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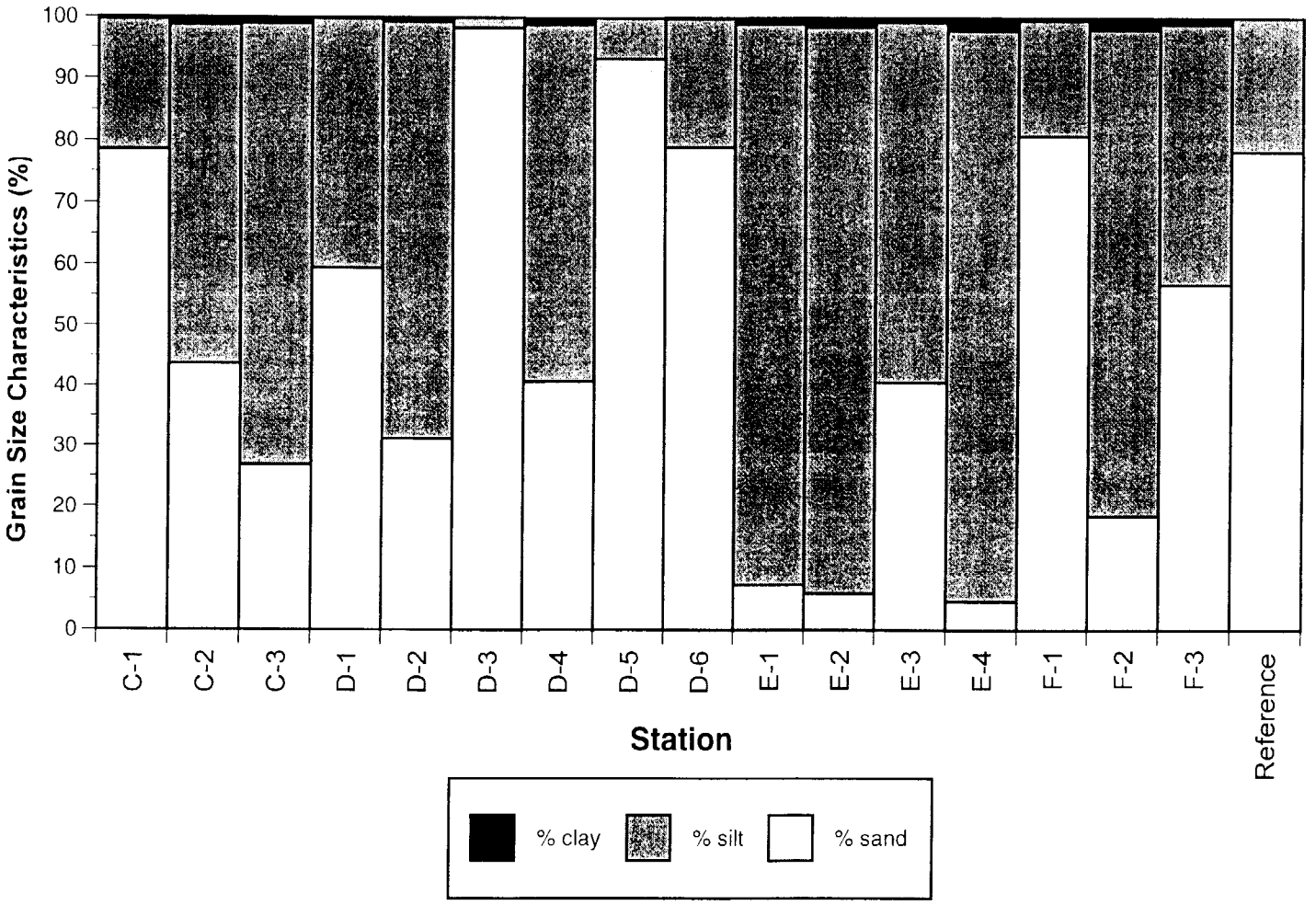
Figure 4.2-1. Grain size characteristics in surface samples from the Raymark study area.



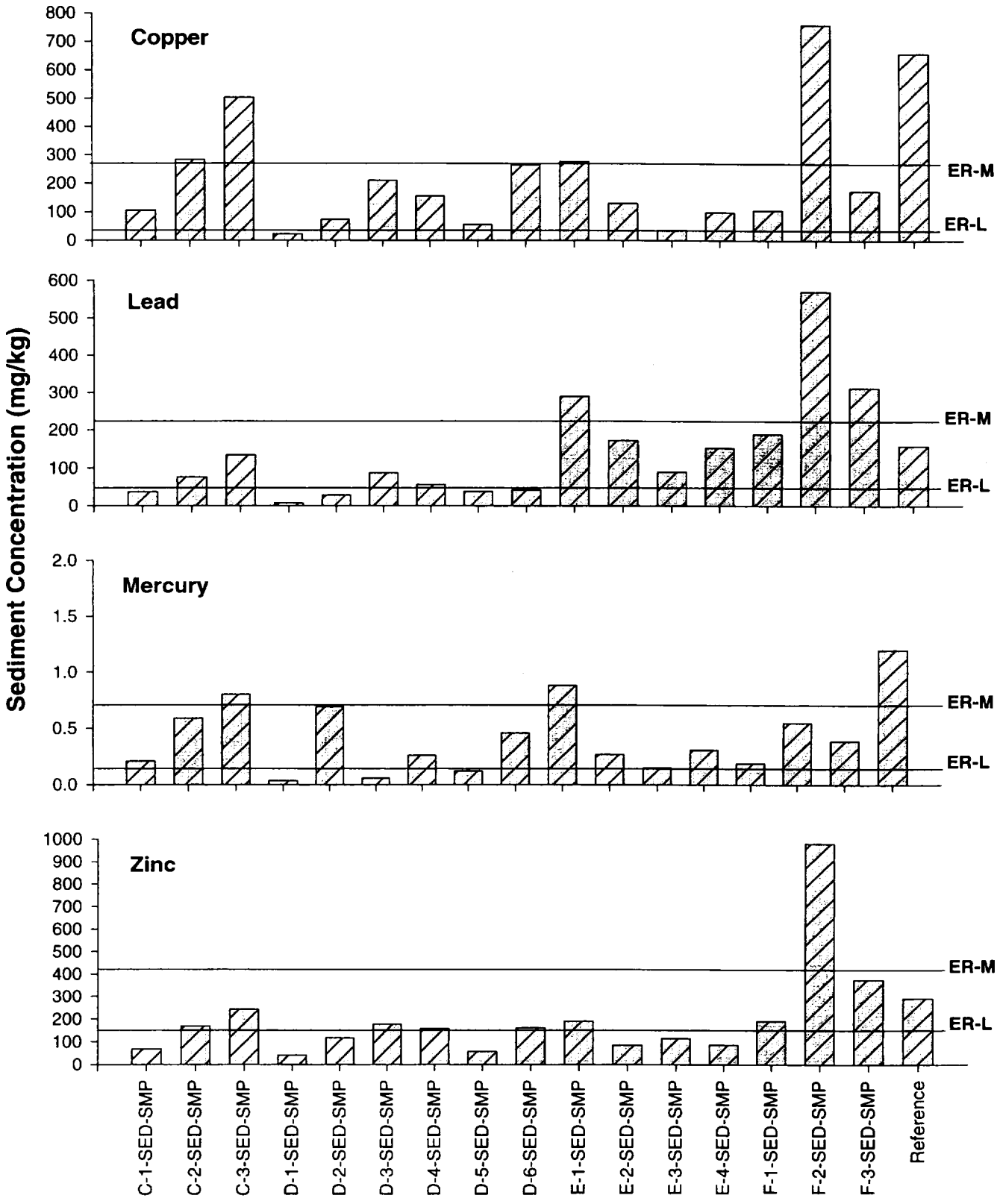


Figure 4.3-1. Concentration (mg/kg) of metals in sediments from the Raymark study area.

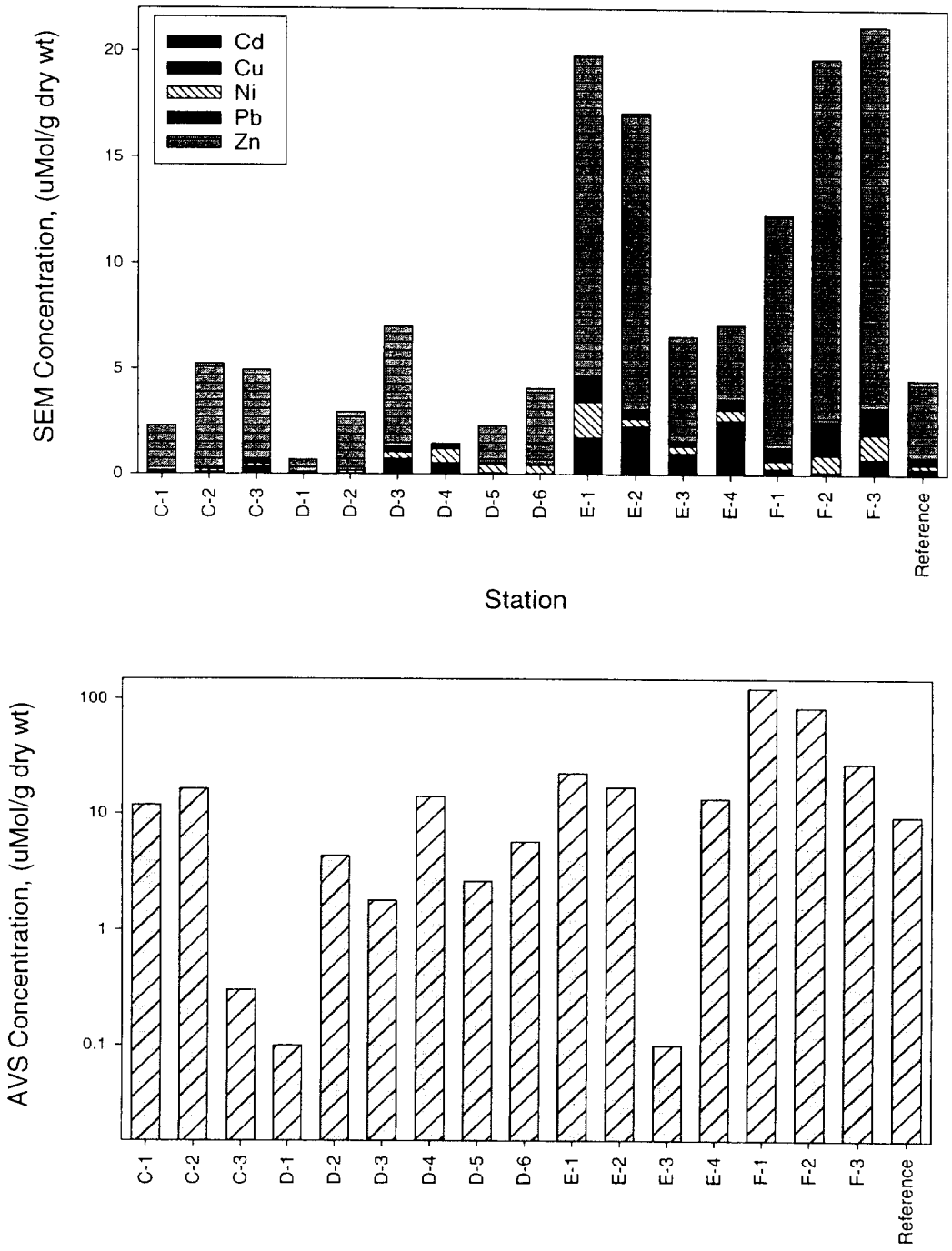


Figure 4.3-2. SEM and AVS concentrations (μMol/g dry wt) of divalent metals in whole sediments collected from the Raymark study area.

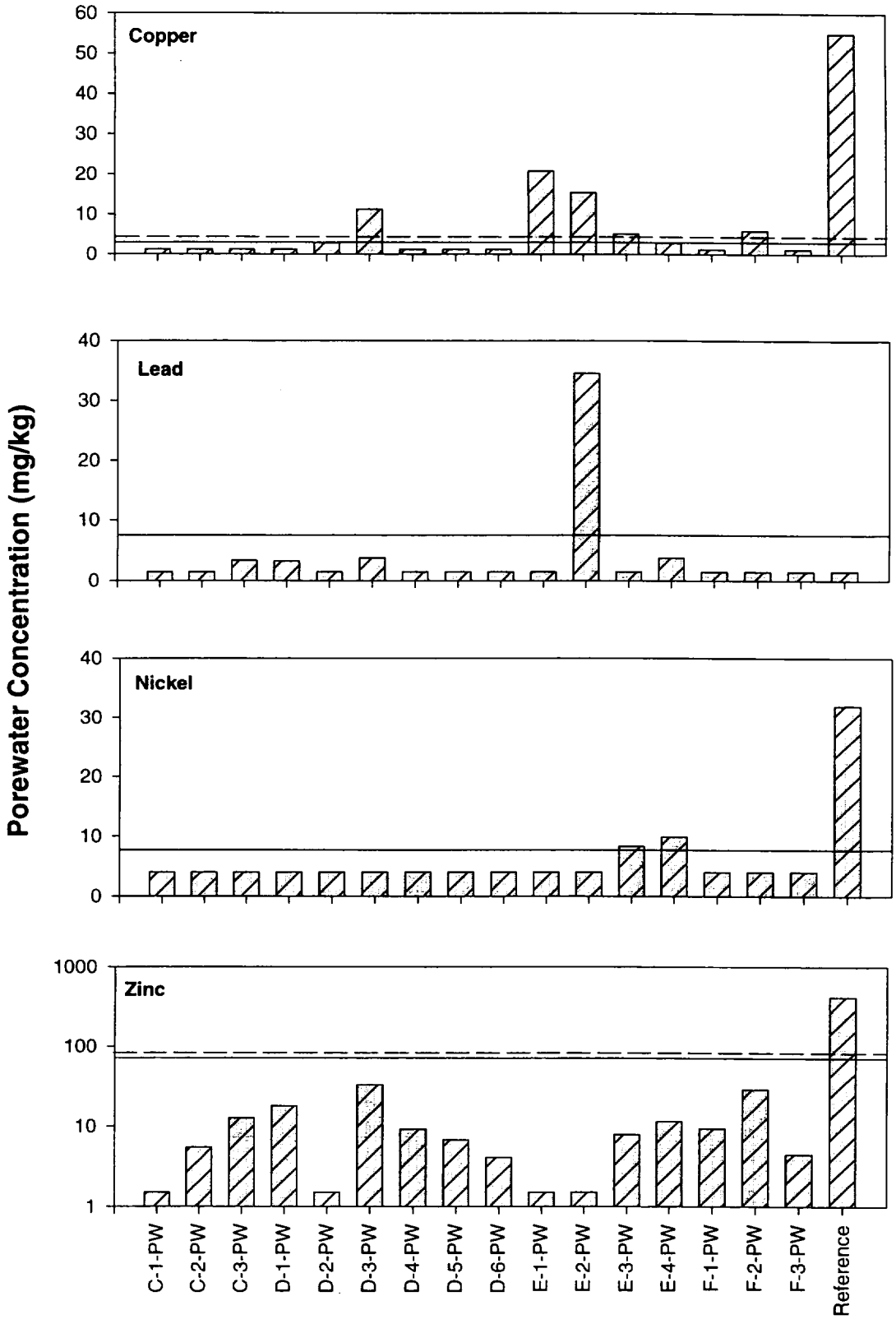


Figure 4.3-3. Concentration (mg/kg) of metals in porewater samples from the Raymark study area. Solid lines designate the Saltwater Chronic Values and dotted lines designate the Saltwater Acute Values.

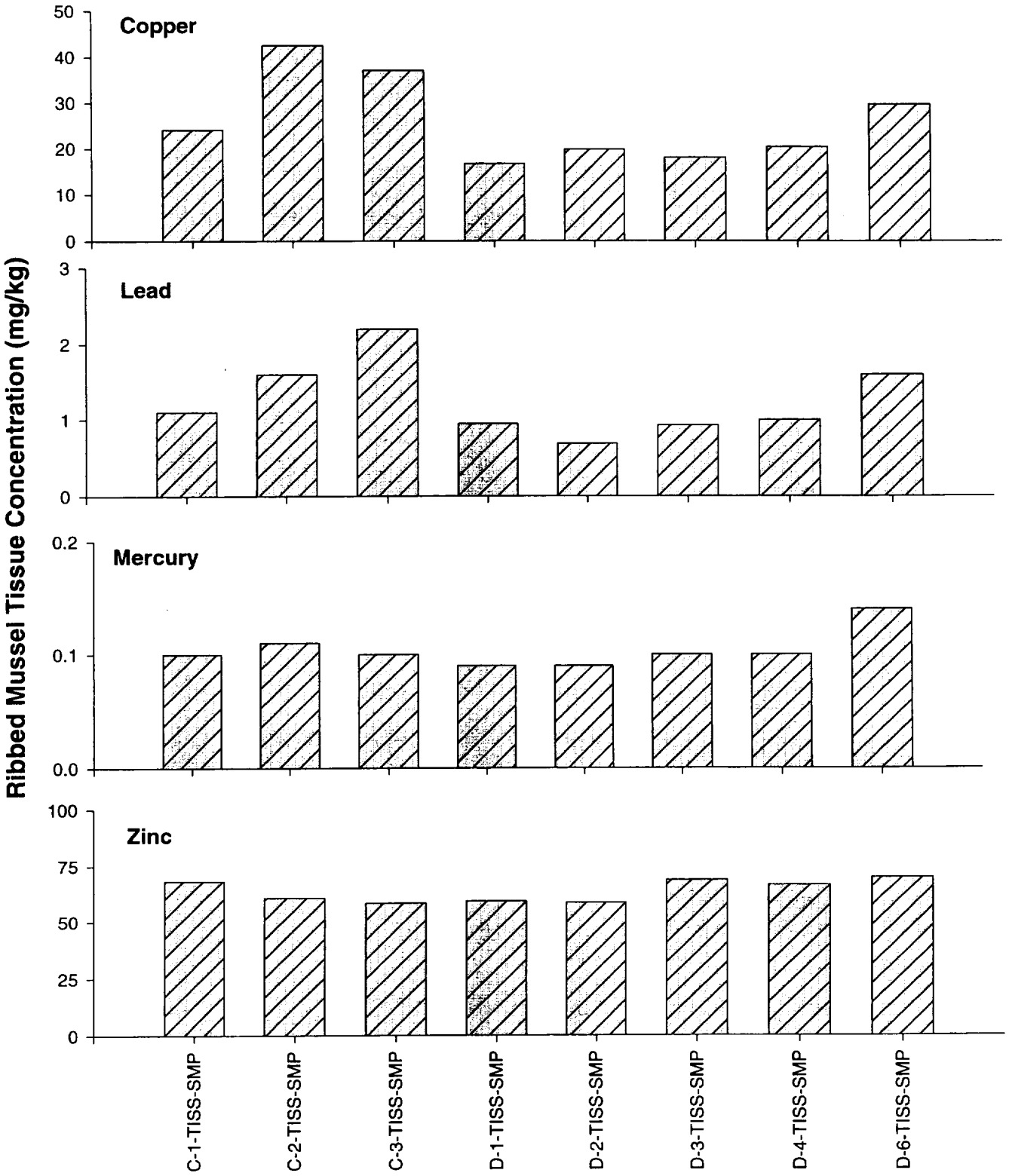


Figure 4.3-4. Concentration (mg/kg) of metals in ribbed mussels from the Raymark study area.

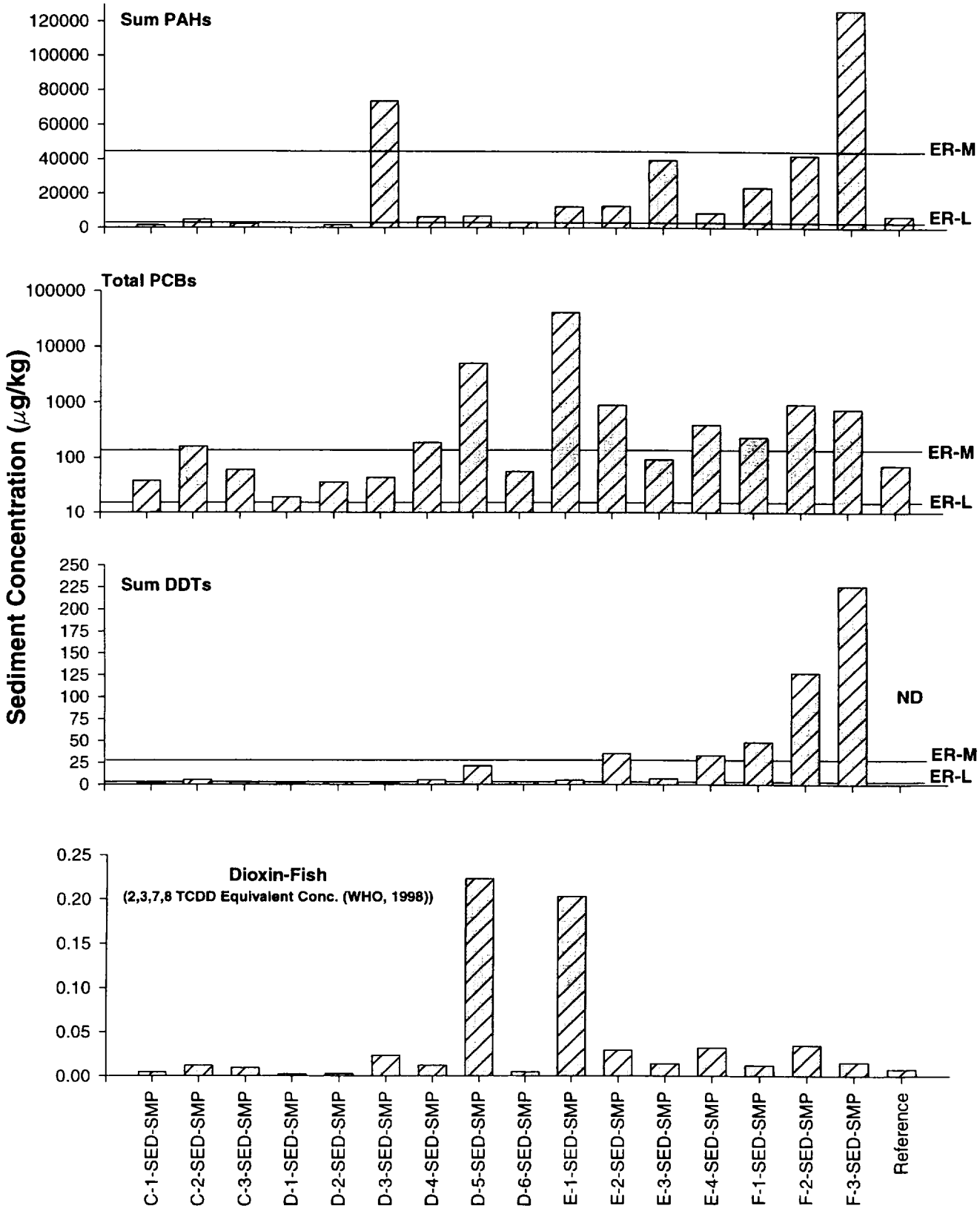


Figure 4.3-5. Concentration (µg/kg) of organics in sediment from the Raymark study area. (ND=No Data)

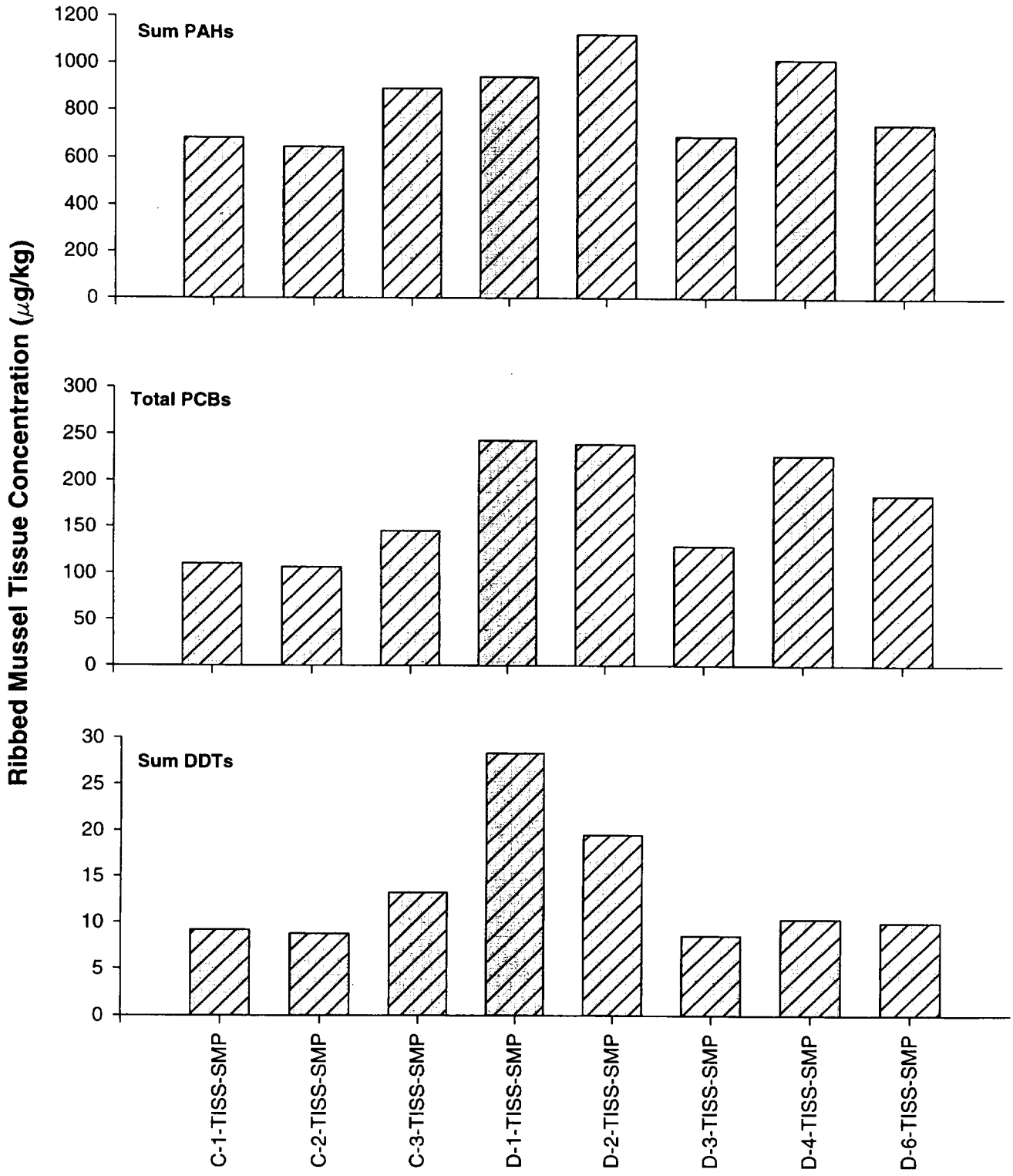


Figure 4.3-6. Concentration (μg/kg) of organics in ribbed mussels from the Raymark study area.

Table 4.2-1. Total Organic Carbon Content (TOC) and Grain Size of surface sediments collected from the Raymark study area.

Station	Surface TOC (%)				%SILT	
		% SAND	% SILT	%CLAY	63-15.6u	<15.6u
C-1	1.30	78.60	21.10	0.30	14.00	7.40
C-2	3.10	43.70	55.10	1.20	34.10	22.10
C-3	4.10	26.90	72.10	1.00	43.50	29.60
D-1	1.70	59.40	40.40	0.20	31.30	9.30
D-2	3.40	31.20	68.10	0.70	38.60	30.30
D-3	2.00	98.30	1.70	0.00	1.00	0.70
D-4	3.40	40.70	58.10	1.10	30.40	28.90
D-5	1.40	93.20	6.70	0.00	4.20	2.60
D-6	1.50	79.00	20.80	0.20	13.70	7.40
E-1	9.30	7.30	91.60	1.10	41.50	51.20
E-2	28.30	5.90	92.50	1.70	36.20	57.90
E-3	7.00	40.50	58.70	0.80	32.80	26.70
E-4	22.00	4.60	93.30	2.10	37.30	58.10
F-1	4.10	80.90	18.70	0.40	8.40	10.80
F-2	14.30	18.40	79.60	2.00	40.20	41.40
F-3	13.90	56.70	42.20	1.10	19.70	23.70
Reference	5.86	78.33	21.67	0.00	8.18	13.49

Table 4.3-1. Results of Simultaneously Extractable Metal (SEM) and Acid Volatile Sulfide (AVS) measurements in sediments and qualitative evaluation of divalent metal bioavailability for the Raymark Phase III Ecological Risk Assessment Investigation.

Station	AVS[1]	SEM		SEM-AVS		Exposure Ranking[3]
	(µMole/g dry)	(µMole/g dry)	FLAG[2]	(µMole/g dry)	FLAG[2]	
C-1	11.81	2.32	-	-9.5	-	-
C-2	16.22	5.23	+	-11.0	-	-
C-3	0.30	4.94	-	4.6	+	-
D-1	0.10	0.68	-	0.6	+	-
D-2	4.29	2.93	-	-1.4	-	-
D-3	1.79	7.02	+	5.2	++	+
D-4	13.95	1.48	-	-12.5	-	-
D-5	2.62	2.30	-	-0.3	-	-
D-6	5.68	4.11	-	-1.6	-	-
E-1	22.22	19.84	++	-2.4	-	+
E-2	16.77	17.12	++	0.4	+	+
E-3	0.10	6.58	+	6.5	++	+
E-4	13.38	7.14	+	-6.2	-	-
F-1	124	12.30	++	-112.1	-	+
F-2	83.45	19.72	++	-63.7	-	+
F-3	27.01	21.23	+++	-5.8	-	++
Reference	9.40	4.53	-	-4.9	-	-

1 - Mean of two replicates per station.
2 - Flag Codes: SEM Conc. < 5 µmol/g; SEM-AVS < 0 µmol/g = "-".
SEM Conc. ≥ 5 µmol/g; SEM-AVS ≥ 0 µmol/g = "+".
SEM Conc. ≥ 10 µmol/g; SEM-AVS ≥ 5 µmol/g = "++".
SEM Conc. ≥ 20 µmol/g; SEM-AVS ≥ 10 µmol/g = "+++".
3 - Exposure Ranking:
Baseline ("-") - Low (+) exposure observed for only one indicator or baseline (-) exposure for both indicators;
Low ("+") - Low (+) exposure observed for both indicators or intermediate (++) exposure for one indicator;
Intermediate ("++") - exposure observed for both indicators or high (+++) exposure for one indicator; and
High ("+++") - exposure observed in both indicators.

5.0. ECOLOGICAL EFFECTS ASSESSMENT

Ecological effects are quantified from the relationships between exposure patterns and resulting responses of ecological systems, as determined from measurement endpoints identified during Problem Formulation (Section 3). Ecological effects assessments include literature-reported evaluations of the known effects of CoCs to receptors of concern (Section 5.1); direct measurement of the toxicity of exposure media (Section 5.2), in this case sediments and porewaters to appropriately sensitive marine species (the amphipod *Ampelisca abdita*); site-specific investigations of the abundance and condition of receptors of concern (Section 5.3); and collation of toxicity-based criteria and standards for exposure media identified in exposure pathways (Section 5.4). Uncertainty associated with these assessments is discussed in Section 5.5.

5.1. Known Effects of CoCs

Contaminants of concern (CoCs) as identified in Section 3 include metals (cadmium, chromium, copper, lead, nickel, zinc and mercury), PAHs, PCBs, pesticides, and dioxins. Potential effects of the CoCs on biological receptors are influenced strongly by their chemical behavior, solubility, and toxicity. For example, nickel, copper, cadmium, and hexavalent chromium have relatively high solubility and thus higher dissolved phase concentrations than many organic contaminants, such as PAHs and relatively insoluble metals, (*e.g.,* lead, zinc, and trivalent chromium). Dissolved contaminants may be transported throughout the water column by current and tidal flows, while contaminants associated with particles tend to settle to the bottom in sediment depositional areas. Once on the bottom the sediment particles can be resuspended, resulting in redistribution of the contaminants. Dissolved or particle/sediment-bound contaminants may be available to biological receptors in the water column, pore waters and sediments, potentially resulting in biological uptake and/or direct toxicological effects. Impacts to organisms are often influenced by the affinity of various contaminants for tissue lipids and reflect the type of cellular or subcellular effects associated with particular compounds and elements. The following describes the chemical behavior and known effects of key contaminants of concern.

5.1.1. Metals

Cadmium. Cadmium in the water column may partition to dissolved and particulate organic carbon. Cadmium speciation yields primarily the divalent form of the metal, divalent cadmium, between pH 4.0 and pH 7.0 (Stephenson and Mackie, 1989). Studies indicate that the divalent cadmium ion is responsible for observed biological effects and that acid volatile sulfides can influence the toxicity and bioaccumulation of cadmium in sediments (Di Toro *et al.*, 1992).

Rule and Alden (1996) 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*). Uptake significantly increased as a function of increasing cadmium concentration in sediment and it 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. 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.

Schmitt and Finger (1987) concluded that cadmium and lead were of potential concern in edible fish tissue although Wiener and Stokes (1990) suggested that cadmium did not generally accumulate to any appreciable extent in the edible muscle tissue of fish.

Cadmium is not a highly mobile element in the aquatic food web and does not biomagnify (Kay, 1985). Cadmium measured in bird eggs was not a reliable indicator of environmental exposure (Kay, 1985). Tissue residue toxicity relationships for cadmium may be variable because detoxification processes allow organisms to sequester this metal in various unavailable forms while analytical measurements continue to detect its presence (Klerks and Bartholomew, 1991). Whole body residues may fail to predict effects concentrations at the organ level because concentrations in target organs may be larger than whole body residues (McKinney, 1993).

In freshwater studies, cadmium has been associated with high mortality, reduced growth, and inhibited reproduction (Eisler, 1985). Generally, resistance to cadmium was higher in marine organisms when compared to freshwater species (Eisler, 1985). Marine organism LC_{50}s ranged from 320 to 430 μg/L, whereas effects in freshwater organisms have been observed at 1-2 μg/L. In tests of Puget Sound sediment, statistically significant effects were noted in the amphipod, oyster larvae, and Microtox™ bioassays (*i.e.*, bacterial bioluminescence tests), at cadmium concentrations ranging between 6.7 and 9.6 μg/g (Long *et al.*, 1990). Cadmium concentrations of 1.2 and 1.7 μg/g were measured in tests of San Francisco Bay sediments and caused significant toxicity in the amphipod and bivalve larvae bioassays, respectively (Long *et al.*, 1990). Highly toxic effects (*i.e.*, 75% mortality) were noted in amphipod tests of Commencement Bay sediments with 41.6 μg/g cadmium (Long *et al.*, 1990). Low abundances of echinoderms and arthropods were observed in Southern California where cadmium concentrations were 6.2 and 4.3 μg/g, respectively (Long *et al.*, 1990). Complete mortality was observed in tests using the polychaete *Nereis verens* exposed to Black Rock Harbor sediments at 1.6 μg/g cadmium (Long *et al.*, 1990). Baltimore Harbor sediments were toxic to mummichogs and spot, where the cadmium concentration in these sediments was
22.8 μg/g (Long *et al.*, 1990).

The ER-L and ER-M benchmarks for cadmium over a range of studies and species, are 1.2 and 9.6 μg/g, respectively (Long *et al.*, 1995).

Chromium. 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 toxic surface waters (Beaubien *et al.*, 1994). In some surface waters, as much as 10 to 15 percent of the dissolved chromium may be present in the colloidal/organic form.

Hexavalent chromium occurs only rarely in nature, except from anthropogenic contamination, because it is readily reduced to trivalent chromium in the presence of oxidizable organic matter. However, chromate and dichromate compounds are stable in many natural waters because of the low concentration of reducing material, and may undergo intermediate transport. In contrast, trivalent chromium compounds, the form most commonly observed in biological systems, are generally insoluble in water.

Chromium appears to have limited mobility in most aquatic habitats because the trivalent form tends to bind to sediments. However, plants can bioaccumulate and reduce chromium.

Bioconcentration of hexavalent chromium in the gills of rainbow trout was significantly higher at pH 6.5 than at pH 8.1 and is directly coupled with oxygen transfer (Van der Putte and Part, 1982). Van der Putte and Part (1982) 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.

Little evidence exists for the biomagnification of chromium in aquatic food webs, although sediments frequently contain elevated concentrations of trivalent chromium (Weis and Weis, 1993).

There is wide variation in sensitivity to chromium among species, even among those which are closely related (Eisler, 1986). Hexavalent chromium is classified as a human carcinogen, but trivalent chromium still is being evaluated for its carcinogenic potential. Most of the chromium in aquatic environments is expected to eventually precipitate to sediments.

Tissue residue-toxicity relationships can 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 when detoxified (Klerks and Bartholomew, 1991). Whole body residues may 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 (McKinney, 1993).

Acute toxicity to marine organisms in water-only exposures is evident at concentrations ranging from 2000 to 105,000 ppm (μg/L) total chromium (Long *et al.*, 1990). Tests with the amphipod *Rhepoxynius abronius* indicate toxic effects in sediments from Commencement Bay where chromium levels ranged between 16.2 to 19.7 μg/g (Long *et al.*, 1990). Survival greater than 97% was observed in the polychaete *Neanthes arenaceodentata* exposed to San Diego Bay sediment with 299.5 μg/g chromium (Long *et al.*, 1990).

The ER-L and ER-M values for chromium in sediments are 81 and 370 μg/g, respectively (Long *et al.*, 1995).

Copper. Copper in the aquatic environment can partition to dissolved and particulate organic carbon. Two processes influence the fate of copper: sorption and chemical speciation. Speciation is determined by the oxidation-reduction potential of the copper compound and the media pH. In contaminated settings, copper may form complexes with organic material in the water; however such complexes ultimately settles out of the water column and are deposited in sediments. Various processes including sorption onto clay minerals, hydrous iron, manganese oxides, and organic material reduce the level of copper compounds in aquatic media. However, the sorbed and precipitated copper in organically rich sediments may become redissolved and can persist in the water for long periods.

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 (*i.e.*, the ionic copper concentration decreases an order of magnitude for every 0.5 increase in pH above 6). The hydroxide species and free copper ions complexes consisting of carbonates, phosphates, nitrates, ammonia, and sulfates are weakly toxic or nontoxic. Ionic copper concentration is highly correlated with toxicity.

Copper is not strongly bioaccumulated. 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. Rule and Alden (1996) 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 to copper concentrations but not cadmium concentrations in sediments. 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.

Bioconcentration factors for copper are in the range of 10 to 100, although in some mollusks it can reach 30,000 (U.S. EPA, 1984). This may be because copper proteins in the blood of many bivalves act as oxygen carriers. For example, American oysters have been documented to have tissue concentrations of 1,500 μg/g (Hammond and Beliles, 1980).

Little evidence exists to support the general occurrence of biomagnification of copper in the aquatic environment (Woodward *et al.*, 1994). Copper concentration does not appear to increase significantly through aquatic (or terrestrial) food webs

although copper is accumulated by aquatic organisms primarily through dietary exposure (Woodward *et al.*, 1994). Most organisms retain only a small proportion of the heavy metals ingested with their diet.

A considerable number of aquatic species are sensitive to dissolved concentrations of copper in the range of 1-10 μg/L. Copper is an essential element for most organisms, although the distinction between deficiency and toxicity in many organisms, including algae and some invertebrates, is small if there is limited ability to control absorption. Fish are sensitive to copper, and it is thought that their gills do not provide an effective barrier to absorption (Hammond and Beliles, 1980).

In addition to affecting survival, copper exposure has been associated with development of histopathological lesions in mollusks and fish (Martin, 1977 and Gardner and LaRoche, 1973), inhibition of egg hatching in fish (Gardner and LaRoche, 1973), impairment of fertilization and larval development in polychaetes and echinoderms (Reisch, 1964; Young and Nelson, 1974 and Bougis, 1965), and retardation of growth in hydroids (Karbe, 1972). Copper is particularly active in disruption of enzymatic systems (Albergoni and Piccinni, 1983).

In water-only exposures, acute responses of marine organisms were observed at concentrations ranging from 5.8 to 600 μg/L (U.S. EPA , 1986 and Anderson *et al.*, 1991). Mortality responses in the amphipod *Rhepoxynius abronius* ranged from 13% to 79% in sediments from Commencement Bay where corresponding copper concentrations ranged from 85.1 to 2820 μg/g (Long *et al.*, 1990). In oyster bioassays, a highly toxic developmental response (*i.e.*, >44% abnormal larvae) was observed in tests of sediments with 918 μg/g copper from Commencement Bay (Long *et al.*, 1990). Eighteen to 67% mortality to *Rhepoxynius abronius* was observed in tests of sediments from San Francisco Bay, where copper concentrations were between 72 and 85 μg/g, respectively (Long *et al.*, 1990). Complete mortality was observed in exposures of the polychaete *Nereis virens* to Black Rock Harbor sediment with 612 μg/g copper (Long *et al.*, 1990).

The ER-L and ER-M values for copper in sediments are 34 and 270 μg/g, respectively (Long *et al.*, 1995).

Lead. Lead is most bioavailable at low pH, and in environments with 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. 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 (Babukutty and Chacko, 1995). The chemistry of lead in aqueous solutions is highly complex because of its occurrence in many forms, although it has a tendency to form compounds of low solubility. The divalent form is the stable ionic

species of lead. Hydroxide, carbonate, sulfide and, more rarely, sulfate may act as solubility controls.

Lead may occur either as adsorbed ions or surface coatings on sediment mineral particles, or it may be carried as a part of suspended living or non-living organic matter in the water (ATSDR, 1988). In the sediments, a portion of lead can be transformed to trimethyl lead and tetraalkyl lead compounds through chemical and microbial processes. The organolead compounds are much more toxic to aquatic organisms than are the inorganic lead compounds (Hodson *et al.*, 1984). The amount of bioavailable lead in sediment is controlled, in large part, by the concentration of acid volatile sulfides (AVS) and organic mater (DiToro *et al.*, 1990, Casas and Crecelius, 1994 and Kemdorff and Schnitzer, 1980).

Lead and lead compounds bioaccumulate in invertebrates, but do not appear to bioaccumulate in fish. Bioaccumulation of organolead compounds is rapid and high; these compounds concentrate in the fatty tissues of aquatic organisms. Babukutty and Chacko (1995) and others reported a strong correlation between soft tissue concentration of lead in worms and that in the exchangeable fraction of the sediment.

Lead is accumulated by aquatic organisms equally from water and through dietary exposure (Woodward *et al.*, 1994). 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 (Woodward *et al.*, 1994 and Hodson *et al.*, 1984).

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 (U.S. Department of Health and Human Services, 1993).

In freshwater tests, adverse effects to test organisms occurred between 1.3 and 7.7 mg/L (Long *et al.*, 1990). Studies indicate that marine organisms in water-only exposures are more sensitive (Long *et al.*, 1990). A proposed marine water quality standard for California was 8 mg/L (Long *et al.*, 1990). Statistically significant responses to amphipods, oyster larvae, and Microtox™ were observed in Puget Sound sediment tests at concentrations ranging between 530 and 660 μg/g (Long *et al.*, 1990).

The ER-L and ER-M values for lead in sediments are 46.7 and 218 μg/g, respectively (Long *et al.*, 1995).

Mercury. Mercury forms a wide variety of complexes with organic ligands, the compounds (*e.g.*, methylmercury) which are toxicologically and environmentally significant (Nriagu, 1979).

Mercury is very persistent when released into the environment, with the major removal mechanism occurring by adsorption onto particles and subsequent settlement to sediments. Mercury can become methylated to a highly toxic form, methylmercury, by biological and chemical processes (Nriagu, 1979). Methylation occurs most readily under anaerobic conditions.

Mercury has no known essential status or function in organisms, and is a mutagen and teratogen (U.S. EPA, 1985). Bioaccumulation and toxic effects of elemental mercury in aquatic systems are highly complex and are influenced by water temperature, salinity, hardness, pH, age of an organism, prior exposure, reproductive state (related to lipid content), trophic level, and metabolism.

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 microbial processes mediate production and destruction of methylmercury This complex process is affected by environmental factors (U.S. EPA, 1996). A significant fraction of the total mercury in water is found in the form of methylmercury, the species predominantly accumulated by aquatic organisms (St. Louis *et al.*, 1994). In a study of the Onondaga Lake food web, the percent of total mercury found to occur as methylmercury was lake water 5%, interstitial water 37%, phytoplankton 24%, zooplankton 40%, benthic macroinvertebrates 26%, and fishes 96% (Becker and Bigham, 1995).

Toxicity of methylmercury is dependent on temperature (Armstrong, 1979), oxygen conditions (Sloof *et al.*, 1991), salinity (McKenney and Costlow, 1981), and the presence of other metals such as zinc and lead (Parker, 1979). 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 relationship is complicated by the fact that some animals exposed to low concentrations of mercury can build up a tolerance to this contaminant. Detoxification of the free metal within their cells occurs via the production of metallothioneins and other metal-binding proteins. Brown *et al.* (1983) propose that toxic effects occur as the binding capacity of these metal-binding proteins becomes saturated.

Fish bioconcentrate methylmercury directly from water by uptake across the gills (Rodgers and Beamish, 1981; Wren *et al.*, 1983; and Xun *et al.*, 1987) and piscivores, such as walleye, readily accumulate mercury from dietary sources (Mathers and Johansen, 1985 and Wiener and Spry, 1996). Methylmercury accumulation from either source may be substantial, but the relative contribution of each pathway may vary with fish species (Huckabee *et al.*, 1978; Norstrom *et al.*, 1976; Phillips *et al.*, 1980; and Rodgers and Qadri, 1982). In addition, invertebrates generally have a lower percentage of methylmercury in their tissues than fish or marine mammals (Beckvar *et al.*, 1996). The percentage of methylmercury increases with age in both fish and invertebrates (Beckvar *et al.*, 1996), but not in 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. But, unlike other aquatic species, the tissue concentrations of methylmercury are higher in

juvenile marine mammals than in adults because the adults can mineralize methylmercury into inorganic mercury (Eisler, 1987a).

In birds, there is a tendency for mercury concentrations to be highest in species feeding on fish (or on other seabirds) (Braune, 1987). However, when mercury levels are compared amongst predominantly fish-eating species, there is no clear pattern or any evident association with diet composition (Elliot *et al.*, 1992). Particularly high concentrations have been found in some species of procellariiforms (Muirhead and Furness, 1988). There is an inverse relationship between total mercury and percent methylmercury in tissues of various avian species (Norheim *et al.*, 1982 and Karlog and Clausen, 1983). 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-molting seabirds avoid the toxic effects of accumulating large quantities of methylmercury (Thompson and Furness, 1989 and Honda *et al.*, 1990).

Among furbearers, mercury burdens are higher in fish-eating species than in herbivores (Sheffy and St. Amant, 1982). Mink and river otter accumulate about 10 times more mercury than predatory fishes from the same areas (Kucera, 1983). Non-marine mammals with mercury concentrations in the liver and kidney in excess of approximately 30 μg/g 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 approximately 2 to 6 μg/g of wet weight produced mercury poisoning in feeding experiments using a range of mammalian species (Thompson, 1996).

Mercury is considered to be one of the most toxic of the heavy metals (Nriagu, 1979). At higher concentrations, mercury is toxic to a wide range of marine invertebrates and fish, and its acute toxicity varies among species. For instance, mercury is acutely toxic to the mysid shrimp *Mysidopsis bahia* at concentrations as low as 3.5 μg/L, whereas the acute value for winter flounder is 1,678 μg/L (U.S. EPA, 1985). In general, fish tend to be less sensitive to mercury than are crustaceans and mollusks.

Acute toxicity in water-only exposures of mercury to marine organisms is observed between 3.5 and 1,678 ppm (Long *et al.*, 1990). Statistically significant responses in Puget Sound sediment tests were observed at concentrations ranging between 0.4 and 2.1 μg/g to amphipods, oyster larvae, and Microtox™ (Long *et al.*, 1990). Highly toxic (*i.e.*, 67 and >78% mortality) effects to the amphipod *Rhepoxynius abronius* were observed in tests of Commencement Bay and San Francisco Bay sediments with 11.2 μg/g and 1.0 μg/g mercury, respectively (Long *et al.*, 1990).

The ER-L and ER-M values for mercury in sediments are 0.15 and 0.71 μg/g, respectively (Long *et al.*, 1995).

Nickel. Nickel in the aquatic environment readily partitions to dissolved and particulate organic carbon which reduce its bioavailability. Nickel bioavailability can also be influenced to some extent by the concentrations of calcium and magnesium in water, and is largely mediated by the concentration of acid volatile sulfides (AVS) (Di Toro *et al.*, 1990). 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. 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. Ankley *et al.* (1991) 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.

Biomagnification of nickel does not normally occur in the aquatic environment (Krantzberg and Boyd, 1992 and Nriagu, 1980).

U.S. EPA (1986) provides the following insights into the adverse effects of nickel:

> "Mechanisms of nickel toxicity are varied and complex, and, as with other heavy metals, significant effects occur at cell membranes and membranous tissues, such as gills. In fish, hematological effects such as hyperglycemia, lymphopenia, and erythocytosis have been reported in association with nickel intoxication..."

Nickel exposure has resulted in reduced photosynthesis in aquatic plants (plankton and macrophytes), inhibition of enzyme systems in a variety of organisms, stunted growth and development, reproductive impairment, and at sufficiently high levels, death. Exposure levels associated with these effects are summarized in U.S. EPA (1986). Nickel is classified as a human carcinogen (U.S. EPA, 1992b).

Acute toxicity to marine organisms has been observed in water-only exposures to nickel at 151.7 ppm (Long *et al.*, 1990). In tests with sediments from Puget Sound, statistically significant toxicity to amphipods, oyster larvae, and Microtox™ was observed at concentrations ranging between 28 and >120 μg/g (Long *et al.*, 1990). Exposures of the amphipod *Rhepoxynius abronius* indicated highly toxic responses (*i.e.*, 67 and >78% mortality) to Commencement Bay and San Francisco Bay sediments with 41 μg/g and 113 μg/g nickel, respectively (Long *et al.*, 1990). No survival was observed in the polychaete *Nereis virens* when exposed to 52.0 μg/g nickel in Black Rock Harbor sediment (Long *et al.*, 1990).

The ER-L and ER-M values for nickel in sediments are 20.9 and 51.6 μg/g, respectively (Long *et al.*, 1995).

Zinc. Sorption onto sediments is probably the predominant fate of zinc in the aquatic environment (Eisler, 1993). Small amounts may be partitioned into the dissolved phase through speciation into soluble zinc compounds. Formation of

complexes with organic and inorganic ligands may increase the mobility of zinc in aquatic media, but these complexes also have a tendency to be adsorbed more strongly onto sediments. 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 (DiToro *et al.*, 1990).

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 (Klerks and Bartholomew, 1991 and Kraak *et al.*, 1992). Whole-body residues also might not be indicative of effect 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 (McKinney, 1993).

Bioavailability of zinc in sediments is controlled by the AVS concentration (Schubauer-Berigan *et al.*, 1993).

Schmitt and Finger (1987) evaluated the effects of sample preparation techniques on measured concentrations of metals in the edible tissue of fish and concluded that there was little direct value in measuring zinc (or copper, iron, or manganese) tissue residues in fish because they do not bioaccumulate to any appreciable extent.

There appears to be little evidence to support the general occurrence of biomagnification of zinc within marine or freshwater food webs (Timmermans, *et al.*, 1992a). However, a biomagnification factor of 790 was determined for the midge *Chironomus riparius* (Timmermans, *et al.*, 1992b).

Zinc is an essential element in maintaining many physiological processes, and zinc deficiency can result in severe adverse effects on growth, reproduction, and survival in plants and animals. However, exposure to excess concentrations of zinc can result in a range of adverse physiological and ecological effects. According to Eisler (1993):

> "The most sensitive aquatic species were adversely affected at nominal water concentrations between 10 and 25 μg Zn/L, including representative species of plants, protozoans, sponges, mollusks, crustaceans, echinoderms, fish, and amphibians. Acute LC_{50} (96-h) values were between 32 and 40,930 μg/L for freshwater invertebrates, 66 and 40,900 μg/L for freshwater teleosts, 195 and >320,000 μg/L for marine invertebrates, and 191 and 38,000 μg/L for marine teleosts. Acute toxicity...was markedly affected by the age and nutrient status of the organism... Pancreatic degeneration occurred in ducks fed diets containing 2,500 mg Zn/kg ration. Ducks died when fed diets containing

3,000 mg Zn/kg feed."

Thus, according to Eisler (1993), adverse effects include decreased growth, survival, and reproduction. While noncarcinogenic effects of zinc on humans and animals are evident, carcinogenic effects are not.

Long *et al.* (1990) report acute LC_{50s} for marine fish in water-only exposures to zinc ranging from 192 to 320,400 ppm . Chronic responses of marine mysids in water-only exposures were noted at 120 ppm (Long *et al.*, 1990). Statistically significant responses in Puget Sound sediment tests were observed at concentrations ranging between 870 and 1600 μg/g to amphipods, oyster larvae, and Microtox™ (Long *et al.*, 1990). A highly toxic response in the amphipod *Rhepoxynius abronius* was observed during testing of sediment with 707 μg/g zinc from Puget Sound (Long *et al.*, 1990). A significant toxic response (*i.e.*, 43% mortality) was observed during exposures of the amphipod *Rhepoxynius abronius* to San Francisco Bay sediment with 158 μg/g zinc (Long *et al.*, 1990). No survival was observed in the polychaete *Nereis virens* exposed to Black Rock Harbor sediment with 334 μg/g zinc (Long *et al.*, 1990).

The ER-L and ER-M values for zinc in sediments are 150 and 410 μg/g, respectively (Long *et al.*, 1995).

5.1.2. Polycyclic Aromatic Hydrocarbons (PAHs)

Most PAHs occur in sediments as complex mixtures. It has been demonstrated that the toxicities of individual PAHs are generally additive (Swartz *et al.*, 1995; Swartz *et al.*, 1997). The primary PAHs at this site are 3-6 ring compounds (*e.g.*, phenanthrene, fluoranthene, pyrene, chrysene, benzo(a)anthracene, benzo(b)fluoranthene, benzo(k)-fluoranthene, benzo(a)pyrene, and dibenzo(a,h)anthracene). These compounds typically have low solubility in water, high partition coefficients (*i.e.*, higher affinity for organic matter, such as in soil and sediments, than for water), and slow degradation. Bioavailability of PAHs decreases as values for partition coefficients increase. Persistence in the environment is positively correlated with increasing partition coefficients. Based on the low water solubility and high affinity to organic matter, significant leaching of HMW PAHs into groundwater is not expected. The primary removal mechanisms for PAHs in aquatic environments generally shift from volatilization to photochemical reactions to and microbial degradation with increasing molecular weight and increasing partition coefficients (ATSDR, 1989).

Bioavailability of sediment-associated PAHs has been shown to decline with increased contact time (Landrum *et al.*, 1992). Depuraton of PAHs from tissues of aquatic organisms is generally rapidly when they are returned to a clean environment.

Bioaccumulation of low molecular weight PAHs from sediments by *Rhepoxynius abronius* (amphipod) and *Armandia brevis* (polychaete) was similar, however, large differences in tissue concentration between the two species were

measured for high molecular weight PAHs (Meador *et al.*, 1995). The authors concluded that the low molecular weight PAHs were available to both amphipod species from the interstitial water, while sediment ingestion was the more significant route of uptake for high molecular weight PAHs. The authors also indicated that high molecular weight PAHs partitioned to dissolved organic carbon were bioavailable.

Metabolism and depuration of PAHs is more efficient in some species than in others, affecting bioaccumulation kinetics and equilibrium tissue residues (Stegeman and Kloepper-Sams, 1987). According to McCarty *et al.* (1992), the toxic body residue of individual PAHs in tissues ranged from 513 to 4,248 μg/g.

The majority of investigations have shown that most aquatic organisms are able to release PAHs, from their tissues rapidly when they were returned to clean environment. However, Tanacredl and Cardenas (1991) 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 reflect their apparent inability to metabolize PAHs to more water soluble and thus easily secreted polar metabolites.

Understanding of the potential bioaccumulation (and hence potential effects) of PAHs requires knowledge of the enzymatic systems which support metabolism of PAHs. Many aquatic vertebrates (primarily fish) and, to a lesser degree, some invertebrate (polychaetes, crustaceans, and mollusks) species do possess the enzyme systems required for detoxification, such that the level of exposure of these organisms to PAHs cannot be directly inferred from the PAH exposure concentrations (National Research Council of Canada, 1983). Such enzymatic systems have also been observed in some bacteria, fungi, and algae.

With respect to PAH activation and carcinogenesis, the National Research Council of Canada (1983, p. 13) states:

> "Structure-activity relationships for mutagenic and carcinogenic activity seem to favor 4-, 5- and 6-ring PAHs rather than smaller or larger compounds. It is believed that PAHs require metabolic activation to exert their carcinogenic effects...with carcinogenesis being initiated by the binding of electrophilic metabolites to critical cellular constituents. Enzymes other than mixed function oxidase (MFO), which may influence the rate of production or destruction of reactive metabolites, are found in aquatic animals and may play an important role in toxicity."

Hence, although the metabolism of PAHs is more common for aquatic vertebrates, significant food chain transfer may occur between invertebrates such as bivalves (*e.g.*, mussels, clams) that do not metabolize PAHs and vertebrates (*e.g.*, seabirds) whose diet may consist of substantial quantities of these prey types. This exposure pathway is addressed in the current investigation as the fourth tier model for

avian aquatic receptors (Figure 3.5-7).

The acute toxicity of hydrocarbons 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. This partitioning 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 (Abernethy *et al.*, 1986). Little is known regarding water quality parameters that may influence toxicity. In studies by Palawski *et al.*, toxicity of benzo(a)anthracene as well as chrysene, and pyrene to striped bass (*Morone saxatilis*) decreased as salinity increased (1985).

PAHs as a group contain a number of individual organic compounds as discussed above and thus may vary in toxicity and ecological effects. According to Eisler (1987b):

> "A wide variety of PAH-caused adverse biological effects have been reported in numerous species of organisms under laboratory conditions, including effects on survival, growth, metabolism, and especially tumor formation. Inter- and intraspecies responses to carcinogenic PAHs were quite variable, and were significantly modified by many chemicals including other PAHs that are weakly carcinogenic or noncarcinogenic. Until these interaction effects are clarified, the results of single substance laboratory tests may be extremely difficult to apply to field situations of suspected PAH contaminants."

Responses to PAHs were observed in sediment tests where total PAH concentrations ranged between 870 and 21,200,000 ng/g (Long *et al.*, 1990). In tests of Puget Sound sediment, statistically significant effects to amphipods, oyster larvae, and Microtox™ were observed with 5,200 ng/g low molecular weight PAHs and between 12,000 and 18,000 ng/g high molecular weight PAHs (Long *et al.*, 1990). Statistically significant effects in the bivalve larval development and amphipod bioassays were observed in tests of sediments from San Francisco Bay with 870 and >15,000 ng/g Total PAHs, respectively (Long *et al.*, 1990). Toxic responses (*i.e.*, >80% amphipod mortality and >44% abnormal larval development) were noted in tests of Commencement Bay sediments with 6,977 and 3,835 ng/g low molecular weight PAHs and with 9,794 and 9,042 ng/g high molecular weight PAHs, respectively (Long *et al.*, 1990). Negative growth was noted in nematode bioassays using Hudson-Raritan Estuary sediments with 42,769 ng/g Total PAHs (Long *et al.*, 1990). Elevated liver somatic condition indices were observed in winter flounder exposed to 228,722 ng/g Total PAHs in spiked sediment tests (Long *et al.*, 1990). Mixed function oxygenase (*i.e.*, P450) induction in winter flounder liver and kidney was elevated in spiked sediment tests with 183,060 and 295,860 ng/g total PAHs, respectively (Long *et al.*, 1990). The 24-hour and 28-day LC50S for the fish *Leiostomus xanthurus* exposed to Elizabeth River sediments, were 530,000 and 21,200,000 ng/g total PAHs, respectively (Long *et al.*, 1990).

ER-L and ER-M sediment values in µg/kg for various PAH CoCs are 16 and 500

for acenaphthene, 44 and 640 for acenapthylene, 85.3 and 640 for anthracene, 261 and 1,600 for benzo(a)anthracene, 430 and 1,600 for benzo(a)pyrene, 384 and 2800 for chrysene, 63.4 and 260 for dibenz(a,h)anthracene, 19 and 540 for fluorene, 600 and 5,100 for fluoranthene, 240 and 1,500 for phenanthrene, and 665 and 2,600 for pyrene, respectively (Long *et al.*, 1995).

Acenaphthene. Acenaphthene is a low molecular weight PAH which has a low log K_{ow} (*i.e.*, 3.92)(Karickhoff and Long *et al.*, 1995).

The acceptable tissue level for acenaphthene, calculated by multiplying the chronic water quality criterion value of 710 ng/gg/L by the bioconcentration factor of 389.05, is 276,222 ng/g (Neff, 1995).

Amphipods, oyster larvae, and Microtox™ exhibited statistically significant responses to acenaphthene in Puget Sound sediment tests at concentrations ranging between 500 and 630 ng/g (Long *et al.*, 1990). A significant toxic response (*i.e.*, 43% mortality) was observed during exposures of the amphipod *Rhepoxynius abronius* to San Francisco Bay sediment with 7.6 ng/g acenaphthene (Long *et al.*, 1990). A highly toxic response was observed during tests using *Rhepoxynius abronius* (*i.e.*, 80% mortality) in sediment from Commencement Bay with 654 ng/g acenaphthene (Long *et al.*, 1990). Significant toxicity was observed in the amphipod *Ampelisca abdita* exposed to Black Rock Harbor sediment with 30 ng/g acenaphthene (Long *et al.*, 1990).

In Puget Sound sediment tests, amphipods, oyster larvae, and Microtox™ exhibited statistically significant responses to anthracene at concentrations ranging between 960 and 1,900 ng/g (Long *et al.*, 1990). Bioassays of San Francisco Bay sediments using bivalve larvae and amphipods indicated significant effects in sediments with 24 ng/g and 1,100 µg/kg anthracene, respectively (Long *et al.*, 1990). Tests of sediments from Commencement Bay and Eagle Harbor, Washington with 363 and 7,597 ng/g anthracene were highly toxic to the amphipod *Rhepoxynius abronius* (Long *et al.*, 1990). In tests with the fish *Leiostomus xanthurus*, the 24-hr and 28-day LC50S for anthracene were 147,840 and 6,600 ng/g, respectively (Long *et al.*, 1990).

The 10-day LC50s for *Eohaustorius estuarius* and *Leptocheirus plumulosus* in water were 374 µg/L and 678 µg/L, respectively (Swartz, 1991). Both amphipods were also exposed to acenaphthene-spiked sediment with total organic carbon ranging from 0.82% to 4.21%. The 10-day LC50S ranged from 1.630 to 4,330 µg/g for *Eohaustorius* and from 7,730 µg/g to > 23,500 µg/g for *Leptocheirus.*

Benzo(a)anthracene. The solubility of benzo(a)anthracene in water is 0.014 mg/L at 25°C (U.S. EPA, 1980a). The log K_{ow} is 5.70 (Karickhoff and Long, 1995).

Mussels exposed to contaminated sediment rapidly accumulated benzo(a)anthracene reaching maximum concentrations at day 20 (Pruell and Quinn, 1987). The concentration factors for mussels exposed to 675 ng/g of benzo(a)anthracene in sediment ranged from 2,470 to 35,700 (U.S. EPA, 1997b).

Benzo(a)anthracene was rapidly taken up by the aquatic plant, *Fontinalis antipyretica* and uptake kinetics plateaued between 48 and 168 h of exposure (Roy *et al.*, 1994). Roy *et al.* suggested that the slow rate of elimination of benzo(a)anthracene from the plant tissue may be due to low aqueous solubility (1994).

Sediment-associated benzo(a)anthracene can be accumulated from two sources: interstitial water and ingested particles. The kinetics of benzo(a)anthracene suggest that uptake occurs via the sediment pore water and by ingested material and that uptake is controlled by desorption from sediment particles and dissolved organic matter (Landrum, 1989).

Benzo(a)anthracene was accumulated in a 24 hour exposure by *Daphnia pulex* mostly from the water, while lower molecular weight PAHs like naphthalene and phenanthrene were accumulated primarily from algal food (Trucco *et al.*, 1983).

Effects of benzo(a)anthracene where observed in bivalve larvae and the fish *Leiostomus xanthurus* when concentrations ranged from 60 ng/g (in tests of sediments from San Francisco Bay) to 350,000 ng/g (in bioassays of sediments from the Elizabeth River), respectively (Long *et al.*, 1990). In tests of Puget Sound sediment, statistically significant effects to amphipods, oyster larvae, and Microtox™ were observed between 1,300 and 1,600 ng/g benzo(a)anthracene (Long *et al.*, 1990). Statistically significant toxicity in the bivalve and amphipod bioassays was observed in exposures to sediments from San Francisco Bay with 60 and 1,100 ng/g of benzo(a)anthracene (Long *et al.*, 1990). Amphipod mortality exceeded 80% in tests of sediments from Commencement Bay and Eagle Harbor where benzo(a)anthracene concentrations were 931 and 11,088 µg/kg, respectively (Long *et al.*, 1990). In tests with the fish *Leiostomus xanthurus,* the 24-hr and 28-day LC50S for benzo(a)anthracene were 196,000 and 8,750 ng/g, respectively (Long *et al.*, 1990).

Benzo(a)pyrene. Benz(a)pyrene is soluble in water (*i.e.,* 0.0038 mg/L at 25°C; MacKay and Shin, 1977), has a half-life of 5.7 days - 1.45 yrs based on aerobic soil die-away test data at 10 - 30°C (U.S. EPA, 1989), a log K_{ow} of 6.11 (Karickhoff and Long, 1995), and a log K_{oc} of 6.01 organic carbon.

Reduced bioavailability has been observed for benzo(a)pyrene accumulation from field-collected sediments compared with laboratory spiked sediments (Varansi *et al.*, 1985). 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 (Harkey *et al.*, 1994). 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 (Harkey *et al.*, 1994).

Short-term exposures (24-h) to 1 mg/L benzo(a)pyrene averaged 8.27 nmol in fish tissue. Of this total, 67% was accumulated in the gallbladder or gut, indicating rapid metabolism and excretion (Goddard *et al.*, 1987). The bioaccumulation of benzo(a)pyrene can be influenced by the lipid reserves (Bruner *et al.*, 1994). In an

experiment conducted by Clements *et al.* (1993), chironomidae larvae rapidly accumulated benzo(a)pyrene from spiked sediment and tissue concentrations was 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 (1983), 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% due to sorption to organic matter (McCarthy, 1983).

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 (Kane-Driscoll and McElroy, 1996). Kane-Driscoll and McElroy (1996) concluded that the body burden of the parent compound may represent less than 10% 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 (Landrum, 1989). Accumulation of benzo(a)pyrene from water was not affected by the simultaneous presence of naphthalene or PCB (Fortner and Sick, 1985).

Trophic transfer of benzo(a)pyrene metabolites has been demonstrated between polychaetes and bottom-feeding fish (McElroy and Sisson, 1989). 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 (Dubroski and Epifanio, 1980) so that the rate of direct uptake by the algae was approximately 20 times faster than the rate of trophic transfer. The authors concluded that the direct uptake and trophic transfer (2 μg/g/day) are equally important in accumulation of benzo(a)pyrene.

Effects of benzo(a)pyrene were observed in bioassays of sediments from San Francisco Bay and Lake Union, Washington, where concentrations ranged from 400 to 220,000 μg/kg (Long *et al.*, 1990). In tests of Puget Sound sediment, statistically significant effects to amphipods, oyster larvae, and Microtox™ were observed between 1,600 and 2,400 μg/kg benzo(a)pyrene (Long *et al.*, 1990). Statistically significant toxicity in the bivalve and amphipod bioassays was observed in exposures to sediments from San Francisco Bay with >1,800 and 1,300 μg/kg of benzo(a)-pyrene, respectively (Long *et al.*, 1990). Amphipod mortality exceeded 80% in tests of sediments from Commencement Bay and Eagle Harbor where benzo(a)pyrene concentrations were 1,192 and 3,485 ng/g, respectively (Long *et al.*, 1990). In tests with the fish *Leiostomus xanthurus*, the 24-hr and 28-day LC50S for benzo(a)pyrene were 55,160 and 2,462 ng/g, respectively (Long *et al.*, 1990).

Benzo(b)fluoranthene. Benzo(b)fluoranthene is a high molecular weight PAH, soluble in water (*i.e.*, 0.0012 mg/L)(Sims and Overcash, 1983) with a half-life of 360 days - 1.67 yrs based on aerobic soil die-away test data (U.S. EPA, 1989). The log K_{ow} is 6.20 (Karickhoff and Long, 1995) and the log K_{oc} is 6.09 L/kg organic carbon.

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 4500 ng/g was established for benzo(b)fluoranthene based on effects in the marine amphipod *Rhepoxynius abronius* (Ingersoll and Nelson, 1990).

Benzo(g,h,i)perylene. Benzo(g,h,i)perylene is a high molecular weight PAH. It is insoluble in water (Pearlman *et al.*, 1984) and has a half-life of 590 days to 650 days based on aerobic soil die-away test data at 30°C (U.S. EPA, 1989), a log K_{ow} of 6.70 (Karickhoff and Long, 1995) and a log K_{oc} of 6.59 L/kg organic carbon.

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 in aquatic organisms (Abernethy *et al.*, 1986). The relatively high theoretical flux through the food chain was not possible to detect.

Benzo(k)fluoranthene. Benzo(k)fluoranthene is a high molecular weight PAH. It is insoluble in water (U.S. EPA, 1995) and has a half-life of 2.49 yrs to 5.86 yrs based on aerobic soil die-away test data (U.S. EPA, 1989). The log K_{ow} is 6.20 (Karickhoff and Long 1995) and the log K_{oc} of 6.09 L/kg organic carbon.

An ecotoxicological in situ study conducted at the Baltic Sea (Broman *et al.*, 1990) 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.

The majority of investigations have shown that aquatic organisms are able to release 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 the effects to the marine amphipod *Rhepoxynius abronius* (Ingersoll and Nelson, 1990).

Chrysene. Chrysene is a high molecular weight PAH which is soluble in water (*i.e.*, 0.0020 mg/L at 25°C (MacKay and Shin, 1977)), has a half-life of 1.02 yrs to 2.72 yrs based on aerobic soil die-away test data (U.S. EPA, 1989), a log K_{ow}: 5.70 (Karickhoff and Long, 1995), and a log K_{oc} of 5.60 L/kg organic carbon.

Responses were observed in amphipod and fish tests of sediments from San Francisco Bay and the Elizabeth River, with chrysene concentrations ranging from 80 ng/g to 317,000 µg/kg, respectively (Long *et al.*, 1990). In tests of Puget Sound sediment, statistically significant effects to amphipods, oyster larvae, and Microtox™ were observed between 1,400 and 2,800 ng/g chrysene (Long *et al.*, 1990). Statistically significant differences in the bivalve larval and amphipod bioassays were indicated when San Francisco Bay sediment concentrations of chrysene were 1,700 and 2,100 ng/g, respectively (Long *et al.*, 1990). Amphipod mortality exceeded 80% in tests of sediments from Commencement Bay and Eagle Harbor, where chrysene

concentrations were 1,363 and 10,574 ng/g, respectively (Long *et al.*, 1990).

Dibenz(a,h)anthracene. Effects were observed when dibenz(a,h)anthracene concentrations were as low as 42 ng/g in bivalve larval bioassays of San Francisco Bay sediments (Long *et al.*, 1990). In tests of Puget Sound sediment, statistically significant effects to amphipods, oyster larvae, and Microtox™ were observed between 230 and 260 ng/g dibenz(a,h)-anthracene (Long *et al.*, 1990). Statistical differences in the bivalve larval and amphipod bioassays with San Francisco Bay sediments were indicated when concentrations of dibenz(a,h)anthracene were 260 and 300 ng/g, respectively (Long *et al.*, 1990). Amphipod mortality exceeded 80% in tests of sediments from Commencement Bay and Eagle Harbor, where dibenz(a,h)anthracene concentrations were 72 and 263 ng/g, respectively (Long *et al.*, 1990). Significant toxicity to bivalve larvae was observed in sediments from Eagle Harbor with 63 ng/g dibenz(a,h)-anthracene (Long *et al.*, 1990).

Fluoranthene. Fluoranthene is a high molecular weight PAH. It is soluble in water (*i.e.*, 0.20-0.26 mg/L (U.S. EPA, 1996)), has a half-life of 140-440 days (U.S. EPA, 1989), a log K_{ow} of 5.12 (Karickhoff and Long, 1995) and a log K_{oc} of 5.03 L/kg organic carbon.

H. azteca accumulated up to 1131 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 (Kane-Driscoll and Landrum, 1995). The data from a draft manuscript indicated that during 30-day bioaccumulation exposures, fed *H. azteca* accumulated significantly more fluoranthene than unfed organisms (Harkey *et al.*, 1997). In the study by Harkey *et al.* (1997), animals accumulated up to 1.4 µmol/g after 30 days in the highest (1004 nmol/g) sediment concentration. In exposures where 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.* (1993) experiments. These data suggest that animals in fed exposures preferentially consumed the food, given the relatively high accumulation of compound in animal tissue.

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 ng/g (Neff, 1995).

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 (Brooke, 1994). 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 (McCarty and MacKay, 1993).

The LC50 values for fluoranthene using freshwater amphipods ranged from 11.7

to 150.3 nmol/g dry weight (Kane-Driscoll and Landrum, 1995). Salinity and particle size of the sediment had no or very little effect on survival of three amphipod species during exposure to fluoranthene (DeWitt *et al.*, 1989).

Previous water-only exposures (Landrum *et al.*, 1991) 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% mortality of *Leptocheirus plumulosus* was 0.69 µmol/g wet wt, which is lower than the predicted critical body residue for nonpolar narcotic compounds (Driscoll *et al.*, 1996).

Bioassays of sediments from southern California and the Elizabeth River indicated significant responses to amphipods at 382 ng/g fluoranthene (Long *et al.*, 1990). In tests of Puget Sound sediment, statistically significant effects to amphipods, oyster larvae, and Microtox™ were observed between 1,700 and 3,900 ng/g fluoranthene (Long *et al.*, 1990). Statistical differences in the bivalve larval and amphipod bioassays with San Francisco Bay sediments were indicated when concentrations of fluoranthene were 2,000 and >3,700 ng/g, respectively (Long *et al.*, 1990). The 24-hour and 28-day LC50S for the fish *Leiostomus xanthurus* exposed to Elizabeth River sediments, were 327,200 and 59,250 ng/g fluoranthene, respectively (Long *et al.*, 1990).

Fluorene. The amphipod *Grandidierella japonica* exhibited a significant response to sediment from southern California with 11 ng/g fluorene (Long *et al.*, 1990). The 24-hour and 28-day LC50S for the fish *Leiostomus xanthurus* exposed to Elizabeth River sediments, were 700,000 and 17,500 ng/g fluorene, respectively (Long *et al.*, 1990). Liver somatic condition indices were elevated in winter flounder exposed to 220,550 ng/g fluorene in spiked sediment bioassays (Long *et al.*, 1990). Mixed function oxygenase (*i.e.*, P450) induction in winter flounder liver and kidney was elevated in spiked sediment tests with 176,510 and 285,290 ng/g fluorene (Long *et al.*, 1990). In tests of Puget Sound sediment, statistically significant effects to amphipods, oyster larvae, and Microtox™ were observed at 540 ng/g fluorene (Long *et al.*, 1990). Statistical differences in the bivalve larval and amphipod bioassays with San Francisco Bay sediments were indicated when concentrations of fluorene were 11 and 210 ng/g, respectively (Long *et al.*, 1990).

Phenanthrene. Phenanthrene is a low molecular weight PAH. Phenanthrene is soluble in water (*i.e.*, 0.6 ± 0.1 mg/L, 22°C (U.S. EPA, 1995)) and a half-life of 16 to 200 days (U.S. EPA 1989). It has a log K_{ow}: 4.55 (Karickhoff and Long, 1995) and a log K_{oc} of 4.47 L/kg organic carbon.

The partitioning between interstitial water and sediment particles increases with sediment aging (Landrum *et al.*, 1992). The increasing partitioning suggests that phenanthrene becomes more tightly bound with increased contact time.

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

(Fuji and Weber, 1995). According to Landrum *et al.* (1994), 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 bioconcentration of phenanthrene by *Hexagenia* was related to the weight of the mayflies (Stehly *et al.*, 1990). 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 ng/g (Neff, 1995).

A high concentration of phenanthrene (0.62 µmol/g) was slightly toxic to the amphipods (12% mortality)(Landrum *et al.*, 1994). 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.* (1989) 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 µg/g (Swartz *et al.*, 1989) (10-day LC50 value), while exposure of amphipods to 2,000 µg/g of phenanthrene in sediment from Eagle Harbor did not produce acute responses.

According to McCarty *et al.* (1992), the toxic (critical) body residue of individual PAHs in tissues ranged from 513 to 4,248 µg/g.

Responses in bivalve larval bioassays were observed using sediments from San Francisco Bay with 88 ng/g phenanthrene (Long *et al.*, 1990). In tests of Puget Sound sediment, statistically significant effects to amphipods, oyster larvae, and Microtox™ were observed between 1,500 and 5,400 ng/g phenanthrene (Long *et al.*, 1990). Amphipod mortality exceeded 80% in tests of sediments from Commencement Bay and Eagle Harbor, where phenanthrene concentrations were 2,838 and 33,603 ng/g, respectively (Long *et al.*, 1990). Significant amphipod mortality (*i.e.*, 67%) was observed in tests of San Francisco Bay sediments with 242 ng/g phenanthrene (Long *et al.*, 1990). The 24-hour and 28-day LC50S for the fish *Leiostomus xanthurus* exposed to Elizabeth River sediments, were 2,363,200 and 105,500 ng/g phenanthrene, respectively (Long *et al.*, 1990). Elevated liver somatic condition indices were observed in winter flounder exposed to 340 ng/g phenanthrene in spiked sediment tests (Long *et al.*, 1990). Mixed function oxygenase (*i.e.*, P450) induction in winter flounder liver and kidney was elevated in spiked sediment tests with 270 and 429 ng/g phenanthrene, respectively (Long *et al.*, 1990).

Pyrene. Pyrene is a high molecular weight PAH. Pyrene is soluble in water (*i.e.*, 0.135 mg/L at 25°C (U.S. EPA, 1995)) and has a half-life of 210 days to 5.2 yrs based on aerobic soil die-away test data at 10-30°C (U.S. EPA, 1989). The log K_{ow} is 5.11

(Karickhoff and Long, 1995) and a log K_{oc} of 5.02 L/kg organic carbon.

The toxic effect of pyrene is believed to occur at a relatively constant concentration within the organism (Abernethy *et al*, 1986). Bioconcentration and depuration of pyrene and its biotransformation products display a clear pH-dependency both in rate and bioconcentration (Wild *et al.*, 1994). 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 (Landrum, 1987a).

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 (Landrum *et al.*, 1992). The increasing partitioning suggests that the compounds are becoming more tightly bound with increased contact time.

Using equilibrium-partitioning theory, the BCF value, and critical body residue (LD50), Landrum *et al.* (1994) calculated the sediment concentration that would produce 50% amphipod mortality. Based on these assumptions, the pyrene concentration of 14.2 μg/g in sediment should produce 50% 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.

Elevated liver somatic condition indices were observed in winter flounder exposed to 360 µg/kg pyrene in spiked sediment tests (Long *et al.*, 1990). Mixed function oxygenase (*i.e.*, P450) induction in winter flounder liver and kidney was elevated in spiked sediment tests with 300 and 182 ng/g pyrene, respectively (Long *et al.*, 1990). In tests of Puget Sound sediment, statistically significant effects to amphipods, oyster larvae, and Microtox™ were observed between 2,600 and 4,300 µg/kg pyrene (Long *et al.*, 1990). Amphipod mortality exceeded 80% and 65% in tests of sediments from Commencement Bay and San Francisco Bay, where pyrene concentrations were 1,820 and 777 ng/g, respectively (Long *et al.*, 1990). The 24-hour and 28-day LC50S for the fish *Leiostomus xanthurus* exposed to Elizabeth River sediments, were 1,350,000 and 33,750 ng/g pyrene, respectively (Long *et al.*, 1990).

5.1.3. Pesticides

DDD and DDE. DDD and DDE, being metabolites of DDT, are very persistent in the environment. DDE is soluble in water (*i.e.*, 0.065 mg/L at 24°C (U.S. EPA, 1996)) and has a half-life of 2.0 - 15.6 years based on biodegradation of DDD in aerobic soils under field conditions (U.S. EPA, 1989). DDE has a log K_{ow} of 6.76

(Karickhoff and Long, 1995) and a log K_{oc} of 6.65 L/kg organic carbon. DDT is very persistent in the environment due to its low vapor pressure, high fat solubility, and resistance to degradation and photo oxidation. DDT is degraded to DDE under aerobic conditions and to DDD in anoxic systems (Charles and Hites, 1987). These metabolites, DDD and DDE, are similar to DDT in both their stability and toxicity. Of the three compounds, evidence strongly indicates that DDE is responsible for most reproductive toxicity in avian species (Blus, 1996). Measurements of residues in eggs of birds are a reliable indicator of adverse effects.

Few specific data are available regarding the environmental fate of DDE; however, both DDT and DDD in water are subject to sedimentation, volatilization, photodegradation, and food web uptake. 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. DDT is absorbed by humans in direct proportion to dietary exposure. Human epidemiological data are not available for DDE, although based on its structural similarity to DDT, it is classified as a probable human carcinogen.

Bioconcentration factors for DDE are from 10^3 to 10^5. Based on the data presented in one study reviewed, BCFs for birds collected from the lower Detroit River ranged from 840,000 to 2,300,000. Concentrations of DDE in birds were 40 to 108 times higher than in sediment. Biomagnification factors of 3.2 and 85 were determined for DDT and DDE, respectively, from alewife to herring gulls in Lake Ontario (Braune and Norstrom, 1989). 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 (Muir *et al.*, 1988).

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 (Blus, 1996).

Statistically significant responses to DDE were noted in tests of sediments from Puget Sound in the amphipod bioassay and in the evaluation of benthic community composition where DDE concentrations were 15 and 9 ng/g, respectively (Long *et al.*, 1990). Statistically significant effects in the bivalve larval development and amphipod bioassays were observed in tests of sediments from San Francisco Bay with 2.2 ng/g DDE (Long *et al.*, 1990). Significant toxicity to amphipods and bivalve larvae were noted in tests of sediments from San Francisco Bay with 1 and 3 ng/g DDE (Long *et al.*, 1990).

The ER-L and ER-M values in sediments for DDE are 2.2 and 27 ng/g, respectively (Long *et al.*, 1995).

DDT. Invertebrate species are generally more susceptible than fish species to effects associated with exposure to DDT in the water column (U.S. EPA 1980b). In general, the low solubility of DDT and its metabolites in water suggests that water

column exposures are likely to be less than exposure from ingestion of food or sediment. Sediments contaminated with pesticides, including DDT, have been shown to impact benthic communities at low concentrations.

The high octanol-water partition coefficient of DDT indicates that it is easily accumulated in tissues of aquatic organisms. Biota Sediment Accumulation Factors (BSAFs) for DDT 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. 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 dover sole collected in southern California ranged from 1.7 to 3.4.

Laboratory studies have shown that these compounds are readily bioconcentrated in aquatic organisms, with reported BCFs for DDT ranging from 1,200 to 4,430,000 and for DDE ranging from 63,500 to 181,000 (U.S. EPA, 1980b).

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. Results of laboratory and field investigations suggest that chronic effects generally occur at total DDT concentrations in sediment exceeding 2 ng/g (Long *et al.*, 1995). Equilibrium partitioning methods predict that chronic effects occur at DDT concentrations in sediment of 0.6 to 1.7 ng/g (Pavlou *et al.*, 1987).

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 (U.S. EPA, 1980b). DDT may also be transferred to embryos from contaminated adults. DDT concentrations of 1.1 to 2.4 μg/g in fish embryos have been associated with fry mortality (Johnson and Pecor, 1969; Smith and Cole, 1973).

Eggshell thinning, embryo mortality, and decreased hatchling survival have been linked to chronic exposure to DDT and its metabolites in the diet of birds. 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.

5.1.4. Polychlorinated Biphenyls (PCBs)

PCBs, also known by the commercial name Aroclors, vary substantially in their chemical, physical, and biological properties based on their degree of chlorination. 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 (Field and Dexter, 1988). 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 usually affect partitioning more significantly, but toxicity is more dependent on position (Phillips, 1986). The less chlorinated Aroclors will sorb less strongly onto sediments than the highly chlorinated components.

Individual PCB congeners are different physically and chemically based on the degree of chlorination and position of chlorine substitution, although differences in the degree of chlorination affect partitioning more significantly, while toxicity is more dependent on position (Fisher *et al.*, 1983). Octanol-water partition coefficients, 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 demonstrate greater dispersion from a point source, whereas the higher-chlorinated compounds remain in sediments closer to the source (Fisher *et al.*, 1983). In addition, the mobility of PCBs in sediment is related to the chlorine substitution pattern and degree of chlorination. It is generally quite low, particularly for the higher chlorinated biphenyls (Pavlou and Dexter, 1979; Fisher *et al.*, 1983). Therefore, high rates of sedimentation could prevent PCBs in the sediment from reaching the overlying water via diffusion (Pavlou and Dexter, 1979; Fisher *et al.*, 1983).

PCB concentrations in sediments are 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 typically contain higher concentrations of PCBs than coarser sediments (Phillips, 1986). Sorption to sediments is a function of total organic carbon content (Chou and Griffin, 1986; Sawhney, 1986).

PCBs are among the most stable organic compounds known, and rates of chemical degradation in the environment are thought to be slow. Generally, highly lipophilic PCBs are found at low concentrations in water and at relatively high concentrations in sediment (Phillips, 1986). PCBs are a class of 209 discrete chemical compounds called congeners, in which one to ten chlorine atoms are attached to a biphenyl. PCBs were commonly produced as complex mixtures of congeners for a variety of uses, including dielectric fluids in capacitors and transformers. In the U.S., Aroclor is the most familiar requested trademark of commercial PCB formulations. 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% chlorine).

Toxicity of PCB congeners is dependent on the magnitude of chlorination as well as the isomer. Lesser chlorinated congeners are more readily absorbed and are metabolized more rapidly than higher chlorinated congeners (Bolger, 1993). 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 may interact with the same receptor as the highly toxic 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) (Erikson, 1993). 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).

Due to their toxicity, high K_{ow} values, and highly persistent nature, PCBs 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 both water and food sources.

Elimination of PCBs from organisms is related to the characteristics of the specific PCB congeners. It has been demonstrated that uptake and depuration rates in mussels are high for lower-chlorinated PCBs and lower for higher-chlorinated congeners (Tanabe *et al.*, 1987; Lech and Peterson, 1983). Elimination of PCBs from the body can occur during egg production and spawning in females of some organisms (Long and Morgan, 1990). Fish and other aquatic organisms have limited ability to biotransform or metabolize PCBs.

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 (Phillips, 1986). The bioconcentration factor for fish is approximately 50,000 (Neff, 1984). This factor represents the ratio of concentration in tissue to the ambient water concentration. PCB concentrations in tissues of aquatic organisms are generally greater than, or equal to, sediment concentrations (Pruell *et al.*, 1986). PCB concentrations in fish have been correlated to lipid content in fish tissues.

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. BSAFs for total PCBs were calculated for red-winged blackbird and tree swallow eggs during a study in the Great Lakes area; 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.

The bioconcentration factor for fish is approximately 50,000 (U.S. EPA, 1980c). A BCF of 4,170 was measured for perch in a Swedish lake. In a study of several lakes in central Ontario, BAFs for fish ranged from 0.13 to 27, as reported in the summary table. PCB concentrations in tissues of aquatic organisms will generally be greater than, or equal to, sediment concentrations (Neff, 1984). 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.

The most toxic congeners have been shown to be selectively accumulated from organisms at one trophic level to the next (Jones *et al.*, 1993). 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 (Biddinger and Gloss, 1984; Kay, 1984; U.S. ACE, 1995). The results from Biddinger and Gloss (1984) and U.S. ACE (1995) generally agreed that highly water-insoluble compounds (including PCBs) have the potential to biomagnify in these types of food webs. Thomann's (1989) model also indicated that highly water-insoluble compounds ($\log_{10} K_{ow}$ values 5 to 7) showed the greatest potential to biomagnify. A biomagnification factor of 28 was calculated by Wiemeyer *et al.* (1993) for transfer of total PCBs from fish to bald eagle eggs.

Oliver and Niimi (1988), 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.* (1990) 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, biomagnification factors for transfer from zooplankton to fish ranged from 0.1 to 9.3, as reported in the attached summary table for total PCBs.

PCBs as a group contain a number of individual congeners which vary with respect to toxicity. Exposure to PCBs in various combinations has resulted in effects on growth of phytoplankton through impairment of photosynthesis and cell division, and has been shown to influence competitive interactions between phytoplankton species (Mosser *et al.*, 1972; Fisher *et al.*, 1974). PCBs also affect reproduction in fish (Hansen *et al.*, 1974), growth in bivalves (Parrish *et al.*, 1972), molting physiology of crustaceans (Fingerman and Fingerman, 1977), and may adversely affect population dynamics in fish (Munns *et al.,* 1997). Hansen *et al.* (1974) demonstrated the adverse influence of PCB exposure (as Aroclor 1254) on recruitment and development of benthic and epibenthic estuarine communities in laboratory exposure systems. At high enough concentrations, PCBs cause death in a number of estuarine organisms (Hansen, 1974).

Responses to PCBs were observed in tests of marine sediments where concentrations ranged between 36.6 and 10,800 ng/g (Long *et al.*, 1990). In tests of Puget Sound sediment, statistically significant effects to amphipods, oyster larvae, and Microtox™ were observed between 130 and 2,500 ng/g PCBs (Long *et al.*, 1990). Statistically significant effects in the bivalve larval development and amphipod bioassays were observed in tests of sediments from San Francisco Bay with 54 and 260 µg/kg PCBs, respectively (Long *et al.*, 1990). Toxic responses (>80% amphipod mortality and >44% abnormal bivalve larval development) were noted in tests of Commencement Bay sediments with 38 and 368 ng/g PCBs, respectively (Long *et al.*, 1990). Negative growth was noted in nematode bioassays using Hudson-Raritan Estuary sediments with 638 ng/g PCB (Long *et al.*, 1990).

Sediments 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 ng/g (Mearns *et al.*, 1991). The LC50 for grass shrimp exposed to PCBs in marine waters for 4 days was 6.1 to 7.8 μg/L (Eisler, 1986). Chronic toxicity of PCBs presents a serious environmental concern because of their resistance to degradation. However, 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 (Eisler, 1986).

The ER-L and ER-M values for total PCBs in sediments are 22.7 and 180 ng/g, respectively (Long *et al.*, 1995).

5.1.5. Dioxins

Sediment quality criteria guidelines for protection of ecological receptors recommended by the U.S. EPA are 60-100 pg/g dry wt for fish, 2.5-25 pg/g for mammals, and 21-210 pg/g for birds (U.S. EPA, 1993d), representing low to high potential risk ranges. Corresponding acceptable tissue concentrations are 50-80 pg/g in fish, 0.7-7 pg/g in mammals and 6-60 pg/g in birds. These values were developed using a tissue residue based approach, which incorporates information on steady state body burdens, acute and chronic toxicity, and partitioning between sediment and tissues. The method supercedes the earlier approach which was based on analytical detection limits.

Much of the impetus for development of guidelines is the concern over the tendency of dioxins to bioaccumulate in fish and wildlife. Biota-sediment accumulation factors (BSAF) between sediment and biota range from 0.3 (fish) to 0.1 (mammals and birds). Despite recent attention to development of the guidelines, relatively little relatively limited data exists on the toxicity of dioxins to aquatic biota. Munns *et al.* (1997) conducted a modeling study to evaluate the effects of dioxins and PCBs on mummichog fish (*Fundulus heteroclitus*) populations in the New Bedford Harbor estuary. Decreased population growth rate was predicted when dioxin body burn concentrations exceeded 125 pg/g dry weight. The value compares favorably to the high end range recommended for fish.

Most recently, the World Health Organization (WHO, 1998) has recommended Toxicity Equivalency Factors for fish, mammals and birds. TEFs are constants multiplied against dioxin congener values in sediment or tissue to predict an equivalent concentration of 2,3,7,8-TCDD, the form of dioxin considered to be the most toxic. The sum of 2,3,7,8-TCDD equivalents is called the Toxicity Equivalency (TEQ) concentration. TEQ values are derived separately for fish, mammals and birds and compared against sediment or tissue residue benchmarks discussed above.

5.1.6. Conclusions

In summary, the CoCs identified in Section 3.3 can be characterized by their