/g† (soft tissue)	Shell thickness index = 14.9; $\bar{x}$ tissue weight = 3.65				[30]	F
	0.08 µg/g dw	38 ±33 ng/L[4]	0.88 µg/g† (soft tisue)	Shell thickness index = 10.6; $\bar{x}$ tissue weight = 1.56				[30]	F
	0.19 µg/g dw	13 ±4 ng/L[4]	1.35 µg/g† (soft tisue)	Shell thickness index = 5.98; $\bar{x}$ tissue weight = 0.89				[30]	F
	0.17 µg/g dw	13 ±5 ng/L[4]	0.50 µg/g† (soft tisue)	Shell thickness index = 12.5; $\bar{x}$ tissue weight = 3.19				[30]	F
	0.07 µg/g dw	8 ±3 ng/L[4]	0.26 µg/g† (soft tissue)	Shell thickness index = 14.7; $\bar{x}$ tissue weight = 7.09				[30]	F

Summary of Biological Effects Tissue Concentrations for Tributyltin

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	**Sediment**	**Water**	**Tissue (SampleType)**	**Effects**	**Log BCF**	**Log BAF**	**BSAF**	**Reference**	**Comments[3]**
	0.06 µg/g dw	15 ±6 ng/L[4]	1.39 µg/ † (soft tissue)	Shell thickness index = 7.56; $\bar{x}$ tissue weight = 1.77				[30]	F
	0.03 µg/g dw	11 ±6 ng/L[4]	1.44 µg/g† (soft tissue)	Shell thickness index = 5.41; $\bar{x}$ tissue weight = 1.32				[30]	F
Crassostrea gigas, Pacific oyster	0.04 µg/g dw	5 ±2 ng/L[4]	0.21 µg/g† (soft tissue)	Shell thickness index = 13.1; $\bar{x}$ tissue weight = 8.07				[30]	F
	0.05 µg/g dw	12 ±10 ng/L[4]	0.30 µg/g† (soft tissue)	Shell thickness index =10.6; $\bar{x}$ tissue weight = 1.56				[30]	F
	0.02 µg/g dw	7 ±6 ng/L[4]	0.49 µg/g† (soft tissue)	Shell thickness index = 12.4; $\bar{x}$ tissue weight = 1.24				[30]	F
	0.03 µg/g dw	6 ±2 ng/L[4]	0.25 µg/g† (soft tissue)	Shell thickness index = 25.7; $\bar{x}$ tissue weight = 5.22				[30]	F

Summary of Biological Effects Tissue Concentrations for Tributyltin

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	**Sediment**	**Water**	**Tissue (SampleType)**	**Effects**	**Log BCF**	**Log BAF**	**BSAF**	**Reference**	**Comments[3]**
	0.02 µg/g dw	6 ±4 ng/L[4]	0.13 µg/g† (soft tissue)	Shell thickness index = 189; $\bar{x}$ tissue weight = 6.23				[30]	F
	4.6 µg/g dw	93 ±45 ng/L[4]	6.35 µg/g† (soft tissue)	Shell thickness index = 3.21; $\bar{x}$ tissue weight = 0.37				[30]	F
	10.8 µg/g dw	1,090 ±1,850 ng/L[4]	3.65 µg/g† (soft tissue)	Shell thickness index = 8.06; $\bar{x}$ tissue weight = 0.12				[30]	F
	1.1 µg/g dw	82 ±9 ng/L[4]	5.60 µg/g† (soft tissue)	Shell thickness index = 4.34; $\bar{x}$ tissue weight = 0.95				[30]	F
	0.23 µg/g dw	25 ±7 ng/L[4]	1.28 µg/g† (soft tissue)	Shell thickness index = 6.73; $\bar{x}$ tissue weight = 1.24				[30]	F
Crassostrea gigas, Oyster			22 mg/kg TBTFl (whole body)[8]	Morphology, ED100				[45]	L and F combined; malformation of shells

Summary of Biological Effects Tissue Concentrations for Tributyltin

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	**Sediment**	**Water**	**Tissue (SampleType)**	**Effects**	**Log BCF**	**Log BAF**	**BSAF**	**Reference**	**Comments[3]**
			5 mg/kg TBTFl (whole body)[8]	Morphology, LOED				[45]	L and F combined; malformation of shells
			22 mg/kg TBTFl (whole body)[8]	Mortality, ED100				[45]	Land F combined; 100% mortality after 170 days
			5 mg/kg TBTFl (whole body)[8]	Mortality, NA				[45]	L and F combined; 30% mortality after 110 days
Crassostrea gigas, Oyster			0.75 mg/kg TBTCl (whole body)[8]	Growth, NA				[47]	F; 44% reduction in condition factor and growth
Crassostrea gigas, Oyster			3.7 mg/kg TBTO (whole body)[8]	Growth, ED100				[52]	L; no growth (weight increase or length) in high test concentration

Summary of Biological Effects Tissue Concentrations for Tributyltin

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (SampleType)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
			4.89 mg/kg TBTO (whole body)[8]	Growth, ED100				[52]	L; no growth (length) in high test concentration (with sediment present at 30 mg/L)
			1.71 mg/kg TBTO (whole body)[8]	Growth, ED100				[52]	L; no growth (length) in low test concentration
			4.89 mg/kg TBTO (whole body)[8]	Growth, NA				[52]	L; 92% reduction in growth (weight increase) in high test concentration relative to control
			1.71 mg/kg TBTO (whole body)[8]	Growth, NA				[52]	L; 70% reduction in growth (weight increase) in low test concentration relative to control

Summary of Biological Effects Tissue Concentrations for Tributyltin

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (SampleType)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
			1.3 mg/kg TBTO (whole body)[8]	Growth, NA				[52]	L; 47% reduction in growth (weight increase) in low test concentration with 30 mg/L sediment present relative to control
			1.71 mg/kg TBTO (whole body)[8]	Growth, NA				[52]	L; 70% reduction in growth (length) in low test concentration with 30 mg/L sediment present relative to control
			1.71 mg/kg TBTO (whole body)[8]	Mortality, NOED				[52]	L; no mortality in low test concentration (both with and without sediment present)
			3.7 mg/kg TBTO (whole body)[8]	Physiological, NA				[52]	L; 63% reduction in condition index relative to control in high test concentration

Summary of Biological Effects Tissue Concentrations for Tributyltin

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	**Sediment**	**Water**	**Tissue (SampleType)**	**Effects**	**Log BCF**	**Log BAF**	**BSAF**	**Reference**	**Comments[3]**
			4.89 mg/kg TBTO(whole body)[8]	Physiological, NA				[52]	L; 42% reduction in condition index relative to control in high test concentration with 30 mg/L sediment present
			1.71 mg/kg TBTO(whole body)[8]	Physiological, NA				[52]	L; 18% reduction in condition index relative to control in low test concentration
			1.3 mg/kg TBTO (whole body)[8]	Physiological, NA				[52]	L; 11% reduction in condition index relative to control in low test concentration with 30 mg/L sediment present
Ostrea edulis, Oyster			0.53 mg/kg TBTO (soft tissue)	No effect on growth in spat				[28]	L; 45-day exposure
Ostrea edulis, Oyster			0.75 mg/kg TBTO (soft tissue)	Reduced growth in spat				[28]	L; 45-day exposure

Summary of Biological Effects Tissue Concentrations for Tributyltin

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	**Sediment**	**Water**	**Tissue (SampleType)**	**Effects**	**Log BCF**	**Log BAF**	**BSAF**	**Reference**	**Comments[3]**
Saccostria commercialis, Sydney rock oyster			0.012 mg/kg TBTCl (whole body)[8]	Morphology, LOED				[51]	F
			0.04 mg/kg TBTCl (whole body)[8]	Morphology, LOED				[51]	F
Saccostria commercialis, Sydney rock oyster			110 ng Sn/g^{-1} (soft tissues)	Shell deformations; shell curl				[39]	F
			107 ng Sn/g^{-1} (soft tissues)	Shell deformations; shell curl				[39]	F
			86 ng Sn/g^{-1} (soft tissues)	Shell deformations; shell curl				[39]	F
			98 ng Sn/g^{-1} (soft tissues)	Shell deformations; shell curl				[39]	F
			87 ng Sn/g^{-1} (soft tissues)	Shell deformations; shell curl				[39]	F
			350 ng Sn/g^{-1} (soft tissues)	Shell deformations; shell curl				[39]	F
Cerastoderma edule, Cockle	445 ± 83 ng/g dw (n=5)	68.2 ± 40.6 ng/L[4] (n=8)	4,128 ng/g, dw† (pooled, soft tissue) (n=1)		4.78			[21]	F

Summary of Biological Effects Tissue Concentrations for Tributyltin

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	**Sediment**	**Water**	**Tissue (SampleType)**	**Effects**	**Log BCF**	**Log BAF**	**BSAF**	**Reference**	**Comments[3]**
Macoma balthica, Clam	445 ± 83 ng/g dw (n=5)	68.2 ± 40.6 ng/L[4] (n=8)	4,587 ± 2,793 ng/g dw† (pooled, soft tissue) (n=4)		4.83			[21]	F
Merceneria mercenaria, Hard shell clam	445 ± 83 ng/g dw (n=5)	68.2 ± 40.6 ng/L[4] (n=8)	8,649 ng/g dw† (pooled, soft tissue) (n=1)		5.10			[21]	F
Venerupis decussata, Clam			2.64 mg/kg TBTO (soft tissue)	Reduced growth in spat				[28]	L; 45-day exposure
Venerupis semidecussata, Clam			1.48 mg/kg TBTO (soft tissue)	No effect on spat growth				[28]	L; 45-day exposure
Mya arenaria, Soft shell clam	445 ± 83 ng/g dw (n = 5)	68.2 ± 40.6 ng/L[4] (n = 8)	36,807 ± 9,800 ng/g dw† (pooled, soft tissue) (n = 4)		5.73			[21]	F
Petricola pholadiformis, American piddock	445 ± 83 ng/g dw (n = 5)	68.2 ± 40.6 ng/L[4] (n = 8)	838 ± 108 ng/g dw† (pooled, soft tissue) (n = 2)		4.09			[21]	F

Summary of Biological Effects Tissue Concentrations for Tributyltin

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	**Sediment**	**Water**	**Tissue (SampleType)**	**Effects**	**Log BCF**	**Log BAF**	**BSAF**	**Reference**	**Comments[3]**
Scrobicularia plana, Clam	445 ± 83 ng/g dw (n = 5)	68.2 ± 40.6 ng/L[4] (n=8)	3,375 ± 232 ng/g dw† (pooled, soft tissue) (n = 4)		4.69			[21]	F
Scorbicularia plana, Clam	0.03 µg/g dw	4.0-17.5 ng/L[4]	0.635 µg Sn/g dw (soft tissues)					[40]	F
	µg/g dw	7.0-10.8 ng/L[4]	0.263 µg Sn/g dw (soft tissues)					[40]	F
	0.03 µg/g dw (n=3)	15.2-51.6 ng/L[4]	2.04 µg Sn/g dw (soft tissues)					[40]	F
	0.039 µg/g dw (n=3)	17.2-51.3 ng/L[4]	1.12 µg Sn/g dw (soft tissues)					[40]	F
	0.22 µg/g dw (n=3)	0.6-213 ng/L[4]	2.05 µg Sn/g dw (soft tissues)					[40]	F
Scorbicularia plana, Clam	0.12 µg/g dw (n=6)	10.9-33.2 ng/L[4]	1.69 µg Sn/g dw (soft tissues)					[40]	F
	0.11 µg/g dw	7.4 ng/L[4]	1.51 µg Sn/g dw (soft tissues)					[40]	F
	0.02 µg/g dw	2.7 ng/L[4]	0.62 µg Sn/g dw (soft tissues)					[40]	F
	0.126 µg/g dw	230 ng/L[4]	5.09 µg Sn/g dw (soft tissues)					[40]	F
			2.91 mg/kg TBTO (soft tissue)	Reduced growth in spat				[28]	L; 45-day exposure

Summary of Biological Effects Tissue Concentrations for Tributyltin

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	**Sediment**	**Water**	**Tissue (SampleType)**	**Effects**	**Log BCF**	**Log BAF**	**BSAF**	**Reference**	**Comments[3]**
Hyalella azteca, Amphipod		4.8 nM[3]	110 nmol/g dw (whole body)	4 week LC50				[22]	L; 1 week to reach equilibrium in tissues
Fishes									
Oncorhynchus mykiss, Rainbow trout		1.41 µg Sn/L		96-hr LC50	406			[8]	L
		0.42 µg Sn/L	1.21 mg Sn/kg (liver) 0.34 mg Sn/kg (gall bladder) 2.30 mg Sn/kg (kidney) 1.38 mg Sn/kg (carcass) 5.56 mg Sn/kg (peritoneal fat) 1.04 mg Sn/kg (gill) 0.67 mg Sn/kg (blood) 0.50 mg Sn/kg (gut) 0.32 mg Sn/kg (muscle) 2.20 mg Sn/kg (brain)					[8]	L; 15-dy exposure period

Summary of Biological Effects Tissue Concentrations for Tributyltin

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	**Sediment**	**Water**	**Tissue (SampleType)**	**Effects**	**Log BCF**	**Log BAF**	**BSAF**	**Reference**	**Comments[3]**
Oncorhynchus mykiss, Rainbow trout			0.11 mg/kg TBTO (whole body)	Behavior, LOED				[43]	L; significantly increased swimming behavior (distances and directions of)
			0.35 mg/kg TBTO (whole body)	Growth, LOED				[43]	L; significantly lower weight increase at lowest test concentration
			0.13 mg/kg TBTO (whole body)	Growth, LOED				[43]	L; significantly increased swimming behavior (distances and directions of)
			0.27 mg/kg TBTO (whole body)	Growth, LOED				[43]	L; significantly lower weight increase at lowest test concentration; residue from

Summary of Biological Effects Tissue Concentrations for Tributyltin

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	**Sediment**	**Water**	**Tissue (SampleType)**	**Effects**	**Log BCF**	**Log BAF**	**BSAF**	**Reference**	**Comments[3]**
Oncorhynchus mykiss, Rainbow trout		0.6 µg TBTO/ L[4]	2.5 µg TBTO/g (whole body)[7]	Histopatho-logical effects Spleen: 20% had lymphocytic depletion; 20% increased erythrophagia; Gills: 10% had cell necrosis within primary lamellae, 30% within secondary lamellae; Pseudobranch: 30% had cell necrosis within pseudobranch-ial tissue				[44]	L; 28-day exposure

Summary of Biological Effects Tissue Concentrations for Tributyltin

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	**Sediment**	**Water**	**Tissue (SampleType)**	**Effects**	**Log BCF**	**Log BAF**	**BSAF**	**Reference**	**Comments[3]**
		1.0 µg TBTO/ L[4]	2.75 µg TBTO/g (whole body)[7]	Histopatho-logical effects Spleen: 90% had lymphocytic depletion; 50% increased erythrophagia; Gills: 20% had cell necrosis within primary lamellae, 50% within secondary lamellae; Pseudobranch: 50% had cell necrosis within pseudobranch-ial tissue				[44]	L; 28-day exposure

Summary of Biological Effects Tissue Concentrations for Tributyltin

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	**Sediment**	**Water**	**Tissue (SampleType)**	**Effects**	**Log BCF**	**Log BAF**	**BSAF**	**Reference**	**Comments[3]**
Oncorhynchus mykiss, Rainbow trout		2.0 µg TBTO/L	5.5 µg TBTO/g (whole body)[7]	Histopathological effects Spleen: 30% had lymphocytic depletion; 70% increased erythrophagia; Gills: 40% had cell necrosis within primary lamellae, 50% within secondary lamellae; Pseudobranch: 20% had cell necrosis within oral mucosa, 30% had cell necrosis within pseudobranchial tissue				[44]	L; 28-day exposure
Oncorhynchus mykiss, Rainbow trout			13.1 mg/kg TBTO oxide (whole body)[8]	Mortality, ED50				[49]	L; median lethal dose

Summary of Biological Effects Tissue Concentrations for Tributyltin

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	**Sediment**	**Water**	**Tissue (SampleType)**	**Effects**	**Log BCF**	**Log BAF**	**BSAF**	**Reference**	**Comments[3]**
Oncorhynchus mykiss, Rainbow trout		4.0 µg TBTO/ L[4]	7.0 µg TBTO/g (whole body)[7]	Histopatho-logical effects; Spleen: 100% had lymphocytic depletion; 90% increased erythrophagia; Gills: 100% had cell necrosis within primary lamellae, 80% within secondary lamellae; Pseudobranch: 60% had cell necrosis within oral mucosa, 70% had cell necrosis within pseudobranch-ial tissue				[44]	L; 28-day exposure

Summary of Biological Effects Tissue Concentrations for Tributyltin

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	**Sediment**	**Water**	**Tissue (SampleType)**	**Effects**	**Log BCF**	**Log BAF**	**BSAF**	**Reference**	**Comments[3]**
Cyprinodon variegatus, Sheepshead minnow			40,800 mg/kg TBTO (liver)[8]	Development, NOED				[9]	L; TBTO as tin; no significant response for length or weight of F1 generation fish (parental exposure)
			1,210 mg/kg TBTO (muscle)[8]	Development, NOED				[9]	L; TBTO as tin; no significant response for length or weight of F1 generation fish (parental exposure)
			2,480 mg/kg TBTO (viscera)[8]	Development, NOED				[9]	L; TBTO as tin; no significant response for length or weight of F1 generation fish (parental exposure)
			2,600 mg/kg TBTO (whole body)[8]	Development, NOED				[9]	L; TBTO as tin in whole body of F1 generation; no significant response for length or weight of F1 generation fish (parental exposure)

Summary of Biological Effects Tissue Concentrations for Tributyltin

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (SampleType)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
			40,800 mg/kg TBTO (liver)[8]	Growth, NOED				[9]	L; TBTO as tin; no significant response for length or weight
			1,210 mg/kg TBTO (muscle)[8]	Growth, NOED				[9]	L; TBTO as tin; no significant response for length or weight
			2,480 mg/kg TBTO (viscera)[8]	Growth, NOED				[9]	L; TBTO as tin; no significant response for length or weight
			2,600 mg/kg TBTO (whole body)[8]	Growth, NOED				[9]	L; TBTO as tin in whloe body of F1 generation; no significant response for length or weight in adults
			40,800 mg/kg TBTO (liver)[8]	Reproduction, NOED				[9]	L; TBTO as tin; no significant response for number of eggs spawned per day per female, or hatching success

Summary of Biological Effects Tissue Concentrations for Tributyltin

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (SampleType)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
			1,210 mg/kg TBTO (muscle)[8]	Reproduction, NOED				[9]	L; TBTO as tin; no significant response for number of eggs spawned per day per female, or hatching success
			2,480 mg/kg TBTO (viscera)[8]	Reproduction, NOED				[9]	L; TBTO as tin; no significant response for number of eggs spawned per day per female, or hatching success
			2,600 mg/kg TBTO (whole body)[8]	Reproduction, NOED				[9]	L; TBTO as tin in whloe body of F1 generation; no significant response for number of eggs spawned per day per female, or hatching success in adults
Ictalurus punctatus, Channel catfish			0.1 mg/kg (whole body tissue residue concentrations)	Significant ($P < 0.05$) suppression of humoral response				[41]	L

Summary of Biological Effects Tissue Concentrations for Tributyltin

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	**Sediment**	**Water**	**Tissue (SampleType)**	**Effects**	**Log BCF**	**Log BAF**	**BSAF**	**Reference**	**Comments[3]**
Poecilia reticulata, Guppy			0.7 mg/kg TBTO (whole body tissue residue concentrations)	Histopatho-logical changes				[42]	
Stenotomus chrysops, Scup			202 mg/kg TBTCl (liver)[8]	Physiological, LOED				[48]	L; statistically significant reduction of hepatic enzyme activity
			16.3 mg/kg TBTCl (whole body)[8]	Physiological, LOED				[48]	L; statistically significant reduction of hepatic enzyme activity
			8 mg/kg TBTCl (liver)[8]	Cellular, NOED				[48]	L; no effect on liver histopathology
			14.7 mg/kg TBTCl (liver)[8]	Cellular, NOED				[48]	L; no effect on liver histopathology
			202 mg/kg TBTCl (liver)[8]	Cellular, NOED				[48]	L; no effect on liver histopathology
			3.3 mg/kg TBTCl (whole body)[8]	Cellular, NOED				[48]	L; no effect on liver histopathology

Summary of Biological Effects Tissue Concentrations for Tributyltin

Species:	Concentration, Units in[1]:			Toxicity:	Ability to Accumulate[2]:			Source:	
Taxa	Sediment	Water	Tissue (SampleType)	Effects	Log BCF	Log BAF	BSAF	Reference	Comments[3]
			8.1 mg/kg TBTCl (whole body)[8]	Cellular, NOED				[48]	L; no effect on liver histopathology
			16.3 mg/kg TBTCl (whole body)[8]	Cellular, NOED				[48]	L; no effect on liver histopathology
			8 mg/kg TBTCl (liver)[8]	Physiological, NOED				[48]	L; statistically insignificant reduction of hepatic enzyme activity
			14.7 mg/kg TBTCl (liver)[8]	Physiological, NOED				[48]	L; statistically insignificant reduction of hepatic enzyme activity
			3.3 mg/kg TBTCl (whole body)[8]	Physiological, NOED				[48]	L; statistically insignificant reduction of hepatic enzyme activity
			8.1 mg/kg TBTCl (whole body)[8]	Physiological, NOED				[48]	L; statistically insignificant reduction of hepatic enzyme 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] Surface water or aqueous concentration; not pore water.
[5] Laboratory toxicity test, co-occurrence of multiple contaminants with listed contaminant.
[6] Outdoor microcosm or artificial stream test, co-occurrence of multiple contaminants with listed contaminant.
[7] Residue concentration estimated from graphical material.
[8] 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.

† Type of tributyltin species not reported.

Conversion Factors:

[TBT] * 0.41 = [Sn]
[TBT] * 1.12 = [TBT Cl]
[TBT Cl * 0.36 = [Sn]
[TBT Cl] * 0.89 = [TBT]

[TBTO] * 0.97 = [TBT]
[Sn] * 2.74 = [TBT Cl]
[Sn] *2.44 = [TBT]

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Chemical Category: PESTICIDE (ORGANOPHOSPHATE)

Chemical Name (Common Synonyms): TERBUFOS **CASRN:**13071-79-9

Chemical Characteristics

Solubility in Water: 15 ppm [1]

Half-Life: No data [2]

Log K_{ow}: No data [3]

Log K_{oc}: —

Human Health

Oral RfD: 1.3 x 10^{-4} mg/kg/day [4]

Confidence: Not available, uncertainty factor = 10

Critical Effect: Inhibition of plasma cholinesterase observed in dogs

Oral Slope Factor: No data [5]

Carcinogenic Classification: D [6]

Wildlife

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

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

Aquatic Organisms

Partitioning Factors: Partitioning factors for terbufos in aquatic organisms were not found in the literature.

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

Toxicity/Bioaccumulation Assessment Profile

Terbufos, an organophosphate pesticide, is the active ingredient of Counter [7]. The application of Counter at the rate of 1.45 kg per hectare resulted in low-level exposure sufficient to induce blood plasma cholinesterase depressions, but generally not at levels sufficient to cause increased mortality to bobwhites and cottontails [8]. Turbofos is highly toxic to mammals. The acute oral LD50 for mice (*Mus musculus*) was 3.5 mg/kg [9], whereas 63 percent of exposed deer mice [7] were killed at 2.48 mg/kg dose. The residue of terbufos in live earthworms (1.73 mg/kg) was significantly lower than the residue (18.1 mg/kg) in dead organisms after a 32-day exposure [10].

Acute toxicity, expressed as the 96-h LC50 of terbufos to aquatic species, ranged from 4.7 µg/L for *Menidia beryllina* to 390 µg/L for *Pimephales promelas* [11]. Terbufos toxicity in the aquatic environment is influenced by pH and other physicochemical factors [12]. Experiments conducted with rainbow trout and *Gammarus* showed that terbufos was least toxic at pH 7.5, and more toxic at higher and lower pH. The accumulation factor (AF) for terbufos was influenced by salinity and temperature [13]. The AF for grass shrimp ranged from 20 at 30 ppt salinity and 22ºC to 64 at 25 ppt salinity and 17ºC, while the AF for sheepshead minnows ranged from 71 at 15 ppt salinity and 22ºC to 287 at 15 ppt salinity and 17ºC.

Summary of Biological Effects Tissue Concentrations for Terbufos

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									
Gammarus pseudolimnaeus, Amphipod			0.168 mg/kg (whole body) [4]	Mortality, ED50				[12]	L; lethal to 50% of animals in 96 hours
Palaemonetes pugio, Grass shrimp			0.07 mg/kg (whole body) [4]	Mortality, ED100				[13]	L; mortality
Fishes									
Oncorhynchus mykiss, Rainbow trout			4.08 mg/kg (whole body)[4]	Mortality, ED50				[12]	L; lethal to 50% of animals in 96 hours
Cyprinodon variegatus, Sheepshead minnow			0.11 mg/kg (whole body)[4]	Mortality, ED100				[13]	L; 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): Total PCBs **CASRN:** 1336-36-3

Chemical Characteristics

Solubility in Water: See Aroclors and congeners [1]

Half-Life: No data [2,3], See Aroclors congeners

Log K_{ow}: —

Log K_{oc}: —

Human Health

Oral RfD: See Aroclors and congeners [4]

Confidence: —

Critical Effect: See Aroclors and congeners

Oral Slope Factor: No data [4]

Carcinogenic Classification: 2A [4]

Wildlife

Partitioning Factors: BSAFs were calculated for red-winged blackbird and tree swallow eggs during a study in the Great Lakes are; with values ranging from 4.2 to 133, as reported in the attached table. BSAFs for tree swallow nestlings were 6.7 and 9.5.

Food Chain Multipliers: 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. Biomagnification factors of 32 and 93 were determined for total PCBs from alewife to herring gull eggs and from alewife to whole body herring gull, respectively [10]. A study of arctic marine food chains measured total PCB biomagnification factors of 3.7 to 8.8 for fish to seal, 7.4 to 13.9 for seal to bear, and 49.2 for fish to bear [11].

Aquatic Organisms

Partitioning Factors: A log BCF of 3.62 was measured for perch in a Swedish lake [40]. In a study of several lakes in central Ontario, BSFs for zooplankton ranged from 1.0 to 9.1. Log BAFs for fish ranged from −0.22 to 0.97, as reported in the summary table, and BSFs from 0.13 to 30 were noted.

Log BAFs for crayfish ranged −0.70 to 0.89 and BSFs ranged from 2.0 to 23.7 in the Ontario lakes study [35]. Log BAFs for clams in that study ranged from −0.05 to 0.32 with BSF values from 2.1 to 10.4.

Food Chain Multipliers: Polychlorinated biphenyls 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 (see following table). 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. In a study of several lakes in Ontario, log biomagnification factors for transfer from zooplankton to fish ranged from 0.00 to 0.97, as reported in the attached summary table for total PCBs.

Observed and Relative Concentrations of PCBs in Organisms of the Lake Ontario Food Web [12]

Species	Observed Concentrations (ng/g ww)	Relative Concentration
Phytoplankton	50	1
Mysids	330	6.6
Pontoporeia affinis	790	15.8
Oligochaetes	180	3.6
Sculpin	1600	32
Alewife	1300	26
Smelt	1400	28
Salmonids	4300	86

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.

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], whereas 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].

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 [23]. 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 [23]. 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 [24]. 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 [25,26]. 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 [27,28]. 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 [27]. Consequently, fish and other aquatic organisms may accumulate more of the higher-chlorinated PCB congeners than are found in the environment [15].

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 [29]. Toxic responses have been noted to occur at concentrations of 0.03 and 0.014 µg/L in marine and freshwater environments, respectively [29]. The LC50 for grass shrimp exposed to PCBs in marine waters for 4 days was 6.1 to 7.8 µg/L

[29]. Chronic toxicity of PCBs presents a serious environmental concern because of their resistance to degradation [30], 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 [31].

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. [32] 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 [33] 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 Total PCBs

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									
Zooplankton, (species not named specifically)	Boshkung Lake: 27.2; 356 (TOC normalized) µg/kg (dw)	0.93 ng/L	11.6, 392 (lipid normalized) µg/kg[4]				1.6	[34,35]	F; seven lakes in central Ontario; water samples are filtered samples collected from the water column at 1 m depth; BSF values appear in the BSAF column; BSF was calculated as the concentration of total PCBs (lipid basis) divided by the concentration in surface sediment (organic carbon basis)
	Wood Lake: 15.2; 156 (TOC normalized) µg/kg (dw)	1.85 ng/L	3.56, 1030 (lipid normalized) µg/kg				6.4		
	St. Nora Lake: 12; 227 (TOC normalized) µg/kg (dw)	1.60 ng/L	4.36, 1550 (lipid normalized) µg/kg				6.7		
	Opeongo Lake: 53.9; 546 (TOC normalized) µg/kg (dw)	1.23 ng/L	6.11, 766 (lipid normalized) µg/kg				1.4		
	Skugog Lake:						9.1		
	Rice Lake:						8.5		
	Clear Lake:						1.0		

Summary of Biological Effects Tissue Concentrations for Total PCBs

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	Stations: M1C = 385 µg/kg (dw) M2B = 325 µg/kg (dw) M4 = 1060 µg/kg (dw) M5 = 2.73 µg/kg (dw) M8 = 1290 µg (dw) M89B = 559 µg/kg (dw)		Stations: M1C = 314 µg/kg M2B = 143 µg/kg M4 = 349 µg/kg M5 = 279 µg/kg M8 = 872 µg/kg M89B= 153 µg/kg					[36]	F; sediment samples from the New York Bight; total PCB concentrations were quantified as a sum of Aroclor 1242 and 1254
Nereis incisa, Polychaete worm	Station: M1C = 385 µg/kg (dw)		Station: M1C = 326 µg/kg					[36]	F; sediment samples from the New York Bight; total PCB concentrations were quantified as a sum of Aroclor 1242 and 1254

Summary of Biological Effects Tissue Concentrations for Total PCBs

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]**
Nereis virens, Sandworm	Day 180: 4,310±640 µg/kg (dw)		Day 180: 522±178 µg/kg					[36]	L; sediment from Passaic River from four stations was composited for bioaccumulation study with commercial species; TOC was 5.7%. sediment and tissue (whole body) concentrations are mean and SD concentrations of three replicate tests
Ninoe nigripes, Polychaete worm	Stations: M5 = 2.73 µg/kg (dw) M89A = 13.3 µg/kg (dw) Ref = 33.1 µg/kg (dw)		Stations: M5 = 48.9 µg/kg M89A = 402 µg/kg MXRef = 176 µg/kg					[36]	F; sediment samples from the New York Bight; total PCB concentrations were quantified as a sum of Aroclor 1242 and 1254

Summary of Biological Effects Tissue Concentrations for Total PCBs

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]**
Pherusa affinis, Polychaete worm	Stations: M1C = 385 µg/kg (dw) M4B = 201 µg/kg (dw)		Stations: M1C = 129 µg/kg M4B = 107 µg/kg					[36]	F; sediment samples from the New York Bight; total PCB concentrations were quantified as a sum of Aroclor 1242 and 1254
Polinices duplicatus, Moon snail	Station: M5 = 2.73 µg/kg (dw)		Station: M5 = 78.1 µg/kg					[36]	F; sediment samples from the New York Bight; total PCB concentrations were quantified as a sum of Aroclor 1242 and 1254
Mytilus edulis, Mussel	0.14-45µg/kg dw	0.045-1.8 ng/L	2.7-3.2 ng/g						
Mytilus edulis, Mussel			0.6 mg/kg (whole body)[5]	Mortality, NA				[64]	L; no significant decrease in anoxic survival time (control 13 days)
			1.4 mg/kg (whole body)[5]	Mortality, NA				[64]	L; decreased anoxic survival time (control 10.7 days)

Summary of Biological Effects Tissue Concentrations for Total PCBs

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]**
			1.4 mg/kg (whole body)[5]	Physiological, NOED				[64]	L; no significant changes in adenylate energy charge or glycogen content
Clams (species not named specifically)	Boshkung Lake: 27.2, 356 (TOC normalized) µg/kg (dw)	0.93 ng/L	8.16, 2330 (lipid normalized) µg/kg			0.59	6.5	[34,35]	F; six lakes in central Ontario; water samples are filtered samples collected from the water column at 1 m depth; BSF values appear in the BSAF column; BSF was calculated as the concentration of total PCBs (lipid basis) divided by the concentration in surface sediment (organic carbon basis)
	Wood Lake: 15.2, 156 (TOC normalized) µg/kg (dw)	1.85 ng/L	4.63, 1670 (lipid normalized) µg/kg			0.20	10.4		
	St. Nora Lake: 12, 227 (TOC normalized) µg/kg (dw)	1.60 ng/L	3.57, 1590 (lipid normalized) µg/kg			0.00	6.9		
	Opeongo Lake: 53.9, 546 (TOC normalized) µg/kg (dw)	1.23 ng/L	6.32, 1630 (lipid normalized) µg/kg			0.32	2.1		
	Rice Lake:					-0.05	6.9		
	Clear Lake					0.46	2.7		

Summary of Biological Effects Tissue Concentrations for Total PCBs

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	2.3 ng/g dw 3.3 ng/g dw	surface water: 43.2 ng/L 6.4 ng/L	7.6 µg/g of lipid 7.2 µg/g of lipid					[38]	F; Rio Santiago and Rio de la Plata, Argentina
Spisula solidissima, Clam	Station: M5B = ND µg/kg (dw)		Station: M5B = 38.1µg/kg					[36]	F; Sediment samples from the New York Bight; total PCB concentrations were quantified as a sum of Aroclor 1242 and 1254.
Macoma nasuta, Clam	Day 180: 4310 ± 640 µg/kg (dw)		Day 120: 68.9 ±10.3µg/kg					[36]	L; sediment from Passaic River from four stations was composited for bioaccumulation study with commercial species; TOC was 5.7%; sediment and tissue whole body concentrations are mean and SD concentrations of three replicate tests
Macoma nasuta, Bent nose clam			1.7 mg/kg (whole body)[5]	Behavior, NOED				[45]	L; no effect on burrowing behavior

Summary of Biological Effects Tissue Concentrations for Total PCBs

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]**
			1.7 mg/kg (whole body)[5]	Mortality, NOED				[45]	L; no effect on mortality
Mercenaria mercenaria, Clam	Stations: M7 = 12.8 µg/kg (dw) MXRef = 33.1 µg/kg (dw)		Stations: M7 = 48.7µg/kg MX>Ref = 95.7µg/kg					[36]	F; sediment samples from the New York Bight; total PCB concentrations were quantified as a sum of Aroclor 1242 and 1254
Pitar morrhuana, Clam	Station: M4B = 201 µg/kg (dw)		Station: M4B = 37.2µg/kg						
Mya truncata, Bivalves:	0.14-45 µg/kg dw	surface water: 0.045-1.8 ng/L	0.89-2.2 ng/g					[37]	F; sum of 47 congeners in Cambridge Bay, Northwest Territories, Canada; sediment samples collected from 65 sites over 3 years
Orchomene sp., Amphipod	0.14-45 g/kg dw	0.045-1.8 ng/L	32-36 ng/g						

Summary of Biological Effects Tissue Concentrations for Total PCBs

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]**
Palaemonetes pugio, Grass shrimp	Mean of day 0 and day 180 replicates: 3,550±1,070 µg/kg (dw)		Day 28: 147 ± 42µg/kg					[36]	TOC was 5.7%; sediment and tissue whole body concentrations are mean and SD concentrations of three replicate tests; early removal of shrimp to avoid preying upon other species (28-day exposure, not yet steady state)
Mysis relicta, Opossum Shrimp			1.9 mg/kg (whole body)[5]	Behavior, NOED				[56]	L; no effect on feeding behavior

Summary of Biological Effects Tissue Concentrations for Total PCBs

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]**
Procambarus sp., Crayfish				Scugog Lake Rice Lake Clear Lake		0.41 -0.70 0.89	23.7 2.0 7.3	[35]	F; three lakes in central Ontario; water samples are filtered samples collected from the water column at 1 m depth; BSF values appear in the BSAF column; BSF was calculated as the concentration of total PCBs (lipid basis) divided by the concentration in surface sediment (organic carbon basis)
Callinectes sapidus, Crab	Station: M5 =2.73µg/kg (dw)		Station: M5 = 69.9 µg/kg (muscle) M5 = 1,870 µg/kg (hepatopancreas)					[36]	F; sediment samples from the New York Bight; total PCB concentrations were quantified as a sum of Aroclor 1242 and 1254
Chironomus riparius, Midge			3.3 mg/kg (whole body)[5]	Behavior, NOED				[57]	L; no effect on swimming behavior
			1.1 mg/kg (whole body)[5]	Behavior, NOED				[57]	L; no effect on swimming behavior

Summary of Biological Effects Tissue Concentrations for Total PCBs

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]**
			0.3 mg/kg (whole body)[5]	Behavior, NOED				[57]	L; no effect on swimming behavior
Ephemera danica, Mayfly			1.5 mg/kg (whole body)[5]	Growth, NOED				[63]	L
			1.5 mg/kg (whole body)[5]	Mortality, NOED				[63]	L
Asterias rubens, Starfish			19.2 mg/kg (gonad)[5]	Reproduction, LOED				[47]	L; concentrations are ug/g lipid gonadal indices evaluated
			0.146 mg/kg (gonad)[5]	Development, LOED				[48]	L; estimated wet weight adult males
			0.324 mg/kg (gonad)[5]	Development, LOED				[48]	L; estimated wet weight adult females
Fishes									
Oncorhynchus mykiss, Rainbow trout			50 mg/kg (whole body)[5]	Physiological, LOED				[53]	L; mixed function oxidase induction, including benzo(a)pyrene hydroxylase induction
			100 mg/kg (whole body)[5]	Physiological, NA				[53]	
			200 mg/kg (whole body)[5]	Physiological, NA				[53]	

Summary of Biological Effects Tissue Concentrations for Total PCBs

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]**
			0.29 mg/kg (whole body)[5]	Physiological, ED50				[54]	L; internal dose used as tissue concentration; induction of aryl hydrocarbon hydroxylase (AHH)
			0.56 mg/kg (whole body)[5]	Physiological, ED50				[54]	
Oncorhynchus mykiss, Rainbow trout			1.3 mg/kg (fat)[5]	Physiological, ED30				[61]	L; 30% decrease in hemoglobin content relative to control
			2.2 mg/kg (Fat)[5]	Physiological, ED30				[61]	L; 30% increase in liver size relative to control
			2.2 mg/kg (fat)[5]	Physiological, ED30				[61]	L; 30% decrease in hemoglobin content relative to control
			1.3 mg/kg (fat)[5]	Physiological, ED30				[61]	L; 30% increase in liver size relative to control
			1.7 mg/kg (fat)[5]	Physiological, ED35				[61]	L; 35% increase in kidney size relative to control
			1.3 mg/kg (fat)[5]	Physiological, ED35				[61]	L; 35% increase in kidney size relative to control

Summary of Biological Effects Tissue Concentrations for Total PCBs

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]**
			2.2 mg/kg (fat)[5]	Growth, ED40				[61]	L; 40% decrease in growth relative to control
			1.3 mg/kg (fat)[5]	Growth, ED40				[61]	L; 40% decrease in growth relative to control
Oncorhynchus kisutch, Coho salmon			645 mg/kg (whole body)[5]	Mortality, ED100				[60]	L; radiolabeled - contaminated food fed
			43 mg/kg (carcass)[5]	Morphology, LOED				[55]	L; decrease in hepatosomatic index
			43 mg/kg (carcass)[5]	Physiological, LOED				[55]	L; lipid levels in carcass decreased
			9.8 mg/kg (carcass)[5]	Morphology, NOED				[55]	L; no decrease in hepatosomatic index
			9.8 mg/kg (carcass)[5]	Physiological, NOED				[55]	L; no effect on lipid levels in carcass
Oncorhynchus tshawytscha, Chinook salmon			3.5 mg/kg (whole body)[5]	Cellular, LOED				[52]	L; structure changes in intestine cells, increased exfoliation of mucosa, mucosal cell inclusions
			3.5 mg/kg (whole body)[5]	Growth, NOED				[52]	L; no effect on weight gain

Summary of Biological Effects Tissue Concentrations for Total PCBs

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]**
Salmo salar, Atlantic salmon			30 mg/kg (whole body)[5]	Mortality, NOED				[46]	L; no effect on mortality
Salmonids						7.81 (log BAF)	1.85	[12]	F
Salvelinus namaycush, Lake trout			0.31 mg/kg (eggs)	Egg hatchability reduced by 57% and fry survival reduced by 19% relative to the control.				[39]	L; Total PCB was measured as Aroclor 1284; total DDT in eggs was 0.15 mg/kg which was also significantly higher than in controls
Salvelinus namaycush, Lake trout	Boshkung Lake: 27.2; 356 (TOC normalized) µg/kg (dw)	0.93 ng/L	87.6, 1,550 (lipid normalized) µg/kg			0.41	4.3	[34,35]	F; four lakes in central Ontario; water samples are filtered samples collected from the water column at 1 m depth; BSF values appear in the BSAF column; BSF was calculated as the concentration of total PCBs (lipid basis) divided by the concentration in surface sediment (organic carbon basis)
	St. Nora Lake: 12; 227 (TOC normalized) µg/kg (dw)	1.6 ng/L	17.4, 2,460 (lipid normalized) µg/kg			0.20	10.7		
	Opeongo Lake: 53.9; 546 (TOC normalized) µg/kg (dw)	1.23 ng/L	48.8, 2,100 (lipid normalized) µg/kg			0.43	3.8		

Summary of Biological Effects Tissue Concentrations for Total PCBs

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]**
	Clear Lake:					0.97	8.8		
Salvenlinus namaycush, Lake trout			2.3 mg/kg (whole body)[5]	Growth, LOED				[58]	L; PCB dosed with acetone carrier; enhanced growth (weight only; not length)
			2.4 mg/kg (whole body)[5]	Growth, LOED				[58]	L; PCB dosed with acetone carrier; enhanced growth (weight and length)
			1.8 mg/kg (whole body)[5]	Growth, LOED				[58]	L; PCB with no acetone carrier; enhanced growth (weight and length)
			0.76 mg/kg (whole body)[5]	Growth, NOED				[58]	L; PCB dosed with acetone carrier; no effect on growth (weight or length)
			2.1 mg/kg (whole body)[5]	Growth, NOED				[58]	L; PCB with no acetone carrier; no effect on growth (weight or length)
			0.76 mg/kg (whole body)[5]	Growth, NOED				[58]	L; on growth (weight or length)

Summary of Biological Effects Tissue Concentrations for Total PCBs

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]**
			0.76 mg/kg (whole body)[5]	Mortality, NOED				[58]	L; PCB dosed with acetone carrier; no effect on mortality
			2.3 mg/kg (whole body)[5]	Mortality, NOED				[58]	L; PCB dosed with acetone carrier; no effect on mortality
			2.4 mg/kg (whole body)[5]	Mortality, NOED				[58]	L; PCB dosed with acetone carrier; no effect on mortality
			0.76 mg/kg (whole body)[5]	Mortality, NOED				[58]	L; PCB with no acetone carrier; no effect on mortality
			2.1 mg/kg (whole body)[5]	Mortality, NOED				[58]	L; PCB with no acetone carrier; no effect on mortality
			1.8 mg/kg (whole body)[5]	Mortality, NOED				[58]	L; PCB with no acetone carrier; no effect on mortality
			1.5 mg/kg (eggs)[5]	Reproduction, LOED				[59]	L
Myoxocephalus quadircornis, Four horn sculpin	0.14-45µg/kg dw	surface water: 0.045-1.8 ng/L	7.3-230 ng/g (whole body excluding liver) 12-1,300 (liver)					[37]	F; 2-4 individuals of each species of sculpin were pooled to make a sample from each site

Summary of Biological Effects Tissue Concentrations for Total PCBs

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]**
Myoxocephalus scorpius, Short-horn sculpin	0.14-45µg/kg dw	0.045-1.8 ng/L	1.4-38 ng/g (whole body excluding liver) 5.5-220 (liver)						
Gados ogac, Greenland cod	0.14-45µg/kg dw	0.045-1.8 ng/L	4.4-39 ng/g (whole body excluding liver) 100-2,500 (liver)						F; analyzed as individual fish
Salvelinus alpinus, Arctic char	0.14-45µg/kg dw	0.045-1.8 ng/L	3.4-3.5 ng/g (whole body excluding liver) 5.1-7.8 (liver)						F; analyzed as individual fish
Prochilodus platensis	3 ng/g dw	13.8 ng/L	6.7, 17.8, 9.2 µg/g of lipid (muscle)					[38]	F; Rio Santiago and Rio de la Plata, Argentina
Pimelodus albicans	3 ng/g dw	13.8 ng/L	3.3 µg/g of lipid (muscle)						
Oligoscarcus jenynsi	58 ng/g dw	42.3 ng/L	4.1 µg/g of lipid (muscle)						
Carassius auratus, Goldfish			253 mg/kg (whole body)[5]	Mortality, ED50				[51]	L; lethal body burden
			271 mg/kg (whole body)[5]	Mortality, ED50				[51]	L; lethal body burden

Summary of Biological Effects Tissue Concentrations for Total PCBs

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]**
			293 mg/kg (whole body)[5]	Mortality, ED50				[51]	L; lethal body burden
			324 mg/kg (whole body)[5]	Mortality, ED50				[51]	L; lethal body burden
			250 mg/kg (whole body)[5]	Mortality, ED50				[51]	L; lethal body burden
			256 mg/kg (whole body)[5]	Mortality, ED50				[51]	L; lethal body burden
			250 mg/kg (whole body)[5]	Behavior, LOED				[51]	L; loss of appetite, lack of coordination
			250 mg/kg (whole body)[5]	Morphology, LOED				[51]	L; color changes
Notemigonvs crysoleucas, Golden shiner	Boshkung Lake: 27.2; 356 (TOC normalized) µg/kg (dw)	0.93 ng/L	5.44,642 (lipid normalized) µg/kg			0.04	1.8	[34,35]	F; six lakes in central Ontario; water samples are filtered samples collected from the water column at 1 m depth; BSF values appear in the BSAF column; BSF was calculated as the concentration of total PCBs (lipid basis) divided by the concentration in surface sediment (organic carbon basis)
	Wood Lake: 15.2; 156 (TOC normalized) µg/kg (dw)	1.85 ng/L	4.25,1170 (lipid normalized) µg/kg			0.04	7.3		
	St. Nora Lake: 12; 227 (TOC normalized) µg/kg (dw)	1.60 ng/L	5.20,683 (lipid normalized) µg/kg			-0.22	3.0		

Summary of Biological Effects Tissue Concentrations for Total PCBs

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]**
	Opeongo Lake: 53.9; 546 (TOC normalized) µg/kg (dw)	1.23 ng/L	11.9,482 (lipid normalized) µg/kg			-0.22	0.9		
	Rice Lake:					-0.40	2.9		
	Clear Lake:					-1.00	0.13		
Phoxinus phoxinus, Minnow			1.6 mg/kg (whole body)[5]	Behavior, LOED				[43]	L; changes in feeding behavior
			170 mg/kg (whole body)[5]	Growth, LOED				[43]	L; increased growth
			170 mg/kg (whole body)[5]	Mortality, LOED				[43]	L; doubling of mortality rate compared to controls after 300 days
			15 mg/kg (whole body)[5]	Reproduction, LOED				[43]	L; reduction in time to hatch, fry death
			170 mg/kg (whole body)[5]	Reproduction, NA				[43]	L; 85% reduction in hatchability of eggs

Summary of Biological Effects Tissue Concentrations for Total PCBs

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 notatus, Bluntnose minnow	Boshkung Lake: 27.2; 356 (TOC normalized) µg/kg (dw)	0.93 ng/L	9.78, 1130 (lipid normalized) µg/kg			0.28	3.1	[34,35]	F; six lakes in central Ontario; water samples are filtered samples collected from the water column at 1 m depth; BSF values appear in the BSAF column; BSF was calculated as the concentration of total PCBs (lipid basis) divided by the concentration in surface sediment (organic carbon basis)
	Wood Lake: 15.2; 156 (TOC normalized) µg/kg (dw)	1.85 ng/L	6.24, 446 (lipid normalized) µg/kg			-0.40	2.8		
	St. Nora Lake: 12; 227 (TOC normalized) µg/kg (dw)	1.60 ng/L	10.4, 993 (lipid normalized) µg/kg			-0.22	4.3		
	Opeongo Lake: 53.9; 546 (TOC normalized) µg/kg (dw)	1.23 ng/L	7.96, 893 (lipid normalized) µg/kg			0.08	1.6		
	Scugog Lake:					0.23	13.2		
	Clear Lake:					1.20	13.8		
Lepomis macrochirus, Bluegill			0.6 mg/kg (muscle)[5]	Physiological, ED50				[49]	L; inhibition of Mg-ATPase activity

Summary of Biological Effects Tissue Concentrations for Total PCBs

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]**
Morone saxatilis, Striped bass			4.4 mg/kg (whole body)[5]	Growth, NOED				[65]	L; parental exposure to PCBs in field, then post yolk absorption exposure of immature to PCB contaminated brine shrimp; no significant change in growth
Micropterus dolomieu, Smallmouth bass	Boshkung Lake: 27.2; 356 (TOC normalized) µg/kg (dw)	9.3 ng/L	25.5, 2420 (lipid normalized) µg/kg			0.6	6.7	[34,35]	F; seven lakes in central Ontario; water samples are filtered samples collected from the water column at 1 m depth; BSF values appear in the BSAF column; BSF was calculated as the concentration of total PCBs (lipid basis) divided by the concentration in surface sediment (organic carbon basis)
	Wood Lake: 15.2; 156 (TOC normalized) µg/kg (dw)	1.85 ng/L	6.17, 1160 (lipid normalized)µg/kg			0,04	7.3		
	St. Nora Lake: 12; 227 (TOC normalized) µg/kg (dw)	1.60 ng/L	35.4, 2910 (lipid normalized)µg/kg			0.28	12.7		
	Opeongo Lake: 53.9; 546 (TOC normalized) µg/kg (dw)	1.23 ng/L	4.77, 2200 (lipid normalized)µg/kg			0.46	4.0		
	Scugog Lake:					-0.22	5.1		
	Rice Lake:					0.26	15.5		
	Clear Lake:					0.60	3.8		

Summary of Biological Effects Tissue Concentrations for Total PCBs

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]**
Perca flavescens, Yellow perch	Boshkung Lake: 27.2; 356 (TOC normalized) µg/kg (dw)	0.93 ng/L	11.4, 4260 (lipid normalized) µg/kg			0.86	11.8	[34,35]	F; seven lakes in central Ontario; water samples are filtered samples collected from the water column at 1 m depth; BSF values appear in the BSAF column; BSF was calculated as the concentration of total PCBs (lipid basis) divided by the concentration in surface sediment (organic carbon basis)
	Wood Lake: 15.2; 156 (TOC normalized) µg/kg (dw)	1.85 ng/L	8.76, 3440 (lipid normalized) µg/kg			0.52	26.8		
	St. Nora Lake: 12; 227 (TOC normalized) µg/kg (dw)	1.60 ng/L	8.43, 3140 (lipid normalized) µg/kg			0.30	13.7		
	Opeongo Lake: 53.9; 546 (TOC normalized) µg/kg (dw)	1.23 ng/L	4.98, 3310 (lipid normalized) µg/kg			0.63	6.0		
	Scugog Lake:					0.52	30		
	Rice Lake:					-0.30	4.4		
	Clear Lake:					0.84	6.6		

Summary of Biological Effects Tissue Concentrations for Total PCBs

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]**
Perca fluviatilis, Perch		Surface water: 8.6 ng/L (geometric mean) (4.2-20.8)	825 ng/g (geometric mean) (513-1,244)		3.62			[40]	F; fat % = 2.3, SD = 0.6. Fish and water were sampled in Lake Jarnsjon, Sweden. PCBs in water were measured continuously in summer and autumn (concentration reflects both dissolved and particulate). Ten fish were collected.
Fundulus heteroclitus, Mummichog			10 mg/kg (whole body)[5]	Physiological, LOED				[50]	L; induction of ethoxyresorufin O-deethylase (EROD)
			32 mg/kg (whole body)[5]	Physiological, LOED				[50]	L; induction of cytochrome P4501a
			100 mg/kg (whole body)[5]	Physiological, not applicable				[50]	L; hepatic enzyme induction (P4501 & EROD)
			0.32 mg/kg (whole body)[5]	Physiological, NOED				[50]	L; no induction of hepatic enzymes
			1 mg/kg (whole body)[5]	Physiological, NOED				[50]	L; no induction of hepatic enzymes
			3.2 mg/kg (whole body)[5]	Physiological, NOED				[50]	L; no induction of hepatic enzymes

Summary of Biological Effects Tissue Concentrations for Total PCBs

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]**
Platyichthes stellatus, Starry flounder			<0.2 mg/kg (eggs)	Reduced reproductive success				[32]	F; field-collected fish injected with carp pituitary extract to induce final stages of gametogenesand spawning; in the field, the fish were exposed to sediments contaminated with PCBs, DDT, and PAHs
Pleuronectes americanus, Winter flounder			7.1 mg/kg (whole body)[5]	Growth, LOED				[44]	L; reduced length and weight of larvae
Limanda limanda, Dab			0.0181 mg/kg (muscle)[5]	Biochemical, LOED				[62]	L; total cytochrome P450 levels significantly different from control / sum of CB 77,105,118,156)
			0.0181 mg/kg (muscle)[5]	Biochemical, LOED				[62]	L; 7-ethoxyresorutin-O-deethylase (EROD) activity significantly different from control/sum of CB congeners 77, 105, 118, 156)

Summary of Biological Effects Tissue Concentrations for Total PCBs

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]**
			0.0181 mg/kg (muscle)[5]	Biochemical, LOED				[62]	L; Cytochrome P4501a (CYPIA) levels significantly different from control/sum of CB congeners77, 105, 118, 156)
Wildlife									
Haliaeetus leucocephalus, Bald eagle			fish tissue (diet in natural system): interior fish = 0.2 mg/kg shoreline fish =2.1 mg/kg	NOAEC at 4.0 mg/kg (egg), 0.14 mg/kg (fish); egg lethality from diet of interior fish at 0.2 mg/kg, shoreline fish at 2.1 mg/kg				[10]	F

Summary of Biological Effects Tissue Concentrations for Total PCBs

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]**
Agelaius phoeniceus, Red-winged blackbird (eggs)	7.4 ng/g TOC=2.5%		223.5 ng/g				16.4	[41]	F; Great Lakes/St. Lawrence River basin; 12 wetlands sites; sediment concentration reported as wet weight concentration which may be a typo-graphical error
	32.6 ng/g TOC=21.0%		50.1 ng/g				5.8		
	68.2 ng/g TOC=7.5%		54.6 ng/g				6.0		
	147.7 ng/g TOC=12%		52.7 ng/g				4.2		
	28.1 ng/g TOC-18.5%		163.5 ng/g				22.4		
	144.1 ng/g TOC=11.5%		247.8 ng/g				6.6		
	2.3 ng/g TOC-10.5%		105.9 ng/g				102.8		
	2.9 ng/g TOC=13.8%		64.9 ng/g				64.4		
	8.0 ng/g TOC=11.1%		108.3 ng/g				31.3		
	11.1 ng/g TOC-23.9%		81.8 ng/g				38.3		
Tachycineta bicolor, Tree swallow (nestlings)	144.1 ng/g TOC=11.5%		(whole body minus feet, beak, wings, and feathers)				9.5	[41]	F; Great Lakes/St. Lawrence River basin; 12 wetlands sites; sediment concentration reported as wet weight concentration which may be a typo-graphical error
	2.9 ng/g TOC=13.8%		754.5 ng/g				6.7		
			11.2 ng/g						
(eggs)	144.1 ng/g TOC=11.5%		1,019.7 ng/g				15.2		
	2.9 ng/g TOC=13.8%		254.6 ng/g				133.1		

Summary of Biological Effects Tissue Concentrations for Total PCBs

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]**
Mustela vison, Ranch mink, (fed PCB-contaminated *Cyprinus carpio* carp)			NOAEL (control group) 0.09 µg PCBs/g liver tissue <5.00 pg TEQ/g liver tissue LOAEL (10% carp in diet group) 2.19 µg PCBs/g liver tissue 496 pg TEQ/g liver tissue	In general, carp diets caused impaired reproduction and/or reduced survival of kits; compared to controls. Kits body weight was significantly reduced in the 20 and 40% carp groups; kit body weight and survival in the 10 and 20% carp groups were significantly reduced at three and six weeks of age. Females fed 40% carp whelped the fewest number of kits, all of which were stillborn or died within 24 hours. Weight of kits and % kit survival to age 6 weeks were inversely proportional to % carp in mothers'diets; physical abnormalities and a dose-related decrease in relative organ weights were also observed in kits				[43]	L; concentration of total PCBs in carp /percent carp in diet per treatment group: 0.015 mg-PCBs/kg-diet/0% 0.72 mg-PCBs/kg-diet/10% 1.53 mg-PCBs/kg-diet/20% 2.56 mg-PCBs/kg-diet/40%; carp also contained 2,3,7,8-TCDD with resulting diet concentrations of 1.03, 19.41, 40.02, and 80.76 ng-TEQs/kg diet in the 0, 10, 20, and 40% diet exposures; mink exposed prior to and throughout reproductive period

[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] Wet weight calculated assuming a dry weight of 25% of the total weight in paper.
[5] 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: PESTICIDE (ORGANOCHLORINE)

Chemical Name (Common Synonyms): TOXAPHENE **CASRN:** 8001-35-2

Chemical Characteristics

Solubility in Water: 3.0 mg/L at room temperature [1].

Half-Life: No data [2]

Log K_{ow}: 5.50 [3]

Log K_{oc}: 5.41 L/kg organic carbon

Human Health

Oral RfD: 3.6×10^{-4} mg/kg/day [4]

Confidence: Not available, uncertainty factor = 100.

Critical Effect: Hepatocellular tumors in mice and thyroid tumors in rats

Oral Slope Factor: $1.1 \times 10^{+0}$ per(mg/kg)/day [5]

Carcinogenic Classification: B2 [5]

Wildlife

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

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

Aquatic Organisms

Partitioning Factors: Toxaphene is a complex mixture of more than 180 chlorinated bornanes. The composition of toxaphene which changes markedly appears to be caused by chemical transformation processes [6]. Toxaphene persistence and degradation in water and biota is modified by numerous and disparate biological and abiotic factors [7]. In lakes, toxaphene persistence was related to depth, stratification, and turnover. Toxaphene can persist in water from several months to more than nine years [8]. Log BCFs for toxaphone ranged from 3.52 for white mullet [10] to 4.72 for fathead minnow [7], as reported in the following table.

Food Chain Multipliers: Biomagnification of toxaphene was demonstrated in 16 species collected in lakes in northeastern Louisiana [9]. The highest residues (1.7 to 5.5 mg/kg ww) were measured among tertiary consumers, such as green-backed heron, spotted gar, and largemouth bass. Secondary consumers (bluegill, blacktail shiner) contained lower toxaphene residues (0.9 to 1.2 mg/kg ww), whereas primary consumers, including crayfish and shad, contained lowest levels (0.6 to 1.0 mg/kg ww).

Toxicity/Bioaccumulation Assessment Profile

Since toxaphene represents a complex mixture of nearly 200 compounds, it is difficult to relate observed toxicity to a specific complex of toxaphene compounds. Fewer than 6 percent of the total number of toxaphene components have been isolated and individually examined for toxicity [10]. Isensee et al. [11] separated toxaphene into nine fractions on a silica gel column. Only the first two fractions and last two fractions revealed reduced toxicity compared with the unfractionated toxaphene, while the middle five fractions were as toxic as or more toxic than the original pesticide. Although chlorinated hydrocarbons have low solubility in water, they are readily absorbed by oils, waxes, and fats [12]. Therefore, toxaphene is generally more toxic to aquatic organisms than are other insecticides and herbicides. Acute toxicity for freshwater fish species range from 3 to 50 µg/L [13]. A concentration as low as 5 µg/L toxaphene can reduce a population of small fish in lakes without affecting the population of large fish [14]. Freshwater fishes of the Arroyo Colorado accumulated up to 31.5 mg/kg wet weight while fish-eating birds contained only up to 3 mg/kg of toxaphene [15]. Unlike fishes, avian species readily metabolize and excrete toxaphene.

Toxaphene compounds have been found in environmental samples and tissues in the Canadian Northern Territories. The toxicity of toxaphene components present in fish and mammals from Yukon Territory is unknown [16]. Toxaphene components are present in northern animals in concert with a suite of other organic contaminants, but neither the risks to the animals bearing the residues nor the risks to people consuming the animals are known.

Summary of Biological Effects Tissue Concentrations for Toxaphene

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, Eastern oyster					4.52			[7]	F
Crassostrea virginica, Eastern oyster			85 mg/kg (whole body)[4]	Growth, ED27				[18]	L; tissue analyses on survivors
			47 mg/kg (whole body)[4]	Growth, ED34				[18]	L; tissue analyses on survivors
			199 mg/kg (whole body)[4]	Growth, ED64				[18]	L; tissue analyses on survivors
			409 mg/kg (whole body)[4]	Growth, ED96				[18]	L; tissue analyses on survivors
Palaemonetes pugio, Grass shrimp			2.7 mg/kg (whole body)[4]	Mortality, ED25				[18]	L; tissue analyses on survivors
			3.3 mg/kg (whole body)[4]	Mortality, ED53				[18]	L; tissue analyses on survivors
			9.7 mg/kg (whole body)[4]	Mortality, ED68				[18]	L; tissue analyses on survivors

Summary of Biological Effects Tissue Concentrations for Toxaphene

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]**
			4.8 mg/kg (whole body)[4]	Mortality, ED70				[18]	L; tissue analyses on survivors
			8.1 mg/kg (whole body)[4]	Mortality, ED75				[18]	L; tissue analyses on survivors
Penaeus duorarum, Pink shrimp			0.36 mg/kg (whole body)[4]	Mortality, ED15				[18]	L; tissue analyses on survivors
			0.54 mg/kg (whole body)[4]	Mortality, ED20				[18]	L; tissue analyses on survivors
			0.83 mg/kg (whole body)[4]	Mortality, ED65				[18]	L; tissue analyses on survivors
			1.7 mg/kg (whole body)[4]	Mortality, ED90				[18]	L; tissue analyses on survivors
Fishes									
Salvelinus fontinalis, Brook trout					4.00			[7]	F

Summary of Biological Effects Tissue Concentrations for Toxaphene

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]**
Salvelinus fontinalis, Brook trout			1 mg/kg (whole body)[4]	Development, LOED				[21]	L; backbone development adversely affected, collagen content decreased
			3.7 mg/kg (whole body)[4]	Development, LOED				[21]	
			0.4 mg/kg (whole body)[4]	Growth, LOED				[21]	L; reduced growth of fry
			0.6 mg/kg (whole body)[4]	Growth, LOED				[21]	
			9.2 mg/kg (whole body)[4]	Development, NA				[21]	L; backbone development adversely affected, collagen content decreased
			38 mg/kg (whole body)[4]	Development, NA				[21]	L; backbone development adversely affected, collagen content decreased
			4.5 mg/kg (whole body)[4]	Development, NA				[21]	

Summary of Biological Effects Tissue Concentrations for Toxaphene

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]**
			18 mg/kg (whole body)[4]	Development, NA				[21]	L; backbone development adversely affected, collagen content decreased
			2.2 mg/kg (whole body)[4]	Development, NA				[21]	
			8.3 mg/kg (whole body)[4]	Development, NA				[21]	
			1.8 mg/kg (whole body)[4]	Growth, NA				[21]	L; reduced growth of fry
			2.6 mg/kg (whole body)[4]	Growth, NA				[21]	L; reduced growth of fry
			0.9 mg/kg (whole body)[4]	Growth, NA				[21]	
			1.4 mg/kg (whole body)[4]	Growth, NA				[21]	
			0.2 mg/kg (whole body)[4]	Development, NOED				[21]	L; no effect on backbone development
			2.6 mg/kg (whole body)[4]	Development, NOED				[21]	
Pimephales promelas, Fathead minnow					52000			[7]	F

Summary of Biological Effects Tissue Concentrations for Toxaphene

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			5.9 mg/kg (whole body)[4]	Development, LOED				[20]	L; significant reduction in bone development, bone collagen in 150 days
			5.9 mg/kg (whole body)[4]	Growth, LOED				[20]	L; significant reduction in growth, both length and weight
			52 mg/kg (whole body)[4]	Mortality, LOED				[20]	L; increased mortality after 150 days
			13 mg/kg (whole body)[4]	Development, NA				[20]	L; significant reduction in bone development, bone collagen in 150 days
			22 mg/kg (whole body)[4]	Development, NA				[20]	
			52 mg/kg (whole body)[4]	Development, NA				[20]	
			13 mg/kg (whole body)[4]	Growth, NA				[20]	L; significant reduction in growth, both length and weight

Summary of Biological Effects Tissue Concentrations for Toxaphene

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]**
			22 mg/kg (whole body)[4]	Growth, NA				[20]	L; significant reduction in growth, both length and weight
			52 mg/kg (whole body)[4]	Growth, NA				[20]	L; significant reduction in growth, both length and weight
Cyprinodon variegatus, Sheepshead minnow					4.32-4.49			[17]	L
Cyprinodon variegatus, Sheepshead minnow			4.1 mg/kg (whole body)[4]	Mortality, ED25				[18]	L; tissue analyses on survivors
			35 mg/kg (whole body)[4]	Mortality, ED85				[18]	L; tissue analyses on survivors
			2.4 mg/kg (whole body)[4]	Mortality, NOED				[18]	L; tissue analyses on survivors
Cyprinodon variegatus, Sheepshead minnow			10 mg/kg (whole body)[4]	Behavior, LOED				[22]	L; decreased swimming activity

Summary of Biological Effects Tissue Concentrations for Toxaphene

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]**
			36 mg/kg (whole body)[4]	Behavior, NA				[22]	L; decreased swimming activity
			36 mg/kg (whole body)[4]	Mortality, NA				[22]	L; 90% mortality in 28 days
			10 mg/kg (whole body)[4]	Mortality, NOED				[22]	L; no effect on mortality
Fundulus similis, Longnose killifish					4.59			[17]	L
Fundulus similis, Longnose killifish			19.3 mg/kg (whole body)[4]	Mortality, ED15				[18]	L; fish are fry (test 2)
			10 mg/kg (whole body)[4]	Mortality, ED17				[18]	L; fish are fry (test 1)
			0.9 mg/kg (whole body)[4]	Mortality, ED25				[18]	L; fish are adults
			46.6 mg/kg (whole body)[4]	Mortality, ED35				[18]	L; fish are fry (test 2)
			24.7 mg/kg (whole body)[4]	Mortality, ED35				[18]	L; fish are juveniles
			34 mg/kg (whole body)[4]	Mortality, ED53				[18]	L; fish are fry (test 1)

Summary of Biological Effects Tissue Concentrations for Toxaphene

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]**
			102 mg/kg (whole body)[4]	Mortality, ED95				[18]	L; fish are juveniles
			0.5 mg/kg (whole body)[4]	Mortality, NOED				[18]	L; fish are adults
			8 mg/kg (whole body)[4]	Mortality, NOED				[18]	L; fish are fry (test 1)
			8.8 mg/kg (whole body)[4]	Mortality, NOED				[18]	L; fish are fry (test 2)
Leiostomus xanthurus, Spot		0.7 µg/L	2.9 µg/g wet wt		3.61			[10]	L
		0.8 µg/L	0.9 µg/g wet wt					[10]	L
		2.4 µg/L	8.4 µg/g wet wt					[10]	L
Mugil curema, White mullet		0.7 µg/L	4.0 µg/g wet wt		3.76			[10]	L
		0.8 µg/L	2.6 µg/g wet wt		3.52			[10	L
		2.4 µg/L	10.4 µg/g wet wt		3.63			[10]	L
		4.1 µg/L	27.0 µg/g wet wt		3.82			[10]	L
Lagodon rhomboides, Pinfish			1.9 mg/kg (whole body)[4]	Mortality, ED25				[18]	L; tissue analyses on survivors

Summary of Biological Effects Tissue Concentrations for Toxaphene

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]**
			1.6 mg/kg (whole body)[4]	Mortality, NOED				[18]	L; tissue analyses on survivors
Ictalurus punctatus, Channel catfish			1.2 mg/kg (whole body)[4]	Cellular, LOED				[19]	L; skin and liver lesions
			1.8 mg/kg (whole body)[4]	Growth, LOED				[19]	L; reduction in growth
			1.2 mg/kg (whole body)[4]	Physiological, LOED				[19]	L; hepatic enzyme induction
			0.8 mg/kg (whole body)[4]	Cellular, NA				[19]	L; skin and liver lesions
			1.8 mg/kg (whole body)[4]	Cellular, NA				[19]	L; skin and liver lesions
			14 mg/kg (whole body)[4]	Cellular, NA				[19]	L; skin and liver lesions
			5.4 mg/kg (whole body)[4]	Cellular, NA				[19]	L; skin and liver lesions
			14 mg/kg (whole body)[4]	Growth, NA				[19]	L; reduction in growth
			5.4 mg/kg (whole body)[4]	Growth, NA				[19]	L; reduction in growth

Summary of Biological Effects Tissue Concentrations for Toxaphene

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]
			0.8 mg/kg (whole body)[4]	Physiological, NA				[19]	L; hepatic enzyme induction
			1.8 mg/kg (whole body)[4]	Physiological, NA				[19]	L; hepatic enzyme induction
			14 mg/kg (whole body)[4]	Physiological, NA				[19]	L; hepatic enzyme induction
			5.4 mg/kg (whole body)[4]	Physiological, NA				[19]	L; hepatic enzyme induction
			1.2 mg/kg (whole body)[4]	Growth, NOED				[19]	L; no effect on growth
			0.8 mg/kg (whole body)[4]	Growth, NOED				[19]	L; no effect on growth

[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: METAL

Chemical Name (Common Synonyms): ZINC **CASRN:** 7440-66-6

Chemical Characteristics

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

Log K_{ow}: — **Log K_{oc}:** —

Human Health

Oral RfD: 3×10^{-1} mg/kg/day [2] **Confidence:** Medium, uncertainty factor = 3

Critical Effect: 47 percent decrease in erythrocyte superoxide dismutase concentration, also decreased serum ferritin and hematocrit values, in adult human females after 10 weeks of zinc exposure; lowered HDL-cholesterol values in human males after several weeks of zinc exposure

Oral Slope Factor: No data [2] **Carcinogenic Classification:** D [2]

Wildlife

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

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

Aquatic Organisms

Partitioning Factors: Zinc in the water column can partition to dissolved and particulate organic carbon. Water hardness (i.e., calcium concentration), pH, and metal speciation are important factors in controlling the water column concentrations of zinc since the divalent zinc ion is believed to be responsible for observed biological effects [17]. Bioavailability of zinc in sediments is controlled by the AVS concentration [18].

Food Chain Multipliers: Most studies reviewed contained data which suggest that zinc is not a highly mobile element in aquatic food webs, and there appears to be little evidence to support the general occurrence of biomagnification of zinc within marine or freshwater food webs [3]. A log biomagnification factor of 2.90 was determined for the midge *Chironomus riparius* [3].

Toxicity/Bioaccumulation Assessment Profile

Zinc does not appear to be a highly mobile element under typical conditions in most aquatic habitats. Tissue residue-toxicity relationships can also be variable because organisms sequester metals in different forms that are measurable as tissue residue but can actually be stored in unavailable forms within the organism as a form of detoxification [4,5]. Whole-body residues also might not be indicative of effects concentrations at the organ level because concentrations in target organs, such as the kidneys and liver, can be 20 times greater than whole body residues [6]. The application of "clean" chemical analytical and sample preparation techniques is also critical in the measurement of metal tissue residues. After evaluating the effects of sample preparation techniques on measured concentrations of metals in the edible tissue of fish, Schmitt and Finger [7] concluded that there was little direct value in measuring copper, zinc, iron, or manganese tissue residues in fish because they do not bioaccumulate to any appreciable extent. It has also been suggested that there is no compelling evidence to support inordinate concern about zinc as a putative toxic agent in the environment, and in fact there is considerable evidence that zinc deficiency is a serious, worldwide human health problem that outweighs the potential problems associated with accidental, self-imposed, or environmental exposure to zinc excess [8].

Summary of Biological Effects Tissue Concentrations for Zinc

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									
Invertebrates, field-collected	Total SEM µg/g µg/g							[9]	F
	10,100 8,873	18900 µg/L	1665 µg/g						
	911 700	1180 µg/L	304 µg/g						
	631 408	187 µg/L	293 µg/g						
	734 562	189 µg/L	453 µg/g						
	365 294	132 µg/L	359 µg/g						
	29 <15	<70 µg/L	212 µg/g						
Tubificidae, Oligochaete worm	2,560 µg/g		203.1 mg/g					[10]	F
	1,110 µg/g		113.9 mg/g						
	3,180 µg/g		264.1 mg/g						
	3,210 µg/g		393.4 mg/g						
	2,550 µg/g		256.6 mg/g						
Nereis diversicolor, Polychaete worm	339 µg/g		199 µg/g					[11]	F
	140 µg/g		163 µg/g						
	99 µg/g		176 µg/g						
	122 µg/g		155 µg/g						
	518 µg/g		185 µg/g						
	532 µg/g		194 µg/g						
	2,237 µg/g								

Summary of Biological Effects Tissue Concentrations for Zinc

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]**
Elliptio complanata, Freshwater mussel	1.5-78.4 µg/g		127 µg/g (foot) 83 µg/g (muscle) 78 µg/g (visceral) 123 µg/g, (hepato-pancreas) 265 µg/g (gills) 173 µg/g (mantle)					[12]	F
	19.1-342 µg/g		144 µg/g (foot) 88 µg/g (muscle) 90 µg/g (visceral) 119 µg/g (hepato-pancreas) 790 µg/g (gills) 275 µg/g (mantle)					[12]	F
	16-433 µg/g		148 µg/g (foot) 119 µg/g (visceral) 208 µg/g (hepato-pancreas) 1360 µg/g (gills) 1190 µg/g (mantle)					[12]	F
Mytilus edulis, Mussel			130 mg/kg (whole body)[4]	Mortality, ED100				[21]	L; 100% mortality in 14 days
Mytilus galloprovincialis, Mussel			14 -20 mg/kg				0.145	[19]	F

Summary of Biological Effects Tissue Concentrations for Zinc

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]**
Dreissena polymorpha, Zebra mussel			21.6 mg/kg (whole body)[4]	Physiological, NOED				[25]	L; no effect on internal zinc regulatory process
			600 mg/kg (whole body)[4]	Mortality, LOED				[26]	L; increased mortality
			130 mg/kg (whole body)[4]	Physiological, LOED				[26]	L; reduced filtration rate
			600 mg/kg (whole body)[4]	Physiological, NA				[26]	L; reduced filtration rate
			22 mg/kg (whole body)[4]	Growth, NOED				[26]	L; no effect on weight gain of surviving mussels
			40 mg/kg (whole body)[4]	Growth, NOED				[26]	L; no effect on weight gain of surviving mussels
			46 mg/kg (whole body)[4]	Growth, NOED				[26]	L; no effect on weight gain of surviving mussels
			130 mg/kg (whole body)[4]	Growth, NOED				[26]	L; no effect on weight gain of surviving mussels
			600 mg/kg (whole body)[4]	Growth, NOED				[26]	L; no effect on weight gain of surviving mussels
			22 mg/kg (whole body)[4]	Mortality, NOED				[26]	L; no effect on mortality

Summary of Biological Effects Tissue Concentrations for Zinc

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]**
			40 mg/kg (whole body)[4]	Mortality, NOED				[26]	L; no effect on mortality
			46 mg/kg (whole body)[4]	Mortality, NOED				[26]	L; no effect on mortality
			130 mg/kg (whole body)[4]	Mortality, NOED				[26]	L; no effect on mortality
			22 mg/kg (whole body)[4]	Physiological, NOED				[26]	L; no effect on filtration rate
			40 mg/kg (whole body)[4]	Physiological, NOED				[26]	L; no effect on filtration rate
			46 mg/kg (whole body)[4]	Physiological, NOED				[26]	L; no effect on filtration rate
Daphnia magna, Cladoceran			1340 mg/kg (whole body)[4]	Reproduction, ED10				[13]	L; 10% reduction in number of offspring
			2690 mg/kg (whole body)[4]	Mortality, ED50				[13]	L; lethal body burden after 21-day exposure
Hyallella azteca, Amphipod		13.0 µg/L	66 µg/g	50% survival				[14]	L
		21.2 µg/L	85 µg/g	56% survival					
		42.3 µg/L	126 µg/g	51% survival					
		185 µg/L	136 µg/g	35% survival					
		316 µg/L	167 µg/g	6% survival					
			167 µg/g	3% survival					

Summary of Biological Effects Tissue Concentrations for Zinc

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]**
	Total SEM µg/g µg/g 10100 8873 911 700 631 408 734 562 365 294 29 <15	 18900 µg/L 1180 µg/L 187 µg/L 189 µg/L 132 µg/L <70 µg/L	 259 µg/g 106 µg/g 80 µg/g 79 µg/g 74 µg/g 56 µg/g					[9]	F
			71.4 mg/kg (whole body)[4]	Mortality, NA				[21]	L; 7.5% mortality in 14 days
Balanus crenatus, Barnacle			3200 mg/kg (whole body)[4]	Behavior, NOED				[28]	L; regulation of metals endpoint - winter experiment
Chironomus riparius, Midge		0.9 mg/L	710 µg/g					[3]	L
Chironomus gr. thummi, Midge			42.89 mg/kg 16.22 mg/kg	Normal larvae Deformed larvae				[15]	L
			61.6 mg/kg (whole body)[4]	Morphology, NOED				[24]	L; 4th instar larvae

Summary of Biological Effects Tissue Concentrations for Zinc

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									
Oncorhynchus mykiss, Rainbow trout			40 mg/kg (whole body)[4]	Physiological, LOED				[20]	L; induction of metallothionein
Salvelinus fontinalis, Brook trout			22.6 mg/kg (whole body)[4]	Reproduction, LOED				[23]	L; reduction in percentage of eggs hatching in second generation trout
			30 mg/kg (gill)[4]	Growth, NOED				[23]	L; no effect on growth
			30 mg/kg (gill)[4]	Growth, NOED				[23]	L; no effect on growth
			30 mg/kg (gill)[4]	Growth, NOED				[23]	L; no effect on growth
			50 mg/kg (liver)[4]	Mortality, NOED				[23]	L; no effect on survival
			50 mg/kg (liver)[4]	Mortality, NOED				[23]	L; no effect on survival
			50 mg/kg (liver)[4]	Mortality, NOED				[23]	L; no effect on survival
			7 mg/kg (kidney)[4]	Reproduction, NOED				[23]	L; no effect on number of eggs produced

Summary of Biological Effects Tissue Concentrations for Zinc

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]**
			7 mg/kg (kidney)[4]	Reproduction, NOED				[23]	L; no effect on number of eggs produced
			7 mg/kg (kidney)[4]	Reproduction, NOED				[23]	L; no effect on number of eggs produced
			19.3 mg/kg (whole body)[4]	Reproduction, NOED				[23]	L; no reduction in percentage of eggs hatching in second generation trout
			15.3 mg/kg (whole body)[4]	Reproduction, NOED				[23]	L; no reduction in percentage of eggs hatching in second generation trout
			6.7 mg/kg (whole body)[4]	Reproduction, NOED				[23]	L; no reduction in percentage of eggs hatching in second generation trout
Salvelinus namaycush, Lake trout			6 cpm/g (whole) 17 cpm/g (spleen) 30 cpm/g (liver) 21 cpm/g (kidney) 9 cpm/g (brain) 32 cpm/g (gonad) 4 cpm/g (muscle) 8 cpm/g (blood) 11 cpm/g (gill) 80 cpm/g (gut)					[16]	F

Summary of Biological Effects Tissue Concentrations for Zinc

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]**
Salmo salar, Atlantic Salmon			60 mg/kg (whole body)[4]	Physiological, LOED				[22]	L; reduced caloric content of fish
			60 mg/kg (whole body)[4]	Growth, NOED				[22]	L; no effect on growth
			42 mg/kg (whole body)[4]	Growth, NOED				[22]	L; no effect on growth
			37 mg/kg (whole body)[4]	Growth, NOED				[22]	L; no effect on survivorship
			60 mg/kg (whole body)[4]	Mortality, NOED				[22]	L; no effect on survivorship
			42 mg/kg (whole body)[4]	Mortality, NOED				[22]	L; no effect on survivorship
			37 mg/kg (whole body)[4]	Mortality, NOED				[22]	L; no effect on survivorship
			42 mg/kg (whole body)[4]	Physiological, NOED				[22]	L; no effect on caloric content of fish
			37 mg/kg (whole body)[4]	Physiological, NOED				[22]	L; no effect on caloric content of fish

Summary of Biological Effects Tissue Concentrations for Zinc

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	770 µg/g 2560 µg/g 1110 µg/g 2180 µg/g 3180 µg/g 3210 µg/g 3120 µg/g 2550 µg/g 2050 µg/g		320.0 mg/g 251.5 mg/g 300.2 mg/g 268.3 mg/g 402.0 mg/g 264.6 mg/g 378.8 mg/g 366.8 mg/g 333.0 mg/g 314.7 mg/g					[10]	F
Jordanella floridae, American flagfish			50 mg/kg (whole body)[4]	Mortality, LOED				[29]	L; body burden estimated from graph
			58 mg/kg (whole body)[4]	Growth, NOED				[29]	L; body burden estimated from graph
			50 mg/kg (whole body)[4]	Mortality, NOED				[29]	L; body burden estimated from graph
			58 mg/kg (whole body)[4]	Reproduction, NOED				[29]	L; body burden estimated from graph
			220 mg/kg (whole body)[4]	Growth, LOED				[30]	L; body burden estimated from graph, total length of females

Summary of Biological Effects Tissue Concentrations for Zinc

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]**
			300 mg/kg (whole body)[4]	Growth, LOED				[30]	L; body burden estimated from graph, total length of males
			220 mg/kg (whole body)[4]	Mortality, LOED				[30]	L; body burden estimated from graph
			230 mg/kg (whole body)[4]	Growth, NOED				[30]	L; body burden estimated from graph, total length of males
			190 mg/kg (whole body)[4]	Growth, NOED				[30]	L; body burden estimated from graph, total length of females
			300 mg/kg (whole body)[4]	Mortality, NOED				[30]	L; body burden estimated from graph
			220 mg/kg (whole body)[4]	Mortality, NOED				[30]	L; body burden estimated from graph
Poecilia reticulata, Guppy			0.284 mg/kg (whole body)[4]	Mortality, NOED				[27]	L

[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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