APPENDIX D

ECOTOXICITY OF SUBSTANCES OF CONCERN FOUND IN COMMENCEMENT BAY

POLYCYCLIC AROMATIC HYDROCARBONS (PAHs)

PAH compounds are a class of relatively persistent environmental contaminants with low water solubilities (0.0003 to 34 mg/l) and log octanol/water partition coefficients ranging between 3.4 and 7.6. Because of their relatively low water solubility, most PAHs are strongly adsorbed to particulate matter in aquatic systems and can accumulate in sediments and biota (Clement Associates, 1985). As a result, PAHs can be found at considerable distances from their source, as fine particles containing PAHs are transported by prevailing water currents (PTI, 1988b). Although relatively insoluble in water, the dissolved portion may undergo rapid, direct photolysis. Singlet oxygen is the oxidant, and quinones are the products in these reactions. Oxidation by chlorine and ozone may be an important fate process when these oxidants are available in sufficient concentrations (Clement Associates, 1985).

In general PAH compounds are rapidly bioaccumulated, but are also quickly metabolized and eliminated from most organisms (shellfish are a known exception). Bioaccumulation, especially in vertebrate organisms, is usually short term, so it is not considered an important fate process in multicellular organisms. Biodegradation and biotransformation are probably the ultimate fate processes for PAHs. The available data suggest that the PAH compounds with high molecular weights are degraded slowly by microbes and readily metabolized by multicellular organisms. Biodegradation probably occurs more slowly in aquatic systems than in soil (Clement Associates, 1985).

Because of the relatively low aqueous solubility of PAHs and their strong affinity for particulate matter in aquatic systems, sediments are the primary reservoir for PAHs. Once in the sediments, PAHs are subjected to burial, resuspension, and degradation. The lower molecular weight PAHs (three aromatic rings or less), because of their greater aqueous solubility and degradability, tend to be lost in the weathering process so that the less labile, higher molecular weight PAHs tend to predominate in sediments.

Based on their molecular weights, the PAHs have been classified as low molecular weight PAHs (LPAH) and high molecular weight PAHs (HPAH). The major LPAHs are: acenaphthene, acenaphthylene, anthracene, fluorene, 2-methylnaphthalene, naphthalene, and

phenanthrene. The major HPAHs are: benz(a)anthracene, benzo(a)pyrene, the benzofluoranthenes, benzo(g,h,i)perylene, chrysene, dibenzo(a,h)anthracene, fluoranthene, and pyrene.

Bioaccumulation

Although PAHs are rapidly bioaccumulated, they are also quickly metabolized and eliminated from most aquatic organisms (shellfish are a known exception) (Eisler, 1987). Uptake of PAHs is highly species specific, greater in algae, mollusks, and other species which are incapable of metabolizing PAH compounds. Bioconcentration factors (BCFs) tend to increase with increasing molecular weight of the PAH compounds, increasing octanol/water partition coefficient values, time (approaching an apparent equilibrium level) increasing dissolved organic matter in the medium, and with increasing lipid content of the organism. A variety of endogenous and exogenous factors can also influence the BCFs (Eisler, 1987).

Bioaccumulation, especially in vertebrate organisms, is usually short term, so it is not considered an important fate process in multicellular organisms. Accumulation by oysters (*Crassostrea virginica*) and clams (*Rangia cuneata*) of naphthalene, phenanthrene, fluorene, and their methylated derivatives increased with increasing methylation and molecular weight. When returned to PAH-free seawater, mollusks released PAHs to non-detectable concentrations within about 60 days, with high molecular weight PAHs depurated more slowly than low molecular weight compounds. Brown shrimp (*Penaeus aztecus*) and longnose killifish (*Fundulus similis*), which have the ability to metabolize PAHs, lost PAHs more quickly than the clams and oysters, which apparently lack the detoxifying enzymes (Neff et al., 1976, as cited in Eisler, 1987).

Time to depurate or biotransform 50 percent of accumulated PAHs ($Tb_{1/2}$) varies widely between organisms. $Tb_{1/2}$ values for *Daphnia pulex* and all PAH compounds studied ranged between 0.4 and 0.5 hours (Southworth et al., 1978, as cited in Eisler, 1987). Some species, such as the hardshell clam, show little or no depuration, while others, such as oysters, eliminated up to 90 percent of accumulated PAHs within two weeks, although the remaining 10 percent was released slowly, and traces may remain indefinitely (Jackim and Lake, 1978, as cited in Eisler, 1987). Teleosts and arthropods usually have low $Tb_{1/2}$ values. In bluegill, 89 percent of benzo(a)pyrene was recorded four hours postexposure; for midge larvae it was

72 percent in eight hours, and for daphnids it was 21 percent in 18 hours (Leversee et al., 1981, as cited in Eisler, 1987).

Aquatic Toxicity

PAHs vary substantially in their toxicity to and bioaccumulation by aquatic organisms. In general, toxicity increases as molecular weight increases and with increasing alkyl substitution on the aromatic ring. In most fish, PAHs are rapidly metabolized and excreted so that concentrations found in edible tissue are generally low. Invertebrates such as mollusks do not metabolize PAHs as efficiently and may accumulate high levels of PAHs in tissue. Toxicity is most pronounced among crustaceans and least among teleosts (Eisler, 1987). The acute toxicities of PAHs have been studied using a number of marine organisms. The 96-hour LC_{50} for exposure to fluorene ranged from 320 µg/l for the grass shrimp (*Palaemonetes pugio*) to 1,680 µg/l in the sheepshead minnow (*Cyprinodon variegatus*) (Finger et al., 1985, as cited in Eisler, 1987). The 96-hour LC_{50} concentration for naphthalene exposure for both of these species was reported to be 2,400 µg/l (Neff, 1979, as reported in Eisler, 1987). The 96-hour LC_{50} concentrations for Coho salmon (*Oncorhyncus kisutch*) and Dungeness crab (*Cancer magister*) exposed to naphthalene are reported to be 3,200 and 2,000 µg/l, respectively (Neff, 1985, as cited in Eisler, 1987).

Cytotoxic, mutagenic, and carcinogenic effects of many PAHs are generally believed to be mediated through active epoxides formed by interaction with microsomal monooxygenases. These highly active arene oxides can interact with macromolecular tissue components and can further be metabolized or rearranged to phenols or various conjugates. They can also be affected by epoxide hydrolase to form dihydrodiols, which are precursors of biologically active diol epoxides, a group that has been implicated as ultimate carcinogens.

Sediment Toxicity

Field experiments testing the colonization of creosote-contaminated sediment by benthic invertebrates indicated that polychaetes and crustaceans were significantly affected at creosote concentrations $\geq$177 mg/kg. PAH compounds are associated with Creosote contamination. Mollusks, however, were apparently more resistant to the effects of creosote contamination, showing effects only at concentrations greater than 844 mg/kg (Tagatz et al., 1983). A laboratory study in which the freshwater amphipod *Diporeia* sp. was exposed to

sediments dosed with a mixture of PAHs reported mortality at total PAH concentrations in the range of 100 mg/kg (dry wt.) after 26 days of exposure (Landrum et al., 1991). The study also reported that the PAH action appears to be additive, with no overt evidence of synergism or antagonism and that the rate of PAH accumulation was dependent upon the concentration of PAHs in the sediment and was not predictable through measured partitioning between interstitial water and sediment particles.

Estuarine fish collected from areas in the Elizabeth River, Virginia, where sediments are contaminated with creosote, showed reduced abundance (reduced total biomass, total numbers of individuals, and abundance of selected species) and increased prevalence of several gross abnormalities that correlated with PAH contamination in the sediments (24,400 to 154,400 µg/kg). Abnormalities observed included fin erosion in hogchoker and toadfish, and cataracts in spot, gray trout and croaker (Huggett et al., 1987). Tumors of thyroid, gonad, skin, and liver have been reported, with tumor frequency greatest near areas contaminated with PAHs. A positive relationship was established between sediment PAH concentrations and prevalence of liver lesions in English sole in Puget Sound (Malins et al, 1984 and Varanasi et al., 1984, as cited in Eisler, 1987).

POLYCHLORINATED BIPHENYLS (PCBS)

As a result of their highly lipophilic nature and low water solubility, PCBs are generally found to have low concentrations in water and relatively high concentrations in sediments. Individual PCB congeners have different physical and chemical properties based on both the degree of chlorination and position of chlorine substitution, although differences in the degree of chlorination are usually more significant (Phillips, 1986). Vapor pressure and water solubility are inversely related to degree of chlorination (Erickson, 1985; Phillips, 1986). 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 may show greater dispersion from a point source, while the higher chlorinated components of a PCB mixture may remain in the sediments closer to the source (Phillips, 1986).

The mobility of PCBs in sediment is a function of the chlorine substitution pattern and degree of chlorination and is generally quite low, particularly for the higher chlorinated biphenyls (Fisher et al., 1983). As a result of this low mobility, in the absence of disturbance of the sediment or bioaccumulation, even low rates of sedimentation may prevent PCBs in the sediment from reaching the overlying water via diffusion (Fisher et al., 1983). The measurement of PCB concentrations in sediment is complicated by their non-uniform distribution on both vertical and horizontal scales.

PCB concentrations are also affected by physical characteristics of the sediment such as grain size and total organic carbon content (Pavlou and Dexter, 1979; Lynch and Johnson, 1982). Fine sediments generally contain higher concentrations of PCBs than coarser sediments, probably as a result of larger surface area on finer sediments (Phillips, 1986). The amount of PCB sorbed to sediments is also a function of the total organic carbon content of the sediment (Chou and Griffin, 1986; Sawhney, 1986).

The persistence of PCBs in the environment is a result of their general resistance to degradation. The rate of degradation of PCB congeners by bacteria decreases with increasing degree of chlorination (Furukawa, 1986); other structural characteristics of the individual PCBs also affected susceptibility to microbial degradation to a lesser extent. 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 (Sawhney, 1986). In addition, reductive dechlorination of the higher chlorinated PCB congeners by anaerobic bacteria in sediments has been reported (Brown et al., 1987). The overall significance of the different degradation pathways in terms of reduction of environmental PCB concentrations is not clear at this time.

Bioaccumulation

Bioaccumulation factors (BAF: biota [PCB]/sediment [PCB]) in freshwater invertebrates ranged from 1 to about 24, whereas BAFs in freshwater fish ranged from 0.13 to as high as 30. Accumulation of PCBs in the tissues of aquatic organisms can occur through uptake from water, sediment, and food. However, in top predators feeding higher in the food web, PCB exposure through the food chain may account for greater than 99 percent of observed tissue concentrations (Thomann and Connolly, 1984). It has been demonstrated that bioaccumulation of PCBs in higher trophic level fish was greater than predicted from bioconcentration from water alone, indicating that contaminated food is a major source of PCB residues in these fish (Oliver and Niimi, 1988).

An additional source of PCBs in some organisms may be through maternal transfer of PCBs to eggs. Ankley et al. (1991) demonstrated that concentrations of polychlorinated aromatic hydrocarbons were 5-fold greater in eggs than in the flesh of spawning Chinook salmon (*Oncorhynchus tschawytscha*).

Aquatic Toxicity

The acute toxicity of PCBs to sensitive aquatic organisms (expressed as LC_{50} concentrations) is reported to vary from 0.1 to 10 µg/l during exposures of 7 to 38 days. In general, toxicity increased with increasing exposure. Crustaceans and younger developmental stages are the most sensitive groups tested, and lower chlorinated biphenyls are more toxic than higher chlorinated biphenyls (Eisler, 1986). The 96-hour LC_{50} concentrations for grass shrimp (*Palaemonetes pugio*) and sheepshead minnow exposed to Aroclor 1254 are reported to be 6.1 to 7.8 and 0.1 to 0.32 µg/l, respectively (Ernst, 1984 as cited in Eisler, 1986).

PCBs elicit a variety of sublethal toxic effects including skin lesions, immunotoxicity, reproductive toxicity, genotoxic and epigenetic effects, hepatomegaly and related liver damage, and the induction of hepatic and extrahepatic drug-metabolizing enzymes. Interspecies differences in sensitivity to PCBs are large, even between species that are closely related taxonomically (Eisler, 1986).

Sediment Toxicity

PCBs in sediments can provide a significant source of contamination for aquatic organisms (Pruell et al., 1993; Ankley et al., 1992; Connolly, 1991; Lake, 1990; Larsson, 1986). Bioaccumulation of PCBs from contaminated sediments may result from uptake from the interstitial or overlying water (via respiration), direct dermal sorption, or indirectly through the food web (Swartz and Lee, 1980; Rubenstein et al., 1984). The availability of PCBs in sediments to aquatic organisms depends on the concentrations of the specific PCBs present, physical properties of the sediment, environmental factors, and characteristics of the organisms. The presence of other contaminants may also influence the availability of PCBs (Shaw and Connell, 1982).

There is relatively limited data regarding the toxicity of sediment-associated PCBs to aquatic organisms. The meiobenthic copepod *Microarthridion littorale* exposed to Aroclor 1254-contaminated sediments was reported to exhibit significant mortality at concentrations of 83,000 µg/kg, whereas lower concentrations (4,000 µg/kg) impaired reproduction (DiPinto et al., 1993).

4,4′-DDD AND 4,4′-DDE

4,4'-DDD (DDD) and 4,4'-DDE (DDE) are metabolites of 4,4'-DDT, a chlorinated hydrocarbon pesticide. DDD and DDE are highly persistent in the environment because of their low vapor pressure and resistance to degradation and photooxidation. Soils and sediments act as a primary reservoir for DDD and DDE. Estimates of the half-life for DDT biodegradation in soil range from 2 to >15 years (Clement, 1992).

Sorption and bioaccumulation are the most important transport processes for DDT and its metabolites. Although it occurs slowly, the ultimate fate process for DDT and DDD is biotransformation to form bis (2-chlorophenyl) methanone. For DDE, direct photolysis is the most important ultimate fate process in the environment, although biotransformation may also be important (Clement Associates, 1985).

Bioaccumulation

DDD and DDE are highly lipophilic, resulting in accumulation in tissues of aquatic organisms and biomagnification through the aquatic food web. The highest residue concentrations are often found in carnivorous predators at the top of the food chain (Clement Associates, 1985).

Aquatic Toxicity

No information was found on the mode of toxicity of DDD and DDE specifically, but DDT is believed to induce toxicity by disrupting ion movement within axons as a result of binding with axonal membrane lipoproteins. This binding of DDT changes the structure of the membrane and allows for continued passage of sodium ions, causing repetitive discharge of nerve impulses after an initial discharge, leading to tremors (Connell and Miller, 1984).

The toxicities of DDD and DDE have not been studied extensively. The 96-hour LC_{50} concentrations for DDD and DDE in the Eastern oyster (*Crassostrea virginica*) are 25 and 14 µg/l, respectively, while that for DDD in the striped bass (*Morone saxatilis*) is 2.5 µg/l (EPA, 1980).

Sediment Toxicity

Few studies have been conducted regarding the direct toxicity of DDD and DDE in sediments; studies are limited primarily to bioaccumulation in the food chain or uptake through the water column. However, it was found that the exposure of mayfly to sediment collected from the Mississippi River containing < 0.2 µg/kg DDE for four days resulted in low survival rates. Exposure to estuarine sediment collected from San Francisco Bay, containing 3 µg/kg DDE and 1 µg/kg DDD, was associated with highly toxic effects on *Rhepoxynius abronius*. Toxic effects to bivalve larvae were observed when exposed to San Francisco Bay sediment with concentrations of 1 µg/kg DDE and 1 µg/kg DDD (Long and Morgan, 1991).

PHENOLS

Phenol

Phenol is not very persistent in the environment. Photooxidation may be an important degradative process, especially in aerated, clear, surface waters. Phenol may also be nonphotolytically oxidized in highly aerated waters that contain iron and copper in solution or as suspended particulates. Biodegradation can be a significant fate pathway in aquatic systems when significant concentrations of microorganisms are present (Clement Associates, 1985).

Bioaccumulation

The relatively low log octanol/water partition coefficient, as well as the available experimental evidence suggest that sorption and bioaccumulation are not important environmental fate processes (Clement Associates, 1985).

Aquatic Toxicity

No information was found regarding the mechanism of action of phenol in aquatic organisms. However, in mammalian systems, phenol acts by denaturing and precipitating cellular proteins (Dreisbach, 1977). The acute toxicity of phenol to aquatic species is expressed over a range of two to three orders of magnitude. Concentrations lethal to fish species range from 5,020 µg/l for juvenile rainbow trout to 67,500 µg/l for the fathead minnow. A concentration of 5,000 µg/l was reported to be lethal to *Daphnia magna*. An early life stage test with the fathead minnow resulted in chronic toxicity at a concentration of 2,560 µg/l. The median effect concentration for oyster and clam embryos is approximately 55,000 µg/l. The LC_{50} concentration for the grass shrimp is 5,800 µg/l (Clement Associates, 1985).

Sediment Toxicity

The only data found that relate sediment concentrations of phenol to toxic effects in aquatic biota were the sediment Apparent Effects Threshold (AET) concentrations developed for Puget Sound which range from 420 to 1,200 µg/kg (dry weight) (PTI, 1988).

2-Methylphenol, 4-Methylphenol, and 2,4-Dimethylphenol

The environmental chemistry of 2-methylphenol, 4-methylphenol, and 2,4-dimethylphenol is probably similar to that of phenol. Photooxidation is probably the primary mechanism for removal of 2,4-dimethylphenol in clear, aerated surface waters, although metal-catalyzed oxidation, sorption, and biodegradation may also play a part in its removal from aquatic systems (Clement Associates, 1985).

Bioaccumulation

No information was found describing the bioaccumulation potential of 2-methylphenol, 4-methylphenol, or 2,4-Dimethylphenol. However, because of their similarity to phenol, it is doubtful that they bioaccumulate to a great extent.

Aquatic Toxicity

No information was found describing the mechanism of action for 2-methylphenol, 4-methylphenol, or 2,4-Dimethylphenol. No toxicity data were found for 2-methylphenol and 4-methylphenol and only limited toxicity information was available for 2,4-Dimethylphenol. Survival of fathead minnow exposed to 2,4-Dimethylphenol for up to four weeks was significantly reduced at a concentration of 5,130 µg/l, while growth was significantly reduced at a concentration of 3,110 µg/l (Holcombe et al., 1982).

Sediment Toxicity

The only data found relating sediment concentrations of 2-methylphenol, 4-methylphenol, and 2,4-Dimethylphenol to effects in aquatic biota are the AET concentrations developed for Puget Sound. The AET concentrations for 2-methylphenol ranged from 63 to >72 µg/kg (dry

weight) while those for 4-methylphenol ranged from 670 to 3,600 µg/kg. The AET concentrations for 2,4-Dimethylphenol ranged from 29 to 210 µg/kg (PTI, 1988).

Pentachlorophenol

Pentachlorophenol in the aquatic environment may be present in a dissolved form, sorbed to suspended matter or sediment, or sorbed to aquatic organisms. Pentachlorophenol is a neutral organic compound in aqueous solutions of pH less than five that becomes increasingly ionized as the pH increases. The degree of dissociation will determine the solubility and degree of adsorption to suspended matter and sediments. Pentachlorophenol is subject to microbial degradation, photodegradation, and volatilization in the aquatic environment. The rate of degradation, however, is dependent on the physical, chemical, and biological characteristics of the aquatic system. Pentachlorophenol is generally considered to be moderately persistent in the aquatic environment (EPA, 1980).

Pierce and Victor (1978) studied the fate of pentachlorophenol in an aquatic ecosystem after a spill of wood treatment waste containing pentachlorophenol into a lake. Pentachlorophenol was reported to have persisted for six months after the spill in the water column and was detected in fish tissue. Sediment samples were found to retain high concentrations of pentachlorophenol throughout the two year investigation. However, it is also reported that pentachlorophenol readily degrades in the environment by chemical, microbiological, and photochemical processes (Kaufman, 1978 and Choudhury et al., 1986, as cited in Eisler, 1989).

Bioaccumulation

Pentachlorophenol bioconcentrates in the tissues of aquatic organisms. Pierce and Victor (1978) observed that fish contained concentrations of pentachlorophenol for a period of at least six months following the release of wood treatment waste containing pentachlorophenol into a lake. Bioconcentration factors (BCFs) in fish tissues ranged from 500 for muscle to 8,000 in liver. Holmberg et al. (1972) reported BCFs on the order of 10 to 350 in fish exposed to 0.1 mg/l of pentachlorophenol. The accumulation of pentachlorophenol in aquatic organisms is rapid, and primarily by direct uptake from water rather than through the food chain or diet (Niimi and Cho, 1983, as cited in Eisler, 1989).

Aquatic Toxicity

Pentachlorophenol affects energy metabolism by partly uncoupling oxidative phosphorylation and increasing oxygen consumption by altering the activities of several glycolytic enzymes and citric acid cycle enzymes, and by increasing the consumption rate of stored lipid (Johansen et al., 1985; Brown et al., 1987; McKim et al., 1987, as cited in Eisler, 1987).

Pentachlorophenol has been reported to be acutely toxic to salmonid species at concentrations ranging from 34 to 120 µg/l (Davis and Hoos, 1975). Acute toxicity has been reported in eastern oyster and in grass shrimp larvae at concentrations of 40 and 649 µg/l, respectively (Borthwick and Schimmel, 1978). Chronic toxicity studies in sockeye salmon (*Oncorhynchus nerka*) reported inhibited growth after a six-week exposure to 3.2 µg/l (Webb and Brett, 1973). Studies with experimental ecosystems have indicated that the effects of pentachlorophenol on community structure and activity are profound. These effects included a reduction in the number of species of estuarine macrobenthos after exposure to 55 to 76 µg/l for five to nine weeks (Tagatz et al., 1977 and 1983, as cited in Eisler, 1989).

Sediment Toxicity

In marine sediments in Puget Sound, no significant biological effects have been noted at pentachlorophenol concentrations of 100 µg/kg dry weight (Tetra Tech, 1986). The AET concentrations for pentachlorophenol in Puget Sound range from >140 to 690 µg/kg dry weight (PTI, 1988).

CHLORINATED BENZENES

1,3-Dichlorobenzene, 1,2-Dichlorobenzene, and 1,4-Dichlorobenzene

Relatively little information was found describing the environmental chemistry and toxicology of the dichlorobenzenes. 1,4-Dichlorobenzene tends to volatilize to the atmosphere from soil and water at a relatively rapid rate. The estimated volatilization half-life in a model river was 4.3 hours (Howard, 1990, as cited in Life Systems, 1991) and reported half-lives in coastal seawater ranged from 10 to 18 days (Wakeman et al., 1983, as cited in Life Systems, 1991). 1,4-Dichlorobenzene (300 ppm) volatilized completely from non-aerated distilled water in less than three days and from aerated distilled water in less than four hours (Garrison and Hill, 1972, as cited in Life Systems, 1991). At the termination of an eight-day stability study, 1,2-, 1,3-, and 1,4-Dichlorobenzene were present at 15, 4, and 25 percent of their initial concentrations, respectively (Canton et al., 1985).

Biodegradation may be an important transformation process for 1,4-Dichlorobenzene in water under aerobic, but not anaerobic, conditions (Life Systems, 1991). The half-lives of 1,2-Dichlorobenzene and 1,3-Dichlorobenzene in a non-adapted inoculum were reported to be greater than four weeks while that of 1,4-Dichlorobenzene was reported to be between 2 and 3 weeks (Canton et al., 1985).

Based on measured soil organic carbon partition coefficient values, which range from 275 to 1,833 in different soils, 1,4-Dichlorobenzene is expected to sorb to soils and sediments. Sorption is primarily to the organic phase (Chiou et al., 1983, as cited in Life Systems, 1991). Canton et al. (1985) reported log octanol/water coefficients of 3.5 for each of the dichlorobenzenes, so that 1,2-Dichlorobenzene and 1,3-Dichlorobenzene may also be expected to sorb to sediments.

Bioaccumulation

1,4-Dichlorobenzene is expected to bioconcentrate in aquatic organisms. Measured mean bioconcentration factors of 370-720 for rainbow trout (Oliver and Niimi, 1983, as cited in Life Systems, 1991) and 1,800 for guppies (Chiou, 1985, as cited in Life Systems, 1991) were reported. The theoretical BCFs for the dichlorobenzenes were reported to be 270

(Canton et al., 1985). Based on the log octanol/water coefficients for the dichlorobenzenes, 1,2-Dichlorobenzene and 1,3-Dichlorobenzene can be expected to bioaccumulate as well. Data on biomagnification through the food chain were not located.

Aquatic Toxicity

No information was found on the mechanism of action for the dichlorobenzenes. 1,2-Dichlorobenzene, 1,3-Dichlorobenzene, and 1,4-Dichlorobenzene were reported to have LC_{50} concentrations of 6,000, 6,000, and 4,000 µg/l, respectively, for exposure to the guppy (*Poecilia reticulata*) for 14 days (Canton et al., 1985). The 48-hour LC_{50} concentrations for *Daphnia magna* exposed to 1,2-Dichlorobenzene, 1,3-Dichlorobenzene, and 1,4-Dichlorobenzene were reported to be 2,200, 6,800, and 2,200 µg/l, respectively (Canton et al., 1985). Richter et al. (1983) reported that a 28-day exposure to 1,500 µg/l of 1,3-Dichlorobenzene resulted in a statistically significant reduction in the number of young produced in *Daphnia magna*.

Sediment Toxicity

The AET concentrations for 1,2-Dichlorobenzene range between 35 and >110 µg/kg (dry weight), while those for 1,3-Dichlorobenzene and 1,4-Dichlorobenzene are >170 µg/kg and between 110 and 120 µg/kg, respectively (PTI, 1988).

Hexachlorobenzene

Hexachlorobenzene is persistent in the environment. The major fate of hexachlorobenzene in the environment is probably reversible sorption to organic material in the sediments. Although hexachlorobenzene will be immobilized through sorption of sediments and particulate matter, desorption may produce continuous, low-level concentrations of hexachlorobenzene in the surrounding media. Degradation in the environment occurs very slowly, if at all. The two possible routes of degradation are photolysis, possibly assisted by the presence of photosensitizing organic materials in aquatic environments, and biodegradation (Clement Associates, 1985).

Bioaccumulation

Aquatic biota can accumulate hexachlorobenzene, but it is unclear whether biomagnification occurs through the food web (Clement Associates, 1985).

Aquatic Toxicity

No information was found on the mechanism of action of hexachlorobenzene, however, pentachlorophenol has been identified as one of its degradation products (Johnson and Finley, 1980). Little information was found regarding the aquatic toxicity of hexachlorobenzene. The highest 96-hour LC_{50} for hexachlorobenzene exposure was in coho salmon (*Oncorhynchus tshawytscha*), which was reported to be greater than 50,000 µg/l, while the lowest was in bluegill (*Lepomis macrochirus*) and largemouth bass (*Micropterus salmoides*) at 12,000 µg/l (Johnson and Finley, 1980).

Sediment Toxicity

The AET concentrations for hexachlorobenzene range between 22 and 230 µg/kg dry weight (PTI, 1988).

Hexachlorobutadiene

Hexachlorobutadiene is somewhat persistent in the environment, adsorbing to organic material in the sediments. Volatilization and adsorption to organic particulates are potentially important transport processes (Clement Associates, 1985).

Bioaccumulation

Limited data were available describing the bioaccumulation potential of hexachlorobutadiene. BCFs have been determined in goldfish (*Carassius auratus*) and range from 920 to 2,300 (EPA, 1980). In crayfish (*Procambarus clarkii*), a BCF of 60 has been reported (EPA, 1980). No information was found on the potential for biomagnification through the food web.

Aquatic Toxicity

No information was found on the mechanism of action of hexachlorobutadiene. Static tests conducted with mysid shrimp (*Mysidopsis bahia*), grass shrimp (*Palaemonetes pugio*), pinfish (*Lagodon rhomboides*), and sheepshead minnow (*Cyprinodon variegatus*) resulted in 96-hour LC_{50} concentrations of 59, 32, 399, and 557 µg/l, respectively (EPA, 1980). Behavioral changes were noted in sheepshead minnow and pinfish at concentrations of 240 and 180 µg/l, respectively.

Sediment Toxicity

AET concentrations for Hexachlorobutadiene range between 11 and 270 µg/kg dry weight (PTI, 1988).

PHTHALATE ESTERS

The environmental fate and transport of bis(2-ethylhexyl)phthalate, di-n-butylphthalate, butylbenzyl phthalate, and di-n-methyl phthalate can be largely inferred from data for phthalate esters as a group. Of the phthalate esters, bis(2-ethylhexyl)phthalate is the most thoroughly studied. The phthalate esters hydrolyze in surface waters, but at such slow rates that this process would not be significant under most conditions. Photolysis, volatilization, and oxidation do not appear to be important environmental fate processes, especially in aquatic systems (Clement Associates, 1985).

Adsorption onto suspended and bed sediments, as well as complexation with natural organic substances are the most important environmental transport processes for the phthalate esters. Phthalate esters are commonly found in freshwater and marine sediments (Clement Associates, 1985).

Bioaccumulation

Aquatic biota have been reported to bioaccumulate phthalate esters, and bioaccumulation is considered an important fate process. Because phthalate esters are degraded under most conditions and can be metabolized by multicellular organisms, it is unlikely that long-term bioaccumulation or biomagnification through the food web occurs (Clement Associates, 1985). Mussels (*Mytilus edulis*) exposed to bis(2-ethylhexyl)phthalate for 28 days were reported to have BCFs of 2500. The depuration half-life of bis(2-ethylhexyl)phthalate in the mussels was reported to be 3.5 days (Brown and Thompson, 1982).

Aquatic Toxicity

Limited information was found describing the mechanism of action of the phthalate esters. Shiner perch (*Cymatogaster aggregata*) exposed to butylbenzyphthalate for 96 hours exhibited changes in schooling behavior and color changes at concentrations of 80 and 240 µg/l, respectively, which the authors attributed to possible effects on catecholamines of the central adrenergic nervous system (Ozretich et al., 1983). The 96-hour LC_{50} concentration for shiner perch exposed to butylbenzylphthalate was reported to average 510 µg/l (Ozretich et al., 1983). The 96-hour LC_{50} concentration for English sole (*Parophrys vetululs*) exposed

to butylbenzyphthalate is reported to be between 550 and 660 µg/l, with sublethal effects (loss of equilibrium) occurring at concentrations as low as 100 µg/l (Randall et al., 1983).

The 96-hour LC_{50} concentrations for freshwater fish exposed to di-n-butylphthalate are reported to range from 730 to 2,910 µg/l, while those in freshwater invertebrates are from 2,100 to 10,000 µg/l (Mayer and Sanders, 1973). Exposure of Daphnia *magna* to bis(2-ethylhexyl)phthalate for 21 days resulted in a significant reduction in reproductive potential (Mayer and Sanders, 1973). Fry survival in guppies and zebra fish (*Brachydanio rerio*) was significantly reduced when parental fish were fed diets containing 50 and 100 µg of bis(2-ethylhexyl)phthalate per gram of food during a 90-day exposure period (Mayer and Sanders, 1973).

Larval grass shrimp exposed to 100,000 µg/l of di-n-menthlyphthalate for the entire larval development cycle were reported to have decreased survival and increased intermolt and developmental periods (Laughlin et al., 1978, as cited in EPA, 1980).

Sediment Toxicity

Very little information was available examining the sediment toxicity of phthalate esters. Tagatz et al. (1986) reported that estuarine macrobenthic communities were significantly altered in laboratory and field experiments when sediment concentrations of dibutyl phthalate reach 1,000 µg/g. In a laboratory study, the number of successful hatches of frog eggs exposed for 60 days to sediments spiked with bis(2-ethylhexyl)phthalate was reported to decline with increasing concentrations of bis(2-ethylhexyl)phthalate. Only 50 percent of the eggs hatched when exposed to a concentration of 150 µg/g bis(2-ethylhexyl)phthalate (Larsson and Thurén, 1987). An AET value for di-n-butylphthalate of 20 mg/kg organic carbon was reported for freshwater sediment using a *Hyalella* bioassay (Ecology, 1994). In general, freshwater AETs are lower than those for marine sediments.

BENZYL ALCOHOL

Little information was found on the environmental chemistry of benzyl alcohol. Laboratory studies indicate that benzyl alcohol can undergo microbial biodegradation and photolysis (CIS, 1991a). Based on its molecular structure, benzyl alcohol is not likely to be persistent in the environment.

Bioaccumulation

No information was found on the bioaccumulation potential of benzyl alcohol, but based in its molecular structure, it is unlikely that it would bioaccumulate to any significant degree or biomagnify through the food chain.

Aquatic Toxicity

No information was found on the mechanism of action of benzyl alcohol in aquatic organisms. Relatively little information was found describing its toxicity in aquatic biota. The 96-hour LC_{50} for the Atlantic silverside (*Menidia menidia*) is reported to be 360,000 µg/l while the 48-hour EC_{50} in *Daphnia magna* is reported to be 15,000 µg/l (CIS, 1991a).

Sediment Toxicity

The only information found relating sediment concentrations of benzyl alcohol to effects in aquatic biota are the AET concentrations developed for Puget Sound which range from 57 to 870 µg/kg dry weight (PTI, 1988).

BENZOIC ACID

Relatively little information was found on the environmental chemistry of benzoic acid. Laboratory studies reported biodegradation of benzoic acid by microorganisms. The log octanol/water partition coefficient of 1.87 indicates that benzoic acid would probably not strongly bind to sediments (CIS, 1991b).

Bioaccumulation

Benzoic acid does not bioaccumulate to any significant extent. The BCF in the mosquito fish is reported to be 1.32 while that in *Daphnia magna* 3.25. Benzoic acid does not biomagnify through the food chain (CIS, 1991b).

Aquatic Toxicity

No information was found on the mechanism of action of benzoic acid in aquatic biota. Toxicity information was found only for freshwater organisms. The 96-hour LC_{50} concentration in the guppy (*Gambusia affinis*) is reported to be 180,000 µg/l. This concentration did not result in mortality to the bluegill (*Lepomis macrochirus*). The 24-hour LC_{50} in *Daphnia magna* is reported to be 1,540,000 µg/l (CIS, 1991c).

Sediment Toxicity

The only information found relating sediment concentrations of benzoic acid to effects in aquatic biota are the AET concentrations developed for Puget Sound which range from 650 to 760 µg/kg dry weight (PTI, 1988).

Tetrachloroethane

Relatively little information is available pertaining specifically to the environmental transport and fate of tetrachloroethane. However, predictions concerning these processes can be made based on comparison with similar compounds such as 1,1,1-trichloroethane. Photolysis and oxidation do not appear to be significant aquatic fate processes for tetrachloroethane. The

observed low reactivity of 1,1,1-trichloroethane suggests that hydrolysis of tetrachloroethane would occur too slowly to be an important fate process. Available data indicate that relatively rapid volatilization of tetrachloroethane from surface water can occur. Based on analogy to 1,1,1-trichloroethane, sorption of tetrachloroethane to sediments is not an important process in the aquatic environment. Available information concerning related compounds suggests that biotransformation and biodegradation occur at low rates or not at all (Clement Associates, 1985).

Bioaccumulation

The log octanol/water partition coefficient of 2.56 for tetrachloroethane indicates that bioaccumulation may occur to some extent, but no adequate empirical data were found to support this hypothesis (Clement Associates, 1985).

Aquatic Toxicity

Very limited information was available describing the toxicity of tetrachloroethane to aquatic organisms. Among marine species, the concentrations resulting in acute toxicity to mysid shrimp and sheepshead minnows are reported to be 9,020 and 12,300 µg/l (Clement Associates, 1985).

Sediment Toxicity

No information was found relating sediment concentrations of tetrachloroethane to effects in aquatic biota.

METALS/METALLOIDS

Trace elements are naturally occurring, environmentally persistent substances found in freshwater and marine environments. In natural water systems metals are partitioned among the aqueous and suspended particulate phases and in bed sediments. Trace elements in all three phases can be bioavailable to organisms, but the dissolved form is generally most bioavailable. A number of processes affect metal speciation in natural waters:

- Complexation and chelation by organic ligands
- Inorganic complexation
- Sorption and desorption by hydrous oxides
- Sorption and desorption by organic and inorganic colloids
- Ion exchange
- Precipitation and co-precipitation
- Dissolution
- Isomorphous substitution.

Antimony (Sb)

Sb is present as the soluble oxide or antimonite (+3) salt in most natural waters. In reducing environments, volatile stibine (SbH_3) may be formed. The formation of stibine in bed sediments, which usually provide a reducing environment, may offer a mechanism for remobilization of Sb previously removed from solution. Biomethylation processes may also cause some remobilization of Sb. It is likely that most species of Sb in natural waters are soluble and quite mobile (Clement Associates, 1985).

Bioaccumulation

Bioaccumulation appears to be only a minor fate process for Sb. In a study with bluegill, no detectable bioconcentration of Sb was observed (Clement Associates, 1985).

Aquatic Toxicity

No information was found describing the mechanism of action of Sb in aquatic biota. However, Sb poisoning in mammals has resulted in atrial fibrillation, liver toxicity, and destruction of blood cells (Beliles, 1975). The LC_{50} concentrations for fathead minnow and the EC_{50} concentrations for *Daphnia magna* range between 9,000 and 12,900 µg/l. Chronic concentrations for the fathead minnow and *Daphnia magna* are 1,600 and 5,400 µg/l, respectively (Clement Associates, 1985).

Sediment Toxicity

The only information found relating sediment concentrations of Sb to effects in aquatic biota are the AET concentrations for Sb which range from 150 to 200 mg/kg dry weight (PTI, 1988).

Arsenic (As)

As occurs in four different oxidation states. The +3 and +5 valence states are most common and are easily interconverted. Complexation with organic carbon can be important. As is generally quite mobile in the aquatic environment. Biological activity or highly reducing conditions can produce arsine or methylarsenicals which are highly volatile. Sorption to sediments is an important fate process for As (Clement Associates, 1985).

Bioaccumulation

As is accumulated from water by a variety of aquatic organisms, however, there is no evidence of magnification along the aquatic food chain (Michnowicz and Weaks, 1984; Dryant et al., 1985; Sanders, 1986, as cited in Eisler, 1988a). The BCF values did not exceed 17 for inorganic As^{+3} in most invertebrates and fish exposed for 21 to 30 days. A BCF of

350 was reported for the American oyster (*Crassostrea virginica*) held in 5 µg As^{+3}/L for 112 days (Zaroogian and Hoffman, 1982, as cited in Eisler, 1988a).

Aquatic Toxicity

The primary toxic mode of inorganic As^{+3} is through reaction with sulfhydryl groups of proteins and subsequent enzyme inhibition; inorganic pentavalent arsenate does not react as readily as the trivalent form with sulfhydryl groups, but may uncouple oxidative phosphorylation (Howard et al., 1984 and EPA, 1985, as cited in Eisler, 1988a). Toxic effects of As to aquatic life are significantly modified by numerous biological and abiotic factors. Acute toxicity is affected by water temperature, pH, Eh, organic carbon content, phosphate concentration, suspended solids, and presence of other substances and toxicants, as well as As speciation and duration of exposure. In general, inorganic arsenicals are more toxic than organoarsenicals (Eisler, 1988a). Table D-1 summarizes information on the toxicity of As to marine organisms.

Sediment Toxicity

AET concentrations for Puget Sound which ranged from 57 to 700 mg/kg (dry weight) for AS (PTI, 1988). Long and Morgan (1991) reported effects range-low (ER-L) and effects range-median (ER-M) concentrations of 33 and 85 mg/kg for As.

Beryllium (Be)

Many common Be compounds are readily soluble in water. Soluble Be salts are hydrolyzed to beryllium hydroxide in water, the solubility of which is 2,000 µg/L in the pH range of most natural waters. It is possible that in most natural aquatic environments Be is present in particulate rather than dissolved form. Based on its similarity to aluminum, it is expected that Be is adsorbed onto clay mineral surfaces at low pH and is complexed to some insoluble compounds at high pH (Clement Associates, 1985).

Table D-1. Concentrations (µg/l) of As resulting in lethality to marine fish and invertebrates.

SPECIES	ENDPOINT	CONCENTRATION (µg/l)	REFERENCE
ACUTE TOXICITY			
Fish			
Grey mullet (*Chelon labrosus*)	96-hr. LC50	27,300	Tayler et al., 1985, as cited in Eisler, 1988
Pink salmon (*Oncorhynchus gorbuscha*)	10-day LC54	3,800	EPA, 1985, as cited in Eisler, 1988
Chum salmon (*Oncorhynchus keta*)	48-hr. LC50	11,000	NAS, 1977, as cited in Eisler, 1988
Invertebrates			
Dungeness crab zoea (*Cancer magister*)	96-hr. LC50	230	EPA, 1985, as cited in Eisler, 1988
Pacific oyster embryos (*Crassostrea gigas*)	96-hr. LC50	330	EPA, 1985, as cited in Eisler, 1988
Blue mussel (*Mytilus edulis*)	Mortality (3 to 16 day exposure)	16,000	NAS, 1977, as cited in Eisler, 1988
Clam (*Macoma balthica*)	192-hr. LC50	15,000-220,000	Bryant et al., 1985, as cited in Eisler, 1988

Bioaccumulation

Be may be accumulated to a slight extent by aquatic organisms, however, there is no evidence of food chain biomagnification. A BCF of 19 was reported in the bluegill and the half-life of one day was reported for Be (Clement Associates, 1985).

Aquatic Toxicity

No information was found describing the mechanism of action of Be in aquatic biota. Data for several freshwater fish species indicate that the acute toxicity of Be decreases by about two orders of magnitude with an increase in water hardness from about 20 to 400 mg/L calcium carbonate. Acute concentrations of Be for the fathead minnow range from 150 to 20,000 µg/L over this range of hardness. Acute and chronic concentrations for *Daphnia magna* were reported to be 2,500 and 5.3 µg/, respectively, at a hardness of 220 mg/L (Clement Associates, 1985).

Sediment Toxicity

No information was found relating sediment concentrations of Be to effects in aquatic biota.

Cadmium (Cd)

Cd is relatively mobile in the aquatic environment. It is removed from aqueous media by complexation with organic materials and subsequent absorbtion to the sediment (Clement Associates, 1985). The availability of Cd to aquatic organisms depends on numerous factors, including adsorption and desorption from terrigenous materials, pH, Eh, chemical speciation, and many other factors. Adsorption and desorption processes are likely to be major factors in controlling the concentration of Cd in natural waters (Eisler, 1985).

Bioaccumulation

Freshwater and marine organisms accumulate Cd from the ambient environment. In American oysters held for 40 weeks in seawater containing 5.0 *ug*/L of Cd, edible tissues contained 13.6 *ug*/L (fresh weight) (Zaroogian and Cheer, 1976, as cited in Eisler, 1985).

Rainbow trout exposed for two weeks to 9 *ug/L* Cd, were reported to have BCFs ranging from 17 for the liver to 260 for gill tissue (Roberts et al., 1979, as cited in Eisler, 1985). The evidence for Cd transfer through the food web suggests that only the lower trophic levels exhibit biomagnification (Eisler, 1985).

Aquatic Toxicity

The exact mechanism of Cd toxicity is unknown, but among teleosts it appears to depend, in part, on duration of exposure, Cd concentration, water temperature, and salinity. Under conditions of high Cd concentration and short exposure, the gill appears to be the primary site of damage and accumulation. Under conditions of prolonged exposure and low concentration, the intestine and kidney are primarily affected (Eisler, 1985). At the molecular level, essential metals are competitively exchanged by Cd at active sites of enzymes or membrane proteins. Its reactions with biologically active groups (e.g., carboxyl, phenoxyl, sulphhydryl, and phosphate groups) appear to be of basic significance for toxic action. Many enzymes are known to be inhibited by Cd (e.g., ATPase, phosphatase, carboxyhydrase, peptidase, and aldolase) (Theede, 1980).

Acute toxicity has been demonstrated in marine fish at concentrations ranging from 0.6 to 15,900 µg/L, whereas toxicity to marine invertebrates has been reported to range between 10 and 611 µg/L (Table D-2) (Mance, 1987). Chronic toxicity has been reported in marine fish at concentrations from 0.5 to 500 µg/L; it ranges from 6.5 to 1,000 µg/L in marine invertebrates (Table D-2) (Eisler, 1985; Mance, 1987).

Sediment Toxicity

Long and Morgan (1991) report ER-L and ER-M Cd concentrations of 5 and 9 mg/kg, respectively.

Table D-2. Toxicity of cadmium in marine fish and invertebrates.

SPECIES	ENDPOINT	CONCENTRATION (µg/l)	REFERENCE
ACUTE TOXICITY			
Fish			
Killfish (*Cyprinodon variegatus*)	96-hr. LC50	15,900	Mance, 1987
Silversides (*Menidia menidia*)	96-hr. LC50	6,400	Mance, 1987
Flounder (*Pseudopleuronectes americanus*)	60% mortality (8-day exposure)	1,066	Mance, 1987
Chinook salmon juveniles (*Oncorhynchus tshawytscha*)	96-hr. LC50	0.6-1.6	Finlalyson and Verrue, 1982, as cited in Eisler, 1985
Coho salmon adults (*Oncorhynchus kisutch*)	215-hr. LC50	3.7	Chapman and Stevens, 1978, as cited in Eisler, 1985
Invertebrates			
Pacific oyster larva *(Ostrea lurida)*	48-hr. EC50 (abnormal development)	611	Mance, 1987
Blue mussel *(Mytilus edulis)*	96-hr. exposure (reduced growth)	10	Mance, 1987
Mysid shrimp (*Mysid opsis bahia*)	96-hr. LC50	17	Mance, 1987
Dungeness crab zoeae *(Cancer magister)*	96-hr. LC50	247	Mance, 1987
CHRONIC TOXICITY			
Fish			
Flounder (*Platichthys flesus*)	Tetanic body spasms (63-day exposure)	500	Mance, 1987
Striped bass (*Morone saxatilis*)	Deceased O_2 consumption & enzyme disruption (30-90 day exposure)	0.5-5.0	Theede et al., 1979 as cited in Eisler, 1985
Invertebrates			
Crab (adult) (*Pagarus longicarpus*)	Mortality (20-60-day exposure)	60-1,000	Mance, 1987
Shrimp (adult) (*Palaeomonetes pugio*)	Mortality (23-40-day exposure)	250-1,000	Mance, 1987
Shrimp (larva) (*Penaeus indicus*)	No effect on growth or survival (28-day exposure)	186	Mance, 1987
Crab (*Pontoporeia affinis*)	Reduced F1 life span (265-day exposure)	6.5	Sundelin, 1983, as cited in Eisler, 1985

Chromium (Cr)

Cr exists primarily in either a trivalent or hexavalent oxidation state. Hexavalent Cr is rather soluble and is quite mobile in surface water and is not adsorbed to any significant degree to clays or hydrous metal oxides. However, in reducing environments, it is rapidly converted to trivalent Cr, which is strongly adsorbed to sediments and is much less mobile. Most trivalent Cr in the aquatic environment is hydrolyzed and precipitates as chromium hydroxide. Sorption to sediments and bioaccumulation will remove much of the remaining trivalent Cr from solution. Trivalent and hexavalent Cr are readily interconvertible in nature depending on microenvironmental conditions such as pH, hardness, and the types of other compounds present (Clement Associates, 1985).

Bioaccumulation

Cr is an essential nutrient and is accumulated in a variety of marine biota, especially benthic organisms, to levels much higher than in ambient water. Concentrations in biota are usually lower than those in sediments. Passage of Cr through the food chain has been demonstrated and the food chain appears to be a more efficient pathway for Cr uptake than direct uptake from seawater (Clement Associates, 1985), although bioaccumulation is believed to be a minor fate process for Cr (Eisler, 1986).

Aquatic Toxicity

The mechanism of action for the toxicity of Cr appears to be related to the strong oxidizing action of chromates (hexavalent Cr), and all biological interactions of chromates appear to result in reduction of the trivalent form and subsequent coordination to organic molecules (Langard and Norseth, 1979, as cited in Eisler, 1986). The acute toxicity of Cr^{+6} in marine teleosts has been reported at concentrations ranging from 12,400 to 91,000 µg/l (EPA, 1980, as cited in Eisler, 1986) while that in marine invertebrates has been reported at concentrations between 200 and 1,000 µg/l (Table D-3) (Reish, 1977, as cited in EPA, 1986). The acute toxicity of trivalent Cr has been reported in a marine fish at concentrations of 53,000 µg/l (Negelski, 1976, as cited in Eisler, 1986) and in marine invertebrates at concentrations from 10,300 to 56,000 µg/l (Table D-3) (Reish, 1977 and EPA, 1980, as cited in Eisler, 1986).

The chronic toxicity of hexavalent Cr to anadromous fish is reported to range from 16 to 500 µg/l (Table D-3) (EPA, 1980 and Sugatt, 1980, as cited in Eisler, 1986). Chronic toxicity in marine invertebrates is reported to be between 50 and 100 µg/l (Oshida et al., 1976 and EPA, 1980, as cited in Eisler, 1986).

Sediment Toxicity

Long and Morgan (1991) reported ER-L and ER-M Cr concentrations of 80 and 145 mg/kg, respectively. Exposure to 0.5 mg/kg of hexavalent Cr for 28 days resulted in shell disease syndrome in grass shrimp (Doughtie et al., 1983, as cited in Eisler, 1986).

Copper (Cu)

Cu, an essential nutrient for many organisms, forms many compounds and complexes that are readily soluble, and therefore highly mobile in soil and surface water. Cu occurs in nature as the elemental metal (zero valence), and in the +1 and +2 valence states. In addition to a variety of inorganic compounds, Cu forms strong complexes with organic ligands. Most Cu (+1) compounds are not stable in the environment, particularly in the presence of water, and tend to change to the more stable +2 valence state (EPA, 1985). The adsorption of Cu to organic matter, clays, and other materials is the major process that limits its dispersal (Clement Associates, 1985). The fate of Cu in the aquatic environment is determined by chemical and biological conditions which affect its speciation and sorption affinity. The nature of chemical speciation of Cu in aquatic media is determined by the oxidation-reduction potential and the pH of the ambient environment as well as the availability of organic and inorganic ligands. In aquatic media of pH <7, copper may exist in the +2 valence state, whereas at pH >7, Cu may exist as the carbonate complex. Various sorption processes reduce the concentration of dissolved Cu compounds in aquatic environments. Sorption onto clay materials, hydrous iron, manganese oxides, and organic material can exert a strong influence on the bioavailability of Cu. In organically rich sediments, sorbed and precipitated Cu may become redissolved through complexation and may persist in the water column for extended periods (EPA, 1985).

Table D-3. Toxicity of chromium to marine fish and invertebrates.

SPECIES	ENDPOINT	CONCENTRATION (µg/l)	REFERENCE
ACUTE TOXICITY			
Fish (Cr +6)			
Atlantic silverside larva (*Menidia menidia*)	96-hr. LC50	12,400-14,300	EPA, 1980, as cited in Eisler, 1986
Mummichog (*Fundulus heteroclitus*)	96-hr. LC50	91,000	EPA, 1980, as cited in Eisler, 1986
Speckled sanddab (*Citharichthys stigmaeus*)	96-hr. LC50	30,000-31,000	EPA, 1980, as cited in Eisler, 1986
Invertebrates (Cr +6)			
Polychaete worms			
(*Neanthes arenaceodentata*)	28-56-day LC50	200-550	Reish, 1977, as cited in Eisler, 1986
(*Nereis virens*)	21-day LC50	1,000	Reish, 1977, as cited in Eisler, 1986
(*Capitella capitata*)	4-28-day LC50	280-5,000	Reish, 1977, as cited in Eisler, 1986
Fish (Cr +3)			
Yellow-eye mullet	96-hr. LC50	53,000	Negelski, 1976, as cited in Eisler, 1986
Invertebrates (Cr +3)			
American oyster (*Crassostrea virginica*)	96-hr. LC50	10,300	EPA, 1980, as cited in Eisler, 1986
Worm (*Neanthes arenaceodentata*)	No effect 21-day exposure	12,500	Reish, 1977, as cited in Eisler, 1986
Crab zoea (*Sesarma haematocheir*)	96-hr. LC50	56,000	EPA, 1980, as cited in Eisler, 1986
CHRONIC TOXICITY			
Fish (Cr +6)			
Chinook salmon fingerlings	Reduced growth (14-16-wk. exposure)	16-21	EPA, 1980, as cited in Eisler, 1986
Coho salmon juveniles	Decreased disease resistance (2-wk. exposure)	500	Sugatt, 1980, as cited in Eisler, 1986
Seaward migrating coho salmon	Reduced salinity tolerance (4-wk. exposure)	230	Sugatt, 1980, as cited in Eisler, 1986
Invertebrates (Cr +6)			
Polychaete worm			
Neanthes arenaceodentata	Disrupted feeding (14-day exposure)	79	EPA, 1980, as cited in Eisler, 1986
Neanthes arenaceodentata	Termination of reproduction (440-day exposure)	100	Oshida et al., 1976, as cited in Eisler, 1986
Capitella capitata	Decreased brood size (150-day exposure)	50 & 100	EPA, 1980, as cited in Eisler, 1986

Bioaccumulation

Copper does not biomagnify extensively in aquatic organisms. Bioconcentration factors range from 12 for algae to 30,000 for mollusks (EPA, 1985). Cu is strongly accumulated by biogenic ligands in plants and animals (Clement Associates, 1985).

Aquatic Toxicity

Cu toxicity is governed by its speciation. No information was found regarding the mechanism of action of Cu. Acute toxicity has been reported in marine fish at Cu concentrations ranging from 28 to 7,340 µg/L and in marine invertebrates from 17 to 560 µg/L (Table D-4) (Mance, 1987). Chronic toxicity in marine fish has been reported at concentrations from 150 to 160 µg/L and in marine invertebrates at Cu concentrations from 22 to 180 µg/L (Table D-4) (Mance, 1987).

Sediment Toxicity

Exposure to sediments contaminated with copper at concentrations between 17 and 60 mg/kg resulted in decreased burrowing time in two species of clam (*Protothaca staminea*, *Macoma* sp.) (Phelps et al., 1983). Bioassays conducted with sediments from several estuaries resulted in toxic responses at copper concentrations between 60 and 80 mg/kg (McGreer, 1979; Qasim *et al.*, 1980; Long and Morgan, 1991). Long and Morgan (1991) report ER-L and ER-M Cu concentrations of 70 snf 390 mg/kg, respectively.

Lead (Pb)

Pb commonly exists in one of three oxidation states in the environment: 0, +2, and +4. Metallic Pb (0 valence) and the more common Pb minerals are largely insoluble in water. Pb leached from ores tends to adsorb to ferric hydroxide or combine with carbonate or sulfate to form compounds of very low solubility (Clement Associates, 1985).

Table D-4. Toxicity of copper in marine fish and organisms.

SPECIES	ENDPOINT	CONCENTRATION (µg/l)	REFERENCE
ACUTE TOXICITY			
Fish			
Atlantic menhaden (*Brevoortia tyrannus*)	14-day LC50	610	Mance, 1987
Mummichog larva (*Fundulus heterocliltus*)	96-hr. LC50	1,000	Mance, 1987
Pinfish larva (*Lagodon rhomboides*)	14-day LC50	150	Mance, 1987
Pinfish adult	96-hr. LC50	2,750-7,340	Mance, 1987
Spot (*Leiostomus xanthurus*)	14-day LC50	160	Mance, 1987
Summer flound larva (*Paralichthys dentatus*)	96-hr. LC50	28	Mance, 1987
Invertebrates			
Polychaete worms			
Capitella capitata	96-hr. LC50	180-200	Mance, 1987
Neanthes arenaceodentata	96-hr. LC50	77-200	Mance, 1987
Oyster			
Crassostrea virginica (embryo)	48-hr. LC50	100	Mance, 1987
C. gigas (adult)	96-hr. LC50	560	Mance, 1987
Clam (*Mya arenaria*)	96-hr. LC50	39	Mance, 1987
Amphipod (*Acartia tonsa*)	96-hr. LC50	17-55	Mance, 1987
Dungeness crab zoeae (*Cancer magister*)	96-hr. LC50	49 ± 20	Mance, 1987
CHRONIC TOXICITY			
Fish			
Pinfish larva	14-day LC50	150	Mance, 1987
Spot	14-day LC50	160	Mance, 1987
Invertebrates			
Polychaete worm			
Capitella capitata (larva)	28-day LC50	180	Mance, 1987
Neanthes arenaceodentata (adult)	28-day LC50	44-100	Mance, 1987
Oyster juveniles (*C. virginica*)	70% mortality (60-day exposure)	22	Mance, 1987
Blue mussel adult (*Mytilus edulis*)	Mortality (630-day exposure)	10	Mance, 1987

The transport of Pb in the aquatic environment is influenced by its speciation. Pb exists predominantly as the divalent cation in most unpolluted waters and adsorbed to particulate matter. However, in polluted waters complexation with organic matter can be important and generally serves to increase Pb mobility. Volatilization of Pb compounds is not important in most aquatic environments (Clement Associates, 1985).

Sorption processes appear to exert a dominant effect on the distribution of Pb in the environment. Adsorption to inorganic solids, organic materials, and hydrous iron and manganese oxides controls the mobility of Pb and results in a strong partitioning of Pb to the bed sediments. The extent of sorption varies with the geological setting, pH, Eh, availability of ligands, dissolved and particulate ion concentrations, salinity, and chemical composition. The equilibrium solubility of Pb with carbonate, sulfate, and sulfide is low. Over most of the normal pH range, Pb carbonate and Pb sulfate control the solubility of Pb in aerobic systems, while Pb sulfide is dominant under anaerobic conditions. Biomethylation of Pb by microorganisms can remobilize Pb (Clement Associates, 1985).

Bioaccumulation

Bioaccumulation of Pb has been demonstrated for a variety of organisms, and bioconcentration factors are within the range of 100 to 1,000. Microcosm studies indicate that Pb is not biomagnified through the food web (Clement Associates, 1985). The storage sites for Pb are uncertain, although they are probably in soft tissues as well as bone. The residence half- life (Tb 1/2) of inorganic Pb is estimated to be 20 days in blood, 28 days in whole body, and 600 to 3,000 days in bone (Harrison and Laxen, 1981, as cited in Eisler, 1988b).

Aquatic Toxicity

Pb modifies the function and structure of kidney, bone, the central nervous system, and the hematopoietic system, and produces adverse biochemical, histopathological, fetotoxic, teratogenic, and reproductive effects (Eisler, 1988b). Biochemically, Pb exerts deleterious effects on hematopoiesis through derangement of hemoglobin synthesis, resulting in a shortened life span of circulating erythrocytes, often resulting in anemia (Eisler, 1988b).

The mode of action for organolead compounds differs from that of inorganic Pb. Organolead concentrates in liver, and it is there that tetraalkyllead is probably converted to trialkyllead. The $Tb_{1/2}$ of organoleads is between 200 and 350 days (Harrison and Laxen, 1981, as cited in Eisler, 1988b). At present, the mode of action for organolead is poorly understood, but organolead compounds are known to inhibit amino acid transport, uncouple oxidative phosphorylation, and inhibit cerebral glucose metabolism (Hong et al., 1983, as cited in Eisler, 1988b).

Acute toxicity of Pb in marine fish has been reported at concentrations between 50 and 180,000 µg/l while acute toxicity in marine invertebrates is reported to range from 100 to greater than 500,000 µg/l (Table D-5) (Eisler, 1988b and Mance, 1987). No information was found describing the chronic toxicity of Pb in marine fish, however, exposure of three generations of brook trout (*Salvelinus fontinalis*) to mean total Pb concentrations (0.9 to 474 µg/l) showed that all second-generation trout exposed to 235 and 474 µg Pb/l and 34 percent of those exposed to 119 µg Pb/l developed severe spinal deformities. Spinal deformities also appeared in 21 percent of the newly hatched third-generation alevins exposed to 119 µg Pb/l, and weights of these fish 12 weeks after hatch were significantly reduced (Holcombe et al., 1976). Chronic toxicity in marine invertebrates has been reported at concentrations between 20 and 3,100 µg/l (Table D-5) (Eisler, 1988b).

Sediment Toxicity

Decreased burrowing time of a marine clam (*Macoma balthica*), decreased benthic diversity, and lower macroinvertebrate taxa richness were reported at lead sediment concentrations between 30 and 42 mg/kg (Gilbert et al., 1976; Lee and Mariani, 1977; McGreer, 1979; Rygg, 1985). Macroinvertebrate richness was lower in sediments of Kishwaukee River, Illinois, with a mean lead concentration of 31 mg/kg (Illinois EPA, 1988a). Long and Morgan (1991) report ER-L and ER-M Pb concentrations of 35 and 110 mg/kg, respectively.

Mercury (Hg)

Several forms of Hg, including insoluble elemental Hg, inorganic species, and organic species, exist in the environment. In general, the mercurous (+1) salts are much less soluble than the more commonly found mercuric (+2) salts. Hg also forms many stable

Table D-5. Lead toxicity to marine fish and invertebrates.

SPECIES	ENDPOINT	CONCENTRATION (µg/l)	REFERENCE
ACUTE TOXICITY			
Fish			
Plaice (*Pleuronectes platessa*)	96-hr. LC50 (Tetramethyl Pb)	50	Maddock and Taylor, 1980, as cited in Eisler, 1988
Plaice	96-hr. LC50 (Pb +2)	180,000	Maddock and Taylor, 1980, as cited in Eisler, 1988
Mummuchog (*Funculus heteroclitus*)	96-hr. LC50	315	EPA, 1985, as cited in Eisler, 1988
Sole (*Limanda limanda*)	96-hr LC50	50	Mance, 1987
Invertebrates			
Blue mussel adult (*Mytilus edulis*)	96-hr. LC50 (Pb +2)	>500,000	Maddock and Taylor, 1980, as cited in Eisler, 1988
Blue mussel larvae	96-hr. LC50 (Pb +2)	476	EPA, 1985, as cited in Eisler, 1988
Blue mussel	96-hr. LC50 (Tetraethyl Pb)	100	Maddock and Taylor, 1980, as cited in Eisler, 1988
Shrimp (*Crangon crangon*)	96-hr. LC50	375,000	Maddock and Taylor, 1980, as cited in Eisler, 1988
Dungeness crab (*Cancer magister*)	96-hr. LC50	575	Eisler, 1988
CHRONIC TOXICITY			
Fish			
No data			
Invertebrates			
Polychaete worm (*Neanthes arenaceodentata*)	Inhibited reproduction (28-day exposure)	20-3,100	Kobayashi, 1971, as cited in Eisler, 1988
American lobster (*Homarus americanus*)	Biochemical alterations (30-day exposure)	50	EPA, 1985, as cited in Eisler, 1988

organic complexes. The nature and solubility of the chemical species that occur in an environmental system depend on a variety of factors including the redox potential and the pH (Clement Associates, 1985).

Hg (zero valence state) and certain Hg compounds, including several inorganic species and dimethyl Hg, can volatilize to the atmosphere from aquatic environments. Volatilization is reduced by conversion of metallic Hg to complexed species and by the formation of HgS in reducing sediments. Adsorption onto suspended and bed sediments is probably the most important process determining the fate of Hg in the aquatic environment. Sorption is strongest onto organic materials (Clement Associates, 1985).

Virtually any Hg compound can be remobilized in aquatic systems by microbial conversion to methyl and dimethyl forms. Conditions reported to enhance biomethylation include high concentrations of available Hg, large numbers of bacteria, the absence of strong complexing agents, near neutral pH, high temperatures, and moderately aerobic environments (Clement Associates, 1985).

Bioaccumulation

Hg is strongly bioaccumulated by numerous mechanisms. Methylmercury is the most readily accumulated and retained form of Hg in aquatic biota. In organisms near the top of the food web, such as carnivorous fish, almost all Hg exists in the methylated form, primarily as a result of the consumption of prey containing methylmercury, and is generally detected at higher concentrations than in prey organisms (Clement Associates, 1985; Eisler, 1987a). Methylmercury is the most toxic and readily bioaccumulated form of Hg, is able to penetrate membranes in living organisms (Beijer and Jernelov, 1979), and accumulates in protein-rich tissues such as fish muscle because it binds strongly with sulfhydryl groups.

Aquatic Toxicity

Hg is among the most toxic of the trace elements, has a complex behavior in the environment, has no known biological function, and will persist for decades following abatement of the source if transport resources are not active. The toxic action of Hg appears to reside in the strong affinity of the mercuric ion for ligands such as phosphates, cysteinyl and histidyl side chains of proteins, or purines, pterdidines and porphyrins (Carty and

Malone, 1979, as cited in Armstrong, 1979). It binds to, and affects the configuration of nucleic acids, inhibits a large number of enzymes by blocking sulphydryl groups, can change catalytic activity in certain enzymes by substituting for zinc, and can disrupt pathways of oxidative phosphorylation (Vallee and Ulmer, 1972, as cited in Armstrong, 1979). The neurotoxic effect of Hg compounds is believed to be related to their ability to react directly or catalytically with a group of lipids important in the structure of membranes of cells of the central nervous system, promoting hydrolysis and hydrolytic decomposition (Armstrong, 1979).

Acute toxicity of Hg has been demonstrated in marine fish at concentrations ranging from 51 to 74 µg/l (Mance, 1987) and in marine invertebrates at concentrations ranging from 1.6 to 150 µg/l (Table D-6) (Mance, 1987). Chronic toxicity has been reported in marine fish at concentrations between 30 and 75 µg/l (Mance, 1987) and in marine invertebrates at concentrations from 0.25 to 6 µg/l (Table D-6) (Eisler, 1987; Mance, 1987).

Sediment Toxicity

Results from a laboratory spiked-sediment bioassay showed adverse effects to rainbow trout eggs upon exposure to sediment containing 0.2 mg/kg of inorganic Hg (Birge et al., 1979). Studies conducted using field-collected sediment from freshwater systems revealed significant mortality to Daphnia magna associated with concentrations of inorganic Hg at 0.30 mg/kg (Trinity River, Texas), 0.20 mg/kg (Keweenaw Waterway, Michigan), 0.30 mg/kg (Torch Lake, Michigan), 1.5 mg/kg (Little Grizzly Creek, California), and 9.4 mg/kg (Phillips Chain of Lakes, Wisconsin). Sediment collected from Waukegan Harbor, Illinois, containing 0.1 mg/kg inorganic Hg were highly toxic to *Hyalella azteca* (Long and Morgan, 1991). Long and Morgan (1991) report ER-L and ER-M Hg concentrations of 0.15 and 1.3 mg/kg, respectively.

Table D- 6. Mercury toxicity in marine fish and invertebrates.

SPECIES	ENDPOINT	CONCENTRATION (µg/l)	REFERENCE
ACUTE TOXICITY			
Fish			
Mummichog embryo (*Fundulus heteroclitus*)	96-hr. LC50	51-74	Mance, 1987
Invertebrates			
Annelid worm (*Limnodriloides verrucosus*)	96-hr. LC50	120-150	Mance, 1987
Oyster (*Crassostrea gigas*)	Abnormal development (48-hr. exposure)	6.7	Mance, 1987
Blue mussel (*Mytilus edulis*)	Growth termination (96-hr. exposure)	1.6	Mance, 1987
Shrimp adult (*Palaeomonetes pugio*)	96-hr. LC50	10-120	Mance, 1987
Dungeness crab zoeae (*Cancer magister*)	96-hr. LC50	8.2	Mance, 1987
CHRONIC TOXICITY			
Fish			
Mummichog embryo	Reduced hatching (32-day exposure)	37-75	Mance, 1987
Mummichog embryo	EC50-Viable hatch (32-day exposure)	30-51	Mance, 1987
Invertebrates			
Shrimp (post-larval) (*Penaeus indicus*)	No effect (28-day exposure)	6	Mance, 1987
Slipper limpet (*Crepidula fornicata*)	Reduced growth (16-week exposure)	0.25	Thain, 1984, as cited in Eisler, 1987

Nickel (Ni)

Ni is a highly soluble metal in aquatic systems because many Ni compounds are highly soluble in water. However, Ni sulfide, formed under reducing conditions and in the presence of sulfur, is quite insoluble. In natural, unpolluted waters, sorption and coprecipitation processes involving hydrous iron and manganese oxides are moderately effective in limiting the mobility of Ni. In more organic-rich, polluted waters, sorption of Ni is less likely. The lack of other controls on Ni mobility makes incorporation into bed sediments an important fate process of Ni in surface waters (Clement Associates, 1985).

Bioaccumulation

Ni is not accumulated to a significant degree by aquatic biota. BCFs are usually on the order of 100 to 1,000 (Clement Associates, 1985).

Aquatic Toxicity

No information was found describing the mechanism of action of Ni in aquatic biota. Acute toxicity in marine fish is reported at concentrations between 7,960 and 350,000 µg/L while in marine invertebrates it is reported to be between 349 and >50,000 µg/L. (Table D-7) (Mance, 1987).

No information was found describing the chronic toxicity of Ni to marine fish or invertebrates.

Sediment Toxicity

The AET concentration for Ni in Puget Sound sediments is greater than 140 mg/kg (dry weight) (PTI, 1988). Long and Morgan (1991) report ER-L and ER-M Ni concentrations of 30 and 50 mg/kg, respectively.

Table D-7. Toxicity of nickel to marine fish and invertebrates.

SPECIES	ENDPOINT	CONCENTRATION (µg/l)	REFERENCE
ACUTE TOXICITY			
Fish			
Mummichog adult (*Fundulus heteroclitus*)	96-hr. LC50	350,000	Mance, 1987
Silversides (*Menidia menidia*)	96-hr. LC50	7,960	Mance, 1987
Invertebrates			
Polychaete worms			
Capitella capitata	96-hr. LC50	>50,000	Mance, 1987
Ctenodrilus serratus	96-hr. LC50	17,000	Mance, 1987
Oyster larva			
Crassostrea gigas	Abnormal development 48-hr. EC50	349	Mance, 1987
C. virginica	96-hr. LC50	1,180	Mance, 1987
Dungeness crab zoeae (*Cancer magister*)	96-hr. LC50	4,360	Mance, 1987
CHRONIC TOXICITY			
No data found			

Selenium (Se)

In natural waters, Se is present in the selenite (+4) or selenate (+6) forms. These chemical species are very soluble and it is probable that most of the Se released to the aquatic environment is transported in these forms. However, Se also strongly sorbs to hydrous metal oxides. Sorption by bed sediments or suspended solids, and precipitation with hydrous iron oxides serve as major controls on mobility of Se in aerobic waters. However, most Se in aquatic systems is probably transported as the dissolved species (Clement Associates, 1985).

Bioaccumulation

Se is a critical nutrient for many organisms (Eisler, 1985) and is strongly bioaccumulated by aquatic biota. Dietary intake is thought to be the most important source of Se in many organisms. Se does not appear to biomagnify (Clement Associates, 1985).

Aquatic Toxicity

No information was found describing the mechanism of action of Se. The acute toxicity of Se has been reported to range from 470 to 67,100 µg/l in marine fish and from 600 to greater than 10,000 µg/l in marine invertebrates (Table D-8) (Eisler, 1985).

Table D-8. Toxicity of selenium to marine fish and invertebrates.

SPECIES	ENDPOINT	CONCENTRATION (µg/l)	REFERENCE
ACUTE TOXICITY			
Fish			
Sheepshead minnow adult (*Cyprinodon variegatus*)	96-hr. LC50	7,400-67,100	Ward et al., 1981, as cited in Eisler, 1985
Sheepshead minnow (eggs through juvenile)	LC50 (life-cycle)	470-970	Ward et al., 1981, as cited in Eisler, 1985
Summer flound larvae (*Paralichthys dentatus*)	96-hr. LC50	3,500	EPA, 1980, as cited in Eisler, 1985
Invertebrates			
Pacific oyster larvae (*Crassostrea gigas*)	48-hr. LC50	>10,000	Glickstein, 1978, as cited in Eisler, 1985
Dungeness crab (*Cancer magister*)	96-hr. LC50	1,040	Glickstein, 1978, as cited in Eisler, 1985
Mysid shrimp juvenile (*Mysidopsis bahia*)	96-hr. LC50	600	EPA, 1980, as cited in Eisler, 1985
CHRONIC TOXICITY			
No data found			

No information was found describing the chronic toxicity of Se to aquatic biota.

Sediment Toxicity

No information was found relating sediment concentrations of Se to effects in aquatic biota.

Silver (Ag)

Ag can exist in several forms in aqueous systems. Metallic Ag, which has very low solubility, is stable over much of the Eh-pH range for water. Precipitation of silver chloride may exert a major control on solubility of Ag where chloride concentrations are relatively high. Under the reducing conditions often found in bed sediments, formation of insoluble Ag sulfides and metallic Ag may also control levels of soluble Ag species. Sorption to bed sediment and suspended particulates is a dominant process leading to removal of dissolved Ag from the water column (Clement Associates, 1985).

Bioaccumulation

Bioaccumulation of Ag by aquatic biota occurs readily and appears to depend primarily on sorption/desorption from sediments. However, the amount of Ag partitioned to the biota appears to be minor in comparison to that in the sediments. Little food-web biomagnification seems to occur (Clement Associates, 1985).

Aquatic Toxicity

No information was found describing the mechanism of action of Ag in aquatic biota. Limited toxicity information was found on the acute and chronic toxicity of Ag to marine fish and invertebrates. Winter flounder (*Pseudopleuronectes americanus*) eggs exposed to 174 µg/l of Ag for eight days showed no apparent effects, while embryos exposed for nine days to 180 µg/l were reported to have experienced reduced growth, yolk sac deformity, and 30 percent larval mortality (Mance, 1987). Fifty percent of Pacific oysters larva exposed to 22 µg/l of Ag for 48 hours were reported to have abnormal development (Mance, 1987). Fifty percent of blue mussel larvae exposed to 14 µg/l of Ag for 48 hours also were reported to have abnormal development while juveniles and adults exposed to 10 µg/l for 630 days showed no apparent effects on growth. Exposure to 50 µg/l for 365 days was reported to result in reduced growth (Mance, 1987).

Sediment Toxicity

The AET concentrations for Ag in Puget Sound which range from greater than 0.56 to greater than 6.1 mg/kg (PTI, 1988). Long and Morgan (1991) report ER-L and ER-M Ag concentrations of 1 and 2.2 mg/kg, respectively.

Zinc (Zn)

Zn generally exists in nature in the +2 valence state. Zn occurs in both suspended and dissolved forms. Dissolved Zn may occur as the free (hydrated) Zn ion or as dissolved complexes and compounds with varying degrees of stability and toxicity. Suspended (undissolved) Zn may be dissolved following minor changes in water chemistry or may be sorbed to suspended matter. The predominant fate of Zn in aerobic systems is sorption of the divalent cation by hydrous iron and manganese oxides, clay minerals, and organic matter. The efficiency of these materials in removing Zn from solution varies according to their compositions and concentrations; the pH and salinity of the water; the concentrations of complexing ligands; and the concentration of Zn. Concentrations of Zn in suspended and bed sediments always exceed concentrations in ambient water. In reducing environments, precipitation of Zn sulfide limits the mobility of Zn. Under aerobic conditions, precipitation of Zn compounds is probably important only where Zn is present at high concentrations. Zn tends to be more readily sorbed at higher pH than lower pH and tends to be desorbed from sediments as salinity increases. Complexes of Zn with the common ligands of surface waters are soluble in most neutral and acidic solutions, so that Zn is readily transported in most unpolluted, relatively organic-free waters (Clement Associates, 1985).

Bioaccumulation

Because Zn is an essential nutrient, it is strongly bioaccumulated even in the absence of abnormally high ambient concentrations. Zn does not appear to biomagnify through the food web. Although Zn is actively bioaccumulated in aquatic systems, biota represent a relatively minor sink compared to the sediments (Clement Associates, 1985).

Aquatic Toxicity

Zn has its primary metabolic effect on Zn-dependent enzymes that regulate RNA and DNA. The gill epithelium is the primary target site in fish. Acute toxicity in marine fish and in marine invertebrates has been reported at concentrations between 192 and 5,600 µg/L and between 80 and 10,700 µg/L respectively (Table D-9) (Eisler, 1993). Studies with salmonids report chronic toxicity in the range of 10 to 242 µg/L while concentrations of 10 to 2,500 have been reported to result in chronic toxicity in marine invertebrates (Table D-9) (Eisler, 1993).

Sediment Toxicity

The AET concentrations for Zn in Puget Sound range between 410 and 1,600 mg/kg (dry weight) (PTI, 1988). Significant mortality was observed among *Daphnia magna* exposed to sediments of the Trinity River in Texas with Zn concentrations of 121 ± 20 mg/kg (Qasim et al., 1980). Reduced benthic taxa richness was reported in the Dupage River in Illinois at Zn concentrations of 327 ± 162 mg/kg (Illinois EPA, 1988). Long and Morgan (1991) report ER-L and ER-M Zn concentrations of 120 and 270 mg//kg, respectively.

Table D-9. Zinc toxicity in marine fish and invertebrates.

SPECIES	ENDPOINT	CONCENTRATION (µg/l)	REFERENCE
ACUTE TOXICITY			
Fish			
Most marine species	96-hr. LC50	>1,000	EPA, 1980, as cited in Eisler, 1993
Tidewater silverside (*Menidia peninsulae*)	96-hr. LC50	5,600	Mayer, 1987, as cited in Eisler, 1993
Coho salmon juveniles (*Oncorhynchus kisutch*)	96-hr. LC50	820-1,810	Buhl and Hamilton, 1990, as cited in Eisler, 1993
Chinook salmon smolts (*O. tshawytscha*)	96-hr. LC50	446	EPA, 1987, as cited in Eisler, 1993
Cabezon larvae (*Scorpaenichthys marmoratus*)	96-hr. LC50	192	EPA, 1987, as cited in Eisler, 1993
Invertebrates			
Polychaete worm (*Capitella capitata*)	48-hr. LC50	10,700	Hunt and Anderson, 1989, as cited in Eisler, 1993
Starfish adults (*Asterias rubens*)	168 hr. LC50	2,300	Eisler and Hennekey, 1977, as cited in Eisler, 1993
Pacific oyster larvae (*Crassostrea gigas*)	Growth reduction (48-hr. EC50)	80-95	EPA, 1987, as cited in Eisler, 1993
Pacific oyster larvae	48-hr. LC50	119-310	EPA, 1980, as cited in Eisler, 1993
American oyster (*C. virginica*)	48-hr. LC50	340	Hunt and Anderson, 1989, as cited in Eisler, 1993
CHRONIC TOXICITY			
Fish			
Rainbow trout larvae (*O. mykiss*)	LC54 (28-day exposure)	10	Spear, 1981, as cited in Eisler, 1993
Sockeye salmon (embryo through smolt) (*O. nerka*)	No apparent effect (18-month exposure)	242	EPA, 1980, as cited in Eisler, 1993
Invertebrates			
Pacific oyster larvae	Reduced larval settlement (20-day exposure)	10-20	EPA, 1987, as cited in Eisler, 1993
Softshell clam (*Mya arenaria*)	100% mortality (70-day exposure)	25,000	Eisler, 1993
Prawn (*Palaemon elegans*)	LC100 (21-day exposure)	562	Nugegoda and Rainbow, 1989, as cited in Eisler, 1993
Capitella capitata larvae	28-day LC50	1,250	EPA, 1987, as cited in Eisler, 1993

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