Fate and Effects Associated with Contaminants Released from Pressure-Treated Wood into Sensitive Environments

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This chapter will discuss the fate of biocides leached from pressure-treated wood into aquatic environments and the effects those contaminants can have on aquatic fauna and flora. The fate of contaminants is considered important because it affects their persistence in the environment and their bioavailability to aquatic organisms. Available sediment and water quality criteria (SQC and WQC) will be reviewed and benchmarks will be recommended when criteria are not available. Understanding these criteria is important because they form the basis for evaluating the suitability of site-specific projects through the risk-assessment process described in Chapter 9. In addition, these criteria are somewhat general in nature in that they are protective of the most sensitive species and therefore necessarily contain significant safety factors. However, the criteria must be carefully considered in light of the special needs of threatened or endangered species or highly valuable communities. The additional discussion provided for some contaminants is intended to assist readers in defining site-specific criteria that are reasonably protective of these sensitive species, communities, or habitats. Some newly developed preservatives rely on combinations of copper and organic pesticides such as propiconazole, tebuconazole, and imidacloprid.

The chapter includes reviews of studies undertaken to better understand the additive, synergistic, or antagonistic action of these combinations of pesticides and the naturally toxic wood extractives released from untreated and treated lumber, timbers, and piling. Sediment- and water-quality criteria have not been established for some of these organic biocides and the bioassays also help fill this gap. This information is not presented in lieu of existing regulatory criteria, but rather as an interim means of assessing risk while those criteria are being developed. Chapter 5 is organized to discuss first the fate and then the effects of each of the following active ingredients: polycyclic aromatic hydrocarbons (PAH), copper, arsenic, chromium, zinc, copper naphthenate, pentachlorophenol, didecyldimethylammonium chloride (DDAC), tebuconazole, propiconazole, and imidacloprid.

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5.1 Polycyclic Aromatic Hydrocarbons

Polycyclic aromatic hydrocarbons (PAH) form a family of compounds whose fates and effects are different for the major classes of PAH. In water, PAH evaporate, disperse into the water column, become incorporated into bottom sediments, and concentrate in aquatic biota, where they are oxidized and/or biodegraded. The following discussion will focus on PAH derived from pressure-treated wood.

5.1.1 Fate of PAH in aquatic environments

As discussed in Chapters 7 and 9, the suite of PAH in pressure-treated wood is dominated by intermediate weight compounds. These compounds have low water solubility (S_w) and were not found in significant dissolved concentrations near creosote-treated wood by Goyette and Brooks (1998, 2000), Colwell and Seesman (1976), or Wade et al. (1988). The reasons for these low dissolved concentrations are found in the physicochemical properties of the various PAH compounds. Low molecular weight (<200 g/Mole) PAH or "LPAH", such as naphthalene(s) (S_w = 32 mg/L), acenaphthene (S_w = 3.42 mg/L), fluorene (S_w = 1.69) and phenanthrene (S_w = 1.00) were the dominant compounds found in concentrations of >1.0 ng/L within 0.25 m of the Sooke Basin (British Columbia) "BMP dolphin" by Goyette and Brooks (1998). Concentrations of these LPAH, detected by using semi-permeable membrane devices, varied between 4.57 and 7.17 nanograms/liter (ng/L). Of the intermediate- and high molecular weight

PAH, only fluoranthene (M.W. = 202) was detected in the water at a maximum concentration of 3.70 ng/L. All higher molecular weight compounds were either below detection limits or were detected in concentrations < 1.0 ng/L. The ∑PAH detected at the open control station was 13.4 ng/L and the sum of all the detected PAH within 0.25 m of the creosote-treated dolphin was <30.8 ng/L. Another characteristic of LPAH is that they have relatively high vapor pressures (P_v = 2.3 to 45,466 mPa), whereas the HPAH do not significantly vaporize with P_v = 0.00001 for the seven ring compounds to 0.7 mPa for fluoranthene.

Based on the results for water and sediment PAH analyses presented by Goyette and Brooks (1998), the authors hypothesized that most creosote-derived PAH is not dissolved and subsequently adsorbed to particulates, but rather is transported to sediments as microparticles or micro-liter-size droplets. Preliminary laboratory studies (Brooks, unpublished data) have supported this hypothesis. Micro-liter quantities of PAH released beneath the air-water interface settled to the bottom of graduated cylinders filled with sterile 30 PSU seawater with speeds that appear consistent with those predicted by Stokes' Equation. Furthermore, the particles settled into either quartz sand or crushed oyster-shell substrates and remained intact for at least 2 y. Small quantities of creosote oil injected above the air-water interface formed sheens on the water's surface. These sheens remained intact until the water was disturbed, as would be occur with waves. The sheen then broke up into small, irregularly shaped particles that settled to the bottom and worked their way into the sediments. This hypothesis, if substantiated, will significantly change our approach to assessing the toxicity of creosote-contaminated sediments. Infauna, rather than being subjected to an environment that is uniformly contaminated by a diffuse pattern of PAH, would be confronted with an environment that is dominantly uncontaminated with foci of high contamination. In this scenario, exposure is best described stochastically with consideration for possible avoidance or attraction to the PAH foci by various organisms. This hypothesis would explain the patchiness of PAH concentrations found in association with PAH-contaminated sediments. It would also help explain why mixtures of PAH, like creosote, are found to be less toxic than an additive toxicity assumption would predict (Tagatz et al. 1983, California EPA 1994). Lastly, this hypothesis would explain the presence of cm^2 microsheens observed at sediment depths of 4 cm in Sooke Basin. Axelman et al. (1999) observed 10 times more PAH in the colloidal phase and 5 times more PAH in the particulate phase than in the dissolved phase. The concentration of PAH in the dissolved phase represented less than 10% of the PAH in the water column. Wade et al. (1987) found creosote-associated polynuclear aromatic hydrocarbons only in surface sheen samples collected at the Charlestown Navy Yard. They found no creosote-associated PAH in the water column immediately adjacent to the piers. In addition, no observable response was seen in sea urchin (*Arbacia punctulata*) bioassays using water from either Piers #2 or #4. All of this evidence indicates that creosote-derived PAH do not readily dissolve in water.

5.1.1.1 Fate of PAH in the water

Borthwick and Patrick (1982) estimated the chemical and biological half-life of dissolved components of marine grade creosote at less than one week in laboratory experiments. More recently, Bestari et al. (1998a, 1998b) observed an exponential decline to background concentrations in their 84-d microcosm study. The most important degradative processes for dissolved PAH in aquatic environments are photooxidation, chemical oxidation, and biological transformation by bacteria and animals (Neff 1979, EPA 1980) and/or evaporation of LPAH from surface sheens. The low concentrations of dissolved PAH observed in water, coupled with high degradation rates, suggest that dissolved PAH do not present a significant risk to aquatic organisms.

5.1.1.2 Fate of sedimented PAH

Because of their low aqueous solubility and hydrophobic character, intermediate and high molecular weight PAH either remain in a particulate form, as discussed above, or they readily adsorb to particulate materials and solid surfaces in water. The ultimate fate of PAH in sediments is believed to be biotransformation and degradation by bacteria, fungi, and algae (EPA 1980, Borthwick and Patrick 1982, Cerniglia 1984, Boldrin et al. 1993).

Low molecular weight PAH, such as naphthalene, degrade rapidly, while the higher molecular weight PAH, such as benz[α]anthracene and benzo[α]pyrene, are more resistant to microbial attack. Herbes (1981) reported turnover times for naphthalene, anthracene and benz[α]anthracene of 13, 62, and 300 h, respectively. Mueller et al. (1991) found that natural microbial communities mineralized 94% of the low molecular weight PAH in 14 d; only 53% of the high molecular weight PAH was degraded during the same period. They also noted that the most

rapid biodegradation of PAH occurred at the water/sediment interface. This is because prokaryotes oxidize PAH as a first step in metabolism. Deeper sediments usually have reduced oxygen, thus inhibiting microbial metabolism of HPAH.

Sayler and Sherrill (1981) and Cerniglia and Heitkamp (1989) summarized the available literature describing the half-life of PAH in aquatic environments. The results were highly variable and depended on PAH species together with a range of environmental and biological factors, such as temperature, the presence of cometabolites, the nature of the microbial community, and the availability of oxygen. A broad range of bacteria and fungi have been observed degrading LPAH and some HPAH (Grifoll et al. 1994, Stringfellow and Aitken 1994, Cerniglia and Heitkamp 1989). Bacterial communities in polluted areas metabolize PAH more quickly than do communities in unpolluted areas, and LPAH are metabolized more quickly than are HPAH (Herbes and Schwall 1978). Naphthalene has a short turnover time (hours to days), whereas the five-ringed benzo[α]pyrene (BαP) has a long turnover time (years under unfavorable conditions). However, Kanaly and Bartha (1999) demonstrated significant biodegradation of BαP in the presence of complex hydrocarbon mixtures. Crude oil, distillates of heating oil, jet fuel, and diesel fuel supported up to 60% mineralization of 80 µg BαP/g soil in 40 d. Millette et al. (1995) also demonstrated the interdependence and cometabolism of mixtures of creosote-derived PAH following an initial lag time of 5 to 7 d, during which the natural microbial community was selected for those phenotypes capable of efficiently metabolizing PAH. In this study, 60% to 75% of the phenanthrene was mineralized within 30 d. This suggested that in the presence of complex cometabolites, phenanthrene, which comprises 19.4% of new creosote oil, may be rapidly lost from the matrix of PAH that move from treated wood into natural environments.

Bogan and Lamar (1995) showed that white rot basidiomycetes are able to degrade a broad spectrum of intermediate (phenanthrene) and heavier creosote-derived PAH. Mueller et al. (1989) provide an excellent review of bioremediation technologies designed to remove PAH, including the high molecular weight compounds, from creosote-contaminated sites. Ingram et al. (1982) observed that the concentration of creosote in leaching vats increased to more than 700 µg/L in the first 72 h and then decreased to less than 34 µg/L at the end of 20 d. He attributed that decrease to bacterial metabolism of the low molecular weight PAH being leached from the pile sections in his study. Tagatz et al. (1983) noted that creosote concentrations decreased by 42% over an 8-wk period in sediments artificially contaminated as part of their mesocosm studies. They attributed the decrease to microbial metabolism.

Neff (1979) attempted to integrate the degradative processes associated with PAH removal from aquatic environments. He concluded that the residence time of PAH in water is brief. The lower molecular weight aromatics (benzene to phenanthrene) are removed primarily by evaporation and microbial activity. Higher molecular weight PAH are removed mainly by sedimentation and photooxidation. Degradation of PAH by animals in the water column is of minor importance. In nutrient rich, biologically active, aerobic sediments, the degradation of PAH is increased by healthy bacterial and fungal communities. However, in anaerobic sediments, the heavier molecular weight PAH (four through seven rings) may persist for years.

5.1.2 Biological effects of PAH in aquatic environments

Polycyclic aromatic hydrocarbons have likely been present on earth since the first fires ignited organic matter (Chapter 8); microbes, plants, and animals have evolved in their presence. These compounds exist throughout our environment at low concentrations. Microbes, plants, and animals have evolved enzyme systems to catabolize and/or detoxify and eliminate these compounds, which are known to be toxic at elevated concentrations. Increased combustion associated with the Industrial Revolution and modern society has increased environmental concentrations of PAH. Uncontrolled discharge of industrial waste in the 19th and early part of the 20th centuries led to significant accumulations of PAH in some aquatic environments, resulting in significant effects on local biota. The question addressed in the following discussion is not whether PAH should be eliminated from our environment, but rather, "At what concentrations do PAH present unacceptable risk to aquatic resources?"

As will be seen, laboratory studies conducted under unrealistic conditions can lead to the identification of *effects per se*. These are effects that can be measured, but that in the real world do no necessarily compromise either individual organisms or communities of organisms. The interaction of sunlight with human skin is an example. Hard work in the sun can cause a person to sweat. Too much exposure to the sun's UV spectrum can lead to

sunburn and eventually to skin cancer. It is quite possible to correlate the amount of sweat with the incidence of skin cancer. Assuming that there is no lower-effect level for carcinogens could lead to the assumption that all sunlight or sweat should be avoided. Obviously, this would be a nonsensical conclusion leading to significant adverse health effects. The observation of sweat is an *effect per se;* some sunlight is not only tolerable, it is essential to human health. This discussion may seem intuitive. However, several instances of published *effects per se* have been used to assert the need for unrealistic PAH benchmarks that unnecessarily restrict efforts to sustain the human population.

5.1.3 Bioconcentration and bioaccumulation of PAH in aquatic environments

Bioconcentration and bioaccumulation of contaminants is of special importance because some aquatic species, most notably bivalves, have demonstrated an ability to rapidly bioconcentrate contaminants in water to high tissue levels. The concern is that persistent contaminants may move up the food chain, biomagnifying to higher concentrations in each trophic level, until contaminants found at non-toxic levels in the ambient environment reach concentrations where they do cause stress and disease at higher trophic levels. For effective biomagnification and movement of contaminants through the food chain, several conditions must be met. First, organisms must have the ability to bioconcentrate low levels of contaminants from the water column or their food.

Second, these contaminants, or their toxic metabolic intermediates, must be retained, unaltered, in the organism until it falls prey to an animal at a higher trophic level. There are a number of factors that mitigate against biomagnification. If contaminants are not absorbed, they cannot accumulate. Numerous organisms, particularly vertebrates, have the ability to either metabolize or to excrete contaminants. The gut, liver, kidney and gall bladder are common sites of PAH concentration, metabolism and excretion in vertebrates. If the contaminants are either rapidly excreted or metabolized to non-toxic compounds, then the chain is broken and biomagnification is not effective in passing contaminants upward through the food chain. DDT is an excellent example of a persistent compound that was bioconcentrated from low levels in the water to higher levels: first in plankton, then in fish, and finally in bird populations, with adverse consequences.

Neff (1982) reported that all species of marine organisms studied to date rapidly bioconcentrate PAH. Bivalve mollusks, particularly the commercially important mussel (*Mytilus edulis*) and oysters of the genera *Ostrea* and *Crassostrea,* have received more attention than other invertebrates, plants, or fish. They are excellent subjects for monitoring pollutants because they are numerous and widespread and because they filter substantial quantities of water over large and highly permeable gills. For these reasons, mussels have been the subject of a number of studies, such as the Global Mussel Watch Program. Many of these studies have focused on the accumulation of metals and the highly carcinogenic molecule benzo[α] pyrene (BαP). Benzo[α]pyrene levels recorded in Neff (1979) for uncontaminated areas fall in the undetectable to perhaps 50 µg/kg range. Dunn and Stich (1976, cited in Dunn and Stich 1979) recorded tissue concentrations averaging 59 µg/kg in areas associated with marinas, and even higher levels, averaging 402 µg/kg, in organisms living on creosote-treated piling. Dobroski and Epifanio (1980) found that direct uptake of BαP from seawater by diatoms was much greater than the rate of trophic transfer from the diatoms to clam larvae. Eisler (1987) recorded elevated PAH concentrations, especially benzo[α] anthracene, chrysene, fluorene, phenanthrene, and pyrene in oyster tissues and sediments from the vicinity of marinas. These levels were notably higher in cooler months, when lipids and glycogen were being stored preparatory to spawning (Marcus and Stokes 1985).

For mussels, the general trend towards lower levels of HPAH relative to the levels in associated sediment suggests an uptake mechanism that involves the solution of PAH in water. Supporting this hypothesis is the observed rapid turnover and shorter half-life of the more soluble LPAH (Dunn 1980 in Eisler 1987). This suggests that the more soluble (and more bioavailable) PAH are effectively removed from sediments and metabolized by bivalves. The HPAH (associated with chronic stress and genetic disorders) remain in the sediments because of their low solubility. Polycyclic aromatic hydrocarbon levels in fish are usually low because this group rapidly metabolizes all PAH (Lawrence and Weber 1984; West et al. 1986a, 1986b) or excretes them. High concentrations of PAH are typically found in the gut, liver, and bile. Raw fish from unpolluted or moderately polluted water seldom contain detectable amounts of PAH; however, smoking and cooking of fish can greatly increase PAH content. The smoky, crunchy taste people enjoy in barbequed food is very high in PAH.

Neff (1982) reported bioconcentration factors (BCF) for several PAH in the clam *Rangia cuneata*. It should be emphasized that the BCF values, which range from 6.1 to 32, are for PAH dissolved in water. Eisler (1987) has summarized BCF values from the literature. The BCF values reported in his own paper contradict his assertion that bivalves accumulate PAH more rapidly than do fish. For all of the values given in his review, the averages are as follows: bivalves 82 (n = 8); fish 6,844 (n=34). (Note: Eisler's 1987 paper reported bioconcentration values from 6 to 236 in the clam *Rangia cuneata*. Four of the five values were less than 33. For fish, bioconcentration values ranged from 44 to 82,916 with most values in the 100–1000 times range.)

5.1.3.1 Bioconcentration of PAH from sediments

The ultimate fate of most HPAH in aquatic environments is sedimentation. Roesijadi et al. (1978) examined the accumulation of Prudhoe Bay crude oil and specific PAH from oil-contaminated sediments by three infaunal invertebrates, the sipunculid worm *Phascolosoma agassizii* and the clams *Macoma inquinata* and *Protothaca staminea*. They found that the efficiency of PAH uptake from sediments was much lower than from water. Bioconcentration factors for uptake of the four PAH from contaminated sediments were 0.2 or less, indicating no significant bioconcentration of PAH by this route. However, bioconcentration factors for uptake of these four PAH from seawater were in the 10.3 to 1349 range, indicating a low to moderate potential for bioconcentration.

Eisler (1987) suggests that bivalves readily take up PAH from sediments. This hypothesis is contradicted by the results of numerous studies. O'Connor (1991) found that at 117 National Status and Trend Sites where there were both mollusks and fine-grained sediments, the average ratio of mollusk tissue to sediment concentration was only 1.2 for total PAH. He also noted that mollusks accumulate the low molecular weight (and more highly soluble) PAH to a greater extent (2.0) than do the high molecular weight PAH (0.64). Eaton and Zitko (1978) reported that PAH concentrations in clams and mussels were two orders of magnitude below those detected in sediments. Neff (1979) cites Perdriau's (1964) finding that in no case did benthic animals contain elevated levels of BαP, compared with sediments collected from the French coast. Tissue concentrations in the animals were, on average, 36% of those in adjacent sediment.

Numerous studies cited above and in Neff (1982) lead to the general conclusion that sediment-adsorbed PAH are not readily assimilated by benthic animals. Accumulation of PAH from sediment, when it occurs at all, may be attributed in large part to the uptake of PAH desorbed from sediment particles into the interstitial water. This hypothesis is supported by Swartz et al. (1989), who concluded that the concentration of chemicals in interstitial water is the primary determinant of sediment toxicity—not the bulk concentration in the sediment.

5.1.3.2 Depuration of PAH

Southworth et al. (1978) found a half life of less than one hour for all PAH metabolized by *Daphnia pulex*. Jackim and Lake (1978) reported that the half life of PAH in most bivalves is on the order of 2 to 16 d. These studies suggest that PAH are either rapidly metabolized or excreted—at least by these species.

5.1.3.3 Bioaccumulation from food

Neff (1979) reported that the annelid, *Neanthes arenaceodentata,* had little, if any, ability to accumulate 2-methylnaphthalene from its food. However, the situation is quite different in marine crustaceans and fish, where uptake from food was much more efficient than uptake from water. Arthropods (crabs, amphipods, shrimp, etc.) rapidly accumulate the lighter weight PAH and very rapidly excrete or metabolize these compounds. The half-life of BαP in *Callinectes sapidus* was 6 d. Neff's (1979) conclusion was that all results demonstrated the importance of metabolism in eliminating PAH by crustaceans. Broman et al. (1990) examined the trophic transfer of PAH in a study involving seston, the blue mussel (*Mytilus edulis*), and the eider duck (*Somateria mollissima*). Contrary to biomagnification, they observed decreasing PAH concentrations with increasing trophic levels.

5.1.4 Summary

Aquatic organisms are able to efficiently bioconcentrate PAH from the water column. It appears that direct transfer from sediments to organisms living within and on those sediments is minimal. Benthic organisms rarely contain higher concentrations of PAH than are found in the sediments in which they live. PAH are rapidly metabolized and excreted by vertebrates and arthropods. Bivalves do not as efficiently metabolize PAH. However, even in bivalves, the half-life of most PAH examined was in the range of 2 to 16 d. These data suggest that PAH are not persistent in

the tissues of aquatic species and that the movement of PAH through the food chain to higher trophic levels is minimal, if it occurs at all. Neff (1979) concluded:

> From the limited data available, it would appear that there are large interspecific differences in ability to absorb and assimilate PAH from food. Polychaete worms have a very limited ability to absorb and assimilate PAH, whereas fish absorption of PAH from the gut is limited and variable depending on species of fish, the PAH, and possibly the food matrix in which PAH is administered. Crustaceans, on the other hand, apparently readily assimilate PAH from contaminated food. In all cases where assimilation of ingested PAH was demonstrated, metabolism and excretion of PAH were rapid. Thus, the potential for food chain biomagnification of PAH seems to be limited. For such biomagnification to occur, the contaminant must be readily absorbed from food, and once assimilated, it must be relatively resistant to metabolism or excretion.

5.1.5 Cellular response of animals to PAH

The ability to metabolize high molecular weight PAH (HPAH) varies significantly between phyla. Among invertebrates, mollusks have reduced aryl hydrocarbon hydroxylase (AHH) activity and a limited ability to metabolize HPAH. Arthropods and annelids show increased AHH activity, and some marine crustaceans have demonstrated significant cytochrome P-450, mixed-function oxygenase (MFO) and AHH activity. Vertebrates, including fish, demonstrate higher MFO, AHH, and cytochrome P-450 capabilities (Varanasi 1989). The liver is the primary site of MFO activity in fish, and the liver, gut and gall bladder are primary sites of PAH concentration, metabolism, and excretion. In crustaceans, the hepato-pancreas, green gland (an excretory organ), pyloric stomach, gills, testes, and eyestalks are major sites of PAH accumulation and AHH enzyme activity. Melanomacrophage centers are an integral part of the teleost immune system. Payne and Fancey (1989) observed that the numbers of melanomacrophage centers were increased in the livers of fish exposed to total PAH (ΣPAH) concentrations between 25 to 50 µg ΣPAH/g. Payne et al. (1988) observed changes in MFO enzyme levels and liver fat content in fish exposed to dissolved hydrocarbon concentrations of 1000 µg/L (perhaps even as low as 200 to 300 µg ΣPAH/L). The increased levels of P-450, MFO, and AHH enzymes in fish and crustaceans exposed to sediment PAH suggest active catabolism of these molecules. However, enzyme induction is an example of an *effect per se*.

5.1.5.1 Acute toxicity associated with dissolved PAH in marine environments

A common measure of acute toxicity is the concentration of a toxicant that causes 50% mortality in a test population within some specified period of time (96 h). This parameter is referred to as the 96-h LC_{50}. Borthwick and Patrick (1982), and Neff (1979) reported 96-h LC_{50} values for PAH in several marine animals. These are summarized in Table 5.1. Interestingly, in Neff's (1979) discussion of the effects of PAH on aquatic animals, he cites Caldwell et al.'s (1977) finding that continuous exposure to dissolved naphthalene concentrations of 19 to 170 µg/L had no effect on the survival of Dungeness crab (*Cancer magister*) larvae. No explanation was presented for the different (8 µg/L) value for the same species and life stage given in Neff's (1979) paper. One might expect that exogenous factors contributed to the differences. The LC_{50} values reported in the literature for most organisms and PAH compounds are in the 500 to 5,000 µg/L range. Neff (1979) found that in all but a few cases, the concentrations of dissolved PAH that are acutely toxic to aquatic animals are several orders of magnitude higher than concentrations found even in the most heavily polluted marine and fresh waters. However, sediments from polluted regions may contain PAH at concentrations similar to or higher than acute toxicity thresholds. The limited bioavailability of sediment-adsorbed PAH undoubtedly renders them substantially less acutely toxic than dissolved PAH. He also noted that PAH-induced stress is cumulative and is exacerbated by exogenous stress factors such as abnormal thermal and osmotic conditions.

Table 5.1 Acute toxicity of various PAH to marine organisms as measured by 96-h LC_{50} values. All values are in µg/L.

Species	96-h LC_{50}
Mysids (*Mysidopsis bahia*)[a]	18 to 21
Oysters (*Crassostrea virginica*)[a]	700
Pink shrimp (*Penaeus duorarum*)[a]	240
Sheepshead minnows (*Cyprinodon variegatus*)[a]	3,500
Mosquito fish (*Gambusia affinis*)[a]	150,000 naphthalene
Mosquito fish (*Gambusia affinis*)[b]	1,180,000 toluene
Dungeness crab larvae (*Cancer magister*)[b]	8 naphthalene
Dungeness crab larvae (*Cancer magister*)[b]	170 naphthalene

a. Borthwick and Patrick (1982)
b. Neff (1979)

5.1.5.2 Acute PAH toxicity in freshwater

Because PAH heavier than naphthalene are hydrophobic, they are generally found at low concentrations in freshwater and have little potential to create acute or chronic stress in aquatic communities.

Photoenhanced PAH toxicity

The interaction of ultraviolet light (UV) with anthracene and fluoranthene results in modified compounds with increased toxicity to aquatic organisms—at least in laboratory experiments (Foote 1968, Bowling et al. 1983). Landrum et al. (1987) reported photoenhanced anthracene LC_{50} of 12 µg/L in bluegill sunfish and 1.2 µg/L for *Daphnia pulex*. The authors were unsure as to whether the UV light sensitized the target tissues or modified the anthracene to a more toxic compound. The observed toxicity was reported to be 400 times greater in the presence of UV. Davenport and Spacie (1991) extended these results by demonstrating increased toxicity to *Daphnia magna* associated with a suite of PAH extracted from Lake Michigan sediments. These authors reported that exposure of the sediment elutriates to UV did not result in increased toxicity in subsequent bioassays. Increased toxicity was observed only when the daphnids were cultured in the presence of PAH-contaminated elutriate and UV. Concentrations of PAH in these tests were not reported.

Krylov et al. (1997) described a quantitative structure-activity relationship model (QSAR) predicting the photoenhanced toxicity of 16 PAH. Their model suggested that photoenhanced PAH toxicity is a function of several factors including the length of exposure to PAH and UV, the relative absorbance of simulated solar radiation (SSR) by each PAH, the resulting quantum yield for formation of triplet-state PAH, and the rate of PAH photomodification. They found that toxicity associated with nine PAH compounds was dominated by the PAH modification constant and that the photosensitization constant was more important in describing toxicity for the remaining seven PAH. This work suggested that photoenhanced PAH toxicity is a function of the particular PAH compound's propensity for modification to a more toxic photoenhanced form and of the susceptibility of target organisms (or tissues) to photosensitization. The photosensitization constant is likely taxa- and life-stage specific. This model provides relative toxicity data and not absolute data upon which to determine numerical estimates of toxicity. The authors concluded that photosensitization of target organism tissues and photomodification of PAH contribute additively (not synergistically) to photoenhanced PAH toxicity.

Gala and Giesy (1992) reported UV enhanced anthracene toxicity in the green alga (*Selenastrum capricornutum*). The 22-h EC_{50} for specific growth rate ranged from 37.4 to 3.9 µg anthracene/L, depending on the intensity of UV-A radiation. Huang et al. (1993) observed similar results for the higher plant *Lemmna gibba* exposed to anthracene, phenanthrene, or benzo[α]pyrene in the presence of UV or simulated sunlight. These authors reported the relative toxicity of anthracene to be greater than that of phenanthrene; both were more toxic than photomodified benzo[α]pyrene. Growth inhibition was reported at values exceeding thresholds of ca. 200 µg anthracene/L, 500 µg phenanthrene/L, and 3,000 µg benzo[α]pyrene/L. The comparatively lower toxicity of phenanthrene (when compared with anthracene) was substantiated by McConkey et al. (1997), who hypothesized that the photoenhanced toxicity of phenanthrene is associated with the intermediate product phenanthrenequinone. These authors reported an EC_{50} of 3,500 µg phenanthrene/L in *Lemna gibba* in simulated solar radiation and 10,800 µg phenanthrene/L in visible light (no UV). In contrast, the EC_{50} for the photomodified compound phenanthrenequinone was independent of the presence of UV at 530 to 570 µg/L.

Ankley et al. (1995) demonstrated that increased UV-enhanced fluoranthene toxicity to *Lumbriculus variegatus* was a function of both dissolved PAH concentration and UV intensity. Oligochaete mortality increased above ca. 29 µg fluoranthene/L in low UV environments. Acute toxicity thresholds were lower under medium light intensity (8 µg/L) and lowest under high intensity UV (75.2 mW/cm^2 UV-A) radiation at 4 µg/L. These authors noted that *L. variegatus* depurates fluoranthene and that the annelid's physiology includes repair mechanisms that decrease short-term toxicity during periods of darkness. Under medium light intensity (33.5 mW/cm^2 UV-A), mortality did not occur until after 26 h at a fluoranthene concentration of 60 µg/L. This is important because in the real world, sunlight is intermittent—lasting for only about 16 h/d at temperate latitudes. Therefore, these values likely overestimate the photoenhanced toxicity of fluoranthene to this species in the real world. Monson et al. (1999) observed similar responses in larval frogs (*Rana pipiens*) where increasing mortality was observed in exposures to 3.5 µg/L following exposure to intense light for periods greater than 30 h. However, photoenhanced fluoranthene toxicity

was reported only at much higher levels in the same species by Hatch and Burton (1998). These authors reported an EC_{50} of 276 µg fluoranthene/L in *Rana pipiens,* 247 µg/L in *Ambystoma maculatum* and 52 µg/L in *Xenopus laevis.*

This review indicates that photomodification increases the acute toxicity of several PAH. The increased toxicity appears to be caused by photomodification of PAH and photosensitization of target tissues in an additive manner. Photomodified anthracene appears to be more toxic than other PAH, including fluoranthene and phenanthrene. In the absence of ameliorative constituents, the threshold for photoenhanced anthracene toxicity has been demonstrated at 1.2 to 4.0 µg/L. However, humic substances significantly ameliorate photoenhanced PAH toxicity because they absorb UV in the water column.

Acute PAH toxicity to aquatic plants

The effects of various PAH on aquatic plant growth are highly variable. At low concentrations (10–20 µg/L) several PAH act as a stimulant to plant growth. At 300 µg/L, chrysene was observed by Boney (1974, cited in Neff 1979) to induce a 58% increase in the growth of the red alga, *Antithamnion plumula*. Other PAH (anthracene and 2-methylanthracene) caused declines of −20% and −12% in the same alga at 300 µg/L. In general, PAH concentrations greater than 1,000 µg/L inhibit algal growth.

5.1.5.3 Chronic toxicity associated with dissolved PAH

Chronic stress can result in reduced growth, reduced life span, and/or reduced reproductive output. All of these factors can diminish a population's viability. Neff (1979) addressed chronic stress associated with PAH contamination. He cited Ott et al. (1978) and noted that the copepod, *Eurytemora affinis,* suffered statistically significant reductions in the length of life, total number of nauplii produced, and brood size when exposed to 10 µg/L naphthalene, 2-methylnaphthalene, 2,6-dimethylnaphthalene, or 2,3,5-trimethylnaphthalene for the duration of its lifespan. It should be noted that these compounds are the most soluble and therefore the most bioavailable of the suite of PAH found in creosote.

Reduced feeding

Mollusks elicit reduced ventilation (feeding) rates at PAH levels as low as 30 to 40 µg/L in seawater (Moore et al. 1989). The feeding inhibition probably resulted from the narcotic effect of hydrocarbons, particularly aromatic hydrocarbons. These compounds have a direct effect on cilia, muscles, and/or the nervous system, which controls their activity. Reduced feeding rates were likely the cause of significantly reduced growth in mussel populations as a function of proximity to the Sooke Basin creosote-treated dolphins (Goyette and Brooks 1998). It should be noted that these reduced mussel growth rates were associated with ng ∑PAH/L concentrations. However, the authors hypothesized that creosote-derived PAH were transported from the piling to sediments in the form of µL size particles. It is possible that mussels may have filtered these PAH particles, bringing them into intimate contact with their ctenidia, causing the observed reduction in growth rather than the uptake of ng/L concentrations of dissolved PAH across their gills. This hypothesis has not been investigated. Reduced feeding rates result in a reduction in "scope for growth," a commonly measured parameter that quantitatively describes the energy available for tissue growth, reproduction, and activity. In bivalve populations, the major problem caused by reduced scope for growth is poor reproductive output. While this does not have immediate consequences at the organismal level, the long-term consequences of reduced reproductive potential could be significant for the population.

Reproductive effects in herring (*Clupea pallasii*) and mussels (*Mytilus edulis edulis*)

Polycyclic aromatic hydrocarbons are hydrophobic and lipophilic. Thus, there is a potential for these compounds to associate with stable lipid pools in aquatic organisms. Energy is generally stored as glycogen in bivalves until gameto-genesis, when the glycogen and lipid stores are converted into eggs and sperm. The eggs contain significant lipid reserves and could become a repository for lipophilic PAH. Larval herring normally develop in eggs preferentially attached to eelgrass and/or macroalgae, which provide cover and an abundant food supply for the emergent larvae. Absent these important substrates, herring will spawn on any solid surface, including tree branches, rocks, buoys, nets and bags associated with aquaculture, and, in this case, creosote-treated piling. Vines et al. (2000) investigated the effect of creosote-treated wood on Pacific herring in static laboratory conditions. They immersed one gram of creosote-treated wood in 200 ml of 16 PSU seawater and monitored embryonic development, cardiac function, movement, hatching success, and morphology. In these static conditions, herring eggs attached directly to the wood did not survive to hatching. Eggs deposited elsewhere in the beakers containing creosote-treated wood experienced delayed development and decreased

cardiac function. Embryos exposed to creosote also showed increased, but erratic movement within the chorion when compared to controls. Eighty-nine percent of controls hatched; significantly fewer (73%) of the embryos incubated with untreated wood hatched and only 9% of the free-floating embryos in the creosote treatment hatched. The Vines et al. (2000) studies were carried out in the laboratory without the benefit of normal water movement and dilution of PAH. The authors reported an LC_{50} of 50 µg creosote/L with statistically significant reductions in hatching success at 9 µg creosote/L, which is two orders of magnitude higher than concentrations of dissolved PAH observed near creosote-treated structures in Sooke Basin.

Moore et al. (1989) cited Lowe's and Pipe's (1987) observation that long-term exposure to diesel oil at 30 and 130 ppm caused a decrease in the mass of gametes produced by *Mytilus edulis* and *Macoma balthica*. Goyette and Brooks (1998) did not investigate the number of gametes produced by Sooke Basin mussels, but they conducted two reproductive studies (d 185 and d 569) using mussels from the Sooke Basin piling. Larval hatching and development were normal for all mussel treatments, with 65.25% to 89.89% normal larvae to the "D"-hinge stage; the null hypothesis that all cohorts contained equal numbers of normal larvae was not rejected at $\alpha = 0.05$ in an analysis of variance.

Is it possible to reconcile the differences in results presented by Vines et al. (2000) and Goyette and Brooks (1998)? The life cycles and PAH exposure pathways of herring and mussels are very different. Adult mussels accumulate PAH preferentially in lipid rich gametes (Goyette and Brooks 1998). The eggs and sperm of mussels are liberated into the water column and the zygotes drift away from the creosote-treated piling during development. Therefore, PAH exposure is highest in mussel gametes and it decreases as the larvae develop. In contrast, the PAH exposure of herring gametes in the Vines et al. (2000) study was highest following deposition on creosote-treated wood, where the zygotes developed in intimate contact with the PAH. The following points should be emphasized in reconciling these two studies and in evaluating the environmental risks to herring and mussel reproduction associated with creosote-treated wood:

- The static tests conducted by Vines et al. (2000) resulted in high concentrations of PAH in the diluent. These concentrations would have been significantly diluted in open aquatic environments. Reductions in hatching success were reported at PAH concentrations >9 µg/L in their static conditions. This PAH concentration is 9,000/31 = 290 times higher than those observed using semi-permeable membrane devices in the Sooke Basin study—pointing out the difference between static laboratory environments and the real world.
- Vines et al. (2000) did not state how they obtained the 1-g samples of creosote-treated wood. It is difficult to see how the samples could have been obtained while maintaining the original piling surface, which is typically coated with pitch and/or fouling organisms (see Chapter 10) that reduce the migration of creosote from the interior wood structure and provide a barrier between adhering organisms and PAH. Wood chipped or cut from a piling would expose the underlying wood cells, which would lose creosote at a much faster rate than the original piling's surface (see Chapter 7).

The work of Vines et al. (2000) has been examined in some detail because it represents the only published report of adverse effects on marine biota associated with creosote-treated piling. Their study clearly demonstrated adverse effects on herring larvae exposed to high concentrations of PAH in static conditions. It would be interesting to examine the effects on herring larvae spawned onto a piling's fouling community in open aquatic environments. It is possible that piling heavily fouled with macroalgae, mussels, etc., could provide the necessary refuge from predators and the prey base to support larvae spawned in this environment.

5.1.5.4 Summary with respect to acute and chronic dissolved PAH toxicity in aquatic environments

The literature suggests that sustained water-column concentrations of 30 to 50 µg ∑PAH/L can have subtle chronic impacts on populations of marine organisms. These concentrations are approximately 1,000 times greater than those observed in Sooke Basin, and acute toxicity associated with dissolved PAH has not been reported in association with creosote-treated structures located in either open fresh or marine waters. The review completed herein indicates that the lowest toxic thresholds are associated with photoenhanced anthracene at 1.2 to 4.0 µg/L. Photoenhanced toxicity has been demonstrated in the laboratory, but not in the field. Observed concentrations

of dissolved PAH near creosote-treated piling are either not detected or have been detected at <31 ng∑PAH/L. These observations are consistent with the vibrant invertebrate communities that establish themselves on creosote-treated piling (Chapter 10) located outside industrial areas where there are numerous sources of many toxic contaminants. Taken together, this evidence supports a conclusion that dissolved PAH associated with creosote-treated wood products in open aquatic environments pose no acutely toxic threat to biological resources. Dissolved anthracene was detected at 0.00046 µg/L at Sooke Basin and it represented 7.8% of the PAH found in sediments collected 0.5 m from the piling. The risk assessment described herein has traditionally used a dissolved ∑PAH benchmark of 3.0 µg/L, based on the potential for photoenhanced PAH toxicity, and that benchmark is retained.

5.1.5.5 Biological response to sedimented PAH

Empirical evidence indicates that much of the PAH lost from creosote-treated wood in aquatic environments is deposited in sediments near the base of structures (see Chapter 10). As demonstrated in previous sections, adverse biological responses to dissolved PAH are generally inconsequential because these compounds are hydrophobic and do not readily dissolve in water. This hydrophobicity causes PAH to bind with dissolved and particulate organic substances—thereby reducing their bioavailability in sediments (Johnsen 1987, White et al. 1999, Weinstein and Oris 1999, Haitzer et al. 1999). The biological response to sedimented PAH is well documented.

Acute toxicity associated with sedimented PAH

It has long been recognized that it is the concentration of PAH in sediment interstitial or pore water that correlates with toxicity—not the bulk sediment concentration of PAH. Tagatz et al. (1983) found that the lowest creosote concentration affecting the abundance or number of mollusk species at field-contaminated sites was 844 µg creosote/g dry sediment weight (dw), and for echinoderms, annelids, and arthropods, it was <177 µg/g dw. Similarly, Pardma et al. (1998) reported that the median lethal concentration for *Mysidopsis bahia* in the water soluble fraction of creosote extracted from sediments was 700 µg/L, compared with a significantly lower level of 180 µg/L in the water soluable fraction obtained by direct contamination of water without the mediating influence of sediment. Pastorok et al. (1994) reported sediment ∑PAH concentrations as high as 1,800 µg/g associated with a creosote treating plant at an industrial site in Oregon. They observed significant mortality in *Hyallela azteca* bioassays and reduced light emission from Microtox™ within 300 ft of the plant's pier and shoreline. However, significant increases in neoplastic lesions were not observed in the livers of large-scale suckers (*Catastomus macrocheilus*) and no adverse effects on other demersal species were observed outside the highly contaminated nearshore area. Sediments associated with historical industrial activity and spills in Eagle Harbor (Malins et al. 1985), the Elizabeth River (Huggett et al. 1992), the Willamette River (Pastorok et al. 1994), and Bayou Bonfouca (Catallo and Gambrell 1987) have been contaminated with greater than 49,000 µg/g of creosote-derived PAH. Acute toxicity and changes in microbial, meiofaunal, and macrofaunal communities have been associated with these industrial sites, which have received significant study. Acute toxicity has not been demonstrated at sediment concentrations <10 µg∑PAH/g dw in open environments (Baekken 1994, Carman et al. 1995, Wendt et al. 1994, Brooks 2004b, Goyette and Brooks 1998). However, there is evidence of chronic effects, including increased risk of neoplasia, associated with sedimented ∑PAH at concentrations >10 µg∑PAH/g dw. The results of some of this research are reviewed in the following paragraphs.

Mutagenic effects associated with PAH

Colwell (1986) examined mussels and seawater associated with creosoted marine pilings at the Roosevelt Roads Naval Station Complex in Puerto Rico. She employed *Salmonella typhimurium* in the familiar Ames test (Ames et al. 1975) for mutagenicity and found no detectable mutagenic activity in bacteria from either the water or mollusks associated with the creosote. She concluded that the creosote-treated structure did not exhibit any appreciable leaching into the surrounding water.

Neoplasia associated with polycyclic aromatic hydrocarbons

Hyperplastic, preneoplastic and neoplastic lesions have been reported in fish for a number of years. These same types of lesions are less common in bivalves and other invertebrates. Enzymes produced by the cytochrome P-450, MFO, and AHH systems are responsible for initiating catabolism of lipophilic compounds, including PAH. These systems render hydrophobic molecules more water-soluble and therefore increase their potential for excretion and detoxification. In the case of some high molecular weight

PAH, the intermediate metabolic products are mutagenic and/or carcinogenic. For instance the oxidative catabolism of BαP produces arene oxides, some of which bind covalently to DNA and RNA (particularly with guanine). The resulting chromosomal lesions can result in unregulated cell growth and division (cancer). Vogelbein et al. (1990) described hepatic neoplasms in the Mummichog (*Fundulus heteroclitus*) from a site with 22 µg ∑PAH/g dw. Ninety-three percent of the Mummichogs had gross hepatic lesions and 33% of these fish had hepatocellular carcinomas. Similar cellular lesions have been described in fish from a highly urbanized area (the Duwamish River estuary) in Puget Sound (Pierce et al. 1977).

Johnson et al. (1994) and Horness et al. (1998) are considered together here because they rely on essentially the same data. Both papers also rely heavily on the precept that observance of a biochemical response (induction of an enzyme) implies physiological impairment. Brooks (1994) discussed this issue in response to a request from Environment Canada. He concluded that while genotoxicity and enzyme induction tests are useful as indicators of the presence of PAH in aquatic environments, it is inappropriate to conclude that a positive indication in these tests results in physiological compromise of individuals or populations of animals. Enzyme induction is another example of an *effect per se.* In the case of MFO or cytochrome P450 enzyme systems, their induction implies exposure to some forms of carbon—including polycyclic aromatic hydrocarbons. Payne et al. (1988) supported the hypothesis that many point sources of hydrocarbon contamination could be harmful to fish health. They found that MFO enzyme levels were altered at hydrocarbon levels as low as 1.0 ppm. However, the authors noted that PAH levels in this range are encountered over a broad range of aquatic environments, many of which are not associated with pollution. They suggested that hydrocarbons often occur in sufficient concentrations to affect biological responses in fish, and concluded that "meaningful bioindicators must distinguish between effects per se, and either chronic or acute effects." It is important to recall that Johnson et al. (1994) did not find any adverse effects at the population level in their study (see the discussion at the end of their paper). Another concern with Johnson et al. (1994) and Horness et al. (1998) is that they have apparently underestimated sediment PAH concentrations in Elliott Bay (where they reported 10 mg ∑PAH/kg dw), the Duwamish Waterway (6 mg ∑PAH/kg dw), and Eagle Harbor (90 mg ∑PAH/kg dw). Eagle Harbor sediments contain PAH concentrations as high as 6,461 mg ∑PAH/kg dw (Swartz et al. 1989). Washington State Department of Ecology (WDOE 1995) reports sediment concentrations of PAH for a large number of stations in Elliott Bay. Significant areas contain PAH concentrations in the top 4 cm of the sediment column of 111.3 to 593 mg ∑PAH/kg dw. The Puget Sound Environmental Atlas (PSWQA 1992) indicates sediment ∑PAH concentrations at numerous locations in the Duwamish Waterway at greater than 21 mg ∑PAH/kg. In general, higher contaminant concentrations are found in shallow, nearshore water associated with Seattle's intensely urbanized upland and with numerous waterfront docks and industrial facilities. Concentrations of ∑PAH decline in the middle and outer reaches of Elliott Bay (PSWQA 1992). Sediments in these Puget Sound industrial areas also contain high levels of PCBs and metals. Misitano et al. (1994) also reported higher concentrations of HPAH and LPAH than reported by Johnson et al. (1994) and Horness et al. (1998).

The juvenile English sole (*Pleuronectes vetulus*) studied by Johnson et al. (1994) and Horness et al. (1998) are found in shallow water in the intertidal zone where sediment concentrations of all contaminants are generally highest. As they grow, they move into deeper water, but tend to seasonally migrate from deep water in the winter to shallow water in the spring. In British Columbia, English sole are known to make extensive migrations of at least 700 miles (Hart 1973). The point is that English sole in Elliott Bay and the Duwamish Waterway are exposed to a variety of sediment conditions, including ∑PAH concentrations that greatly exceed those reported by the authors. Assuming that sediment concentrations of PAH at the site where the fish were caught are representative of their long-term exposure is like testing the air in a locker room where workers at a chemical plant change their clothes and assuming that the locker room exposure is representative of their exposure to chemicals while working in the plant. Therefore, while the study did find a correlation between exposure to ∑PAH in contaminated bays (and the mix of other contaminants found in these industrial areas) and hepatic lesions, it was not appropriate to quantify the relationship between sediment PAH at the site of collection and the number of cancerous or pre-cancerous lesions observed. To summarize, Johnson et al. (1994) noted some of these problems in their paper. The authors suggested that the results warranted a closer look at the protectiveness of existing sediment quality criteria. They did not suggest that the results were unequivocal and

called for precipitously invoking new sediment quality benchmarks. Lastly it is important to note that the authors could not demonstrate any adverse effect on the population of English sole in their study.

Highly contaminated sediments in Eagle Harbor, Commencement Bay, and Elliott Bay are associated with depositional environments. The sediments are fine grained and, except when disturbed by the thrust of large vessels, they remain undisturbed. These characteristics are commonly associated with most (but not all) contaminated sediments. The bioassay protocol used by Misitano et al. (1994) contains two elements that make it difficult to compare the results with real world environments. First, the authors swirled 20 g of sediment in 800 ml of seawater in one liter glass beakers to begin the bioassays. This likely resuspended the sedimented PAH particles and PAH adsorbed to clay and/or particulate organic matter —greatly increasing its bioavailability in the water column. The PAH particles and fine-grained particulate organic and particulate inorganic matter to which the PAH were likely adsorbed would be the last to settle and would have accumulated on the surface of the well-sorted sediment at the end of 4 h—again unrealistically increasing the exposure of surf smelt larvae to the contaminants. The authors then placed the beakers under continuous fluorescent light at 3240 lux. The photo-enhanced (ultraviolet spectrum) toxicity of anthracene, phenanthrene, fluoranthene, and benzo[α] pyrene is well known to occur at thresholds of 3 to 12 µg/L (Gala and Giesy 1992, Landrum et al. 1987, Ankley et al. 1995). Photo-enhanced PAH occurs at an order of magnitude lower concentrations than is associated with acute toxicity. It has been well documented in laboratory bioassays and in microcosm studies—but not well documented in natural aquatic systems.

The results of this study demonstrate that the observed effects can be associated with larval exposure to a mixture of contaminants in industrial sediments under the conditions imposed (resuspension of contaminated, fine-grained sediments in a small volume of water containing photoactivation PAH. Those conditions are not characteristic of very many depositional areas, which more frequently occur in water deep enough to attenuate incident sunlight and where sediments are infrequently disturbed to the degree implicit in swirling the sediments, as done by the authors. It is not appropriate to infer that the same response is likely in the real world. The literature suggests that there is a reasonable correlation between TPAH concentrations exceeding 10 mg ∑PAH/kg organic carbon and preneoplastic lesions or neoplasia. The bioassay would have been much more realistic had the authors placed the sediments in the beaker and then gently flowed water over them in a flow-through chamber for a period of 12 to 24 h before beginning their bioassay. The purpose of this review is not to diminish the credibility of these studies. As examples of good science, the authors clearly reported what they did and the results they observed. The purpose is to demonstrate that these citations do not provide unequivocal or even reasonable support leading to an assertion of adverse effects in wild populations of fish at PAH exposures of 0.5 to 2.0 mg ∑PAH/kg dw.

Bioindicator studies

Are genotoxicity and enzyme induction tests appropriate indicators of environmental risk? There is growing interest in enzyme induction and genotoxicity tests as indicators of environmental risk making it important to understand what these tests actually tell us. Numerous levels of protection mediate effects at the organismal level from external stressors. Stressful environmental factors such as abnormal temperatures, desiccation, disease organisms, and low dissolved oxygen are often avoided by mobile animals, such as fish, and sessile animals (including many bivalves), which isolate themselves within tightly closed valves in an attempt to avoid harmful conditions. At the next level of protection, an animal's integument isolates internal organs and structures from harmful conditions. The skin and gut epithelia are capable of selective absorption of material. For instance, HPAH, adsorbed to sediments, apparently pass through the digestive tract of many annelids without being absorbed through the gut epithelia. Once foreign materials are absorbed into blood serum through the skin, gills, or gut, organisms respond by sequestering them in vacuoles, metabolizing them in the liver, or cleansing them from the serum as it passes through the kidney. Whether or not a molecule is metabolized or excreted depends, in part, on its ability to penetrate cell membranes. The plasma lemma is highly permeable to essential molecules such as glucose, amino acids, and lipids. The cell's phospholipid bilayers are not very permeable to ions or to large, charged polar molecules. The four- to seven-ring HPAH are generally not charged and therefore they pass across the cell membrane and are actively metabolized by vertebrates. It is well documented that some metabolic intermediates of HPAH, particularly arene oxides, can bind covalently to guanine, producing DNA lesions, which may result in the hepatic lesions (including hepatic carcinomas) reported in demersal fish

associated with PAH contaminated sediments. However, the levels of contamination observed at Eagle Harbor, the Duwamish River, Elizabeth River, etc., at which significant increases in hepatic carcinomas have been observed, are generally greater than 25 to 50 µg/g. In some areas, Eagle Harbor sediments contain ∑PAH concentrations as high as 6,000 µg/g.

Cytochrome P450, MFO, Ethoxy Resofurin-O-Deethylase (EROD), and AHH are important enzyme systems for the metabolism of high molecular weight PAH. There are reports in the literature suggesting that PAH-metabolizing enzyme systems are activated at sediment PAH levels as low as 1.0 µg ∑PAH/g (e.g. Johnson et al. 1994). As previously stated, intermediate PAH metabolites can create DNA lesions. However, DNA contains numerous mechanisms that repair miscoded or damaged sequences. This repair is achieved by a suite of enzymes capable of recognizing damaged or mismatched base pairs and excising them. Environmental and/or random damage to DNA is not unusual and the presence of nicks or double- stranded breaks in nuclear or ribosomal DNA does not often lead to unregulated cell growth. Increased DNA damage obviously increases the risk for failure of these repair mechanisms, resulting in a number of diseases. The point is that there are numerous levels of protection involved in maintaining the biological integrity of an organism. In evaluating environmental risks, the presence and importance of these cellular safeguards must be recognized. The questions we ask must recognize that different levels of biological organization will respond differently to the same level of insult. Therefore, questions must be posed carefully and caution exercised when extrapolating biological responses at one level of organization to responses at another level. Put simply, the sun feels good on our skin and it is necessary for the synthesis of vitamin D. Peel away the skin and expose the underlying tissue to the same beneficial sun and the underlying cells die. Almost anyone would recognize that evaluating sunlight, based on the response of a naked cell, has nothing to do with an organism's response to the same level of light. This may seem simplistic, but these same principles must be applied to genotoxicity tests.

What question is asked by genotoxicity tests? Ernst (1994) reported the results of genotoxicity tests that used subtidal sediments collected at varying distances from a wharf constructed of creosote-treated wood. PAH were extracted from the sediments, dried, and re-dissolved in dimethylsulfoxide (DMSO). Trout hepatocytes were exposed to varying concentrations of the PAH preparation and genotoxicity assayed using the nick translation assay (NTA) of Gagne et al. (1995) and a modified version of the alkaline precipitation assay (APA) described by Olive (1988). The results were quantified by defining a toxicity threshold (TT) as the geometric mean of the lowest observed effect concentration (LOEC) and the no observed effects concentration (NOEC). This test measured the response of DNA in naked digestive gland cells to isolated PAH suspended in a material that is an exceptionally powerful solvent for both polar and nonpolar compounds. DMSO is often used as a reaction medium for bimolecular nucleophilic reactions in which the attacking nucleophile (arene oxide) bears a negative charge. Its use in these genotoxicity studies greatly facilitated transfer of PAH across the plasma lemma and of arene oxides into the nucleus.

Much of what is known about DNA-adduct formation and tumorigenesis is based in studies of mammalian models, including human studies. DNA-adduct formation is clearly evidence of *exposure* of an organism to the adduct parent molecule. Beyond that, literature on binding of xenobiotics to DNA and DNA adduct formation is without consensus on the biological significance of these observations. As illustrated below, the dogma

chemical binding to DNA → mutation → cancer

is known to not be universally operational within species and across species. Participants in an International Life Sciences Institute workshop convened in April 2004, titled, "DNA Adducts: Biological Consequences and Application to Risk Assessment" noted that PAH exposure in rodents can lead to PAH-DNA adduct formation, but no correlation could be found between tissue adduct formation and tumor target tissues. That is, rodent tissues expressing high adduct formation did not correlate with organs in which tumors ultimately formed. Parenthetically, the absence of a correlation between sites of elevated adduct formation and tissue pathology was also seen for non-PAH carcinogens such as acrylamide. Because of the relative ease of obtaining biological samples such as blood, environmentally and occupationally exposed human cohorts have been examined for biomarkers of PAH exposure. Usually DNA adduct or micronuclear bodies (MN) are used as the index of exposure. The majority of this work has concentrated on DNA adduct formation in circulating white blood cells or the presence of micronuclear bodies in white blood or other readily available cells. Work has

shown the method to be characterized by great individual variability and subject to major change from nontarget influences. In the case of PAH biomarkers, diet, and personal habits such as smoking are considered important sources of marker interference, but seasonal differences are known to create a greater change in adduct levels than do personal or occupational exposures (Lewtas et al. 1997). On the occupational side, higher adduct concentrations in people working in the aluminum production industry were measured independently of whether or not subjects were exposed (Eder 1999). For example, variations in occupational exposure to PAH in an aluminum production plant produced a 3.94 elevation factor between workers who had exposure and those who did not. However, the magnitude of the difference between the exposure level of the groups was small, compared to the 13-fold increase seen in the general population in the area of the study between summer and winter heating (Eder 1999).

Although touted as an important index of PAH inhalation exposure, if not evidence of toxicological hazard, DNA adduct formation and MN are not sensitive to cigarette smoking (Karahalil et al. 1999). Lewtas et al. (1997) also showed that environmental exposure can have a much greater effect on DNA adduct formation than occupation exposure. She showed that food, smoking habit, and even seasonal differences created the greatest change in adduct levels and that adduct levels in the aluminum production industry were not linear and in some cases did not correlate with subject exposure. These reports corroborated earlier reports of occupational environmental exposure to PAH typified by Eder (1999), who found that in a PAH-rich environment, DNA adducts correlated with low exposures but not high exposures, or Kubiak et al. (1999), who found that MN could not distinguish between foundry workers and coke oven workers. In Kubiak's work, BαP was elevated in air of coke workers, but MN actually decreased with time of exposure and did not correlate with smoking in any group. Kriek et al. (1998) concluded that variation in measurements of human DNA adduct formation requires that several endpoints be combined for multirational analysis. Clearly, factors in addition to cell cycle time and DNA repair mechanism (discussed below) mitigate expression of genetic lesions as a result of DNA-adduct formation. The consensus for genotoxicity-mediated carcinogenesis involving DNA-adduct formation may not extend beyond the recognition that DNA-adduct formation is necessary, but by itself is not sufficient to cause tumor formation.

Based on the preceding paragraphs, it appears that the question being asked is: "How many DNA nicks and breaks occur when we eliminate, or impair, many of a cell's nuclear defense mechanisms and expose DNA to PAH and their intermediate metabolic products?" This is an interesting question, and, as expected, we find that the degree of DNA insult is proportional to the PAH exposure. In other words, this study revealed a quantifiable dose-response relationship. The dose is isolated PAH and the response is from naked cells whose nuclear and cell membranes have been compromised in the presence of DMSO. Does our current understanding of bioindicator tests allow their use in assessing environmental risks? There are numerous weaknesses in our current understanding. Consider the factors in Table 5.2.

High molecular weight PAH at sufficiently high concentrations can result in disease in demersal fish. The review presented here is intended to provide insight into the mechanisms leading to the observed hepatic carcinomas. However, before these genotoxicity tests can be used to establish environmental criteria, we need to correlate the

Table 5.2 Comparisons between how PAHs behave and potentially affect exposed organisms under laboratory test and field exposure conditions.

Genotoxicity test environment	Real world environment
1. PAH are desorbed and extracted from sediments and dissolved in DMSO	1. PAH are bound to sediments. They are not readily available in a dissolved form and have reduced bioavailability.
2. No organismal epithelium present	2. After desorption from sediments, PAH must cross ectodermal tissues (skin, gills, gut) before entering the blood stream for delivery to the digestive gland.
3. No kidney present to clear PAH	3. Kidney functions to clear some xenobiotics. Fish rapidly excrete most PAH.
4. Plasma lemma compromised by DMSO	4. Cell membranes selectively restrict movement of PAH into the cell. This increases the probability of excretion and decreases the probability of metabolism.
5. Lysosomal membranes compromisedby DMSO	5. Lysosomal membranes help contain intermediate metabolites during metabolism
6. Nuclear membrane compromised by DMSO	6. Nuclear membrane provides another level of protection for DNA
7. DNA lesions assumed to result in unregulated cell growth	7. DNA repair mechanisms reduce the probability of unregulated cell growth.

observed cellular responses with responses at the organismal or population levels of organization. The response of a naked cell, with at least seven layers of protection stripped away, to isolated PAH, does not describe the response of an organ or of whole organisms living in close association with sedimented PAH.

Other bioindicator tests (primarily enzyme induction tests) suffer from the same weakness. The response of an enzyme system to an appropriate substrate has little to do with the response of the organism to that substrate. Bioindicators certainly have a future in environmental studies. However, adequate correlations between cellular or genetic responses and organismal or population responses to pollutant levels have not been made.

Payne et al. (1988) reported a study supporting the hypothesis that many point sources of hydrocarbon contamination could be harmful to fish health. They found that MFO enzyme levels were altered at hydrocarbon levels as low as 1.0 µg ∑PAH/g dw. The authors noted that PAH levels in this range are encountered over a broad range of aquatic environments, many of which are not associated with pollution. They suggested that hydrocarbons often occur in sufficient concentrations to affect biological responses in fish. Consistent with the discussion presented here, they concluded that meaningful bioindicators must distinguish between *effects per se* and chronic or acute effects. The development of simple, timely, and effective tests to evaluate the risks posed by pollutants to aquatic organisms is important work. However, until a better understanding of the correlation between effects observed in bioindicator studies and the response of organisms and populations living in open environments is achieved, bioindicators have little value as either regulatory tools or for assessing environmental health.

5.1.5.6 Effects of PAH contamination on populations of aquatic organisms

Mesocosm studies by Stekoll et al. (1980), Widdows et al. (1982, 1985) reported similar community responses to petroleum and PAH contamination. Significant, long term reductions in the abundance and diversity of invertebrate fauna were reported when concentrations of diesel oil reached 130 µg/L for 2 mo. Less significant population effects were observed on a rocky shore community exposed to 30 µg/L diesel oil for 2 mo. Tagatz et al. (1983) examined the impacts of creosote-contaminated sand on macrofaunal communities. He found that the lowest concentration, at either of his sites, that affected the number of individuals or species was 844 mg ∑PAH/kg dw for mollusks and <177 mg ∑PAH/kg for echinoderms, annelids and arthropods.

The adaptation of microbial communities in the gut of *Limnoria tripunctata* and in sediment was discussed in Neff (1979). Similar adaptations were described by Wade et al. (1989) in Gulf of Mexico hydrocarbon seep communities including numerous species of annelids, crustaceans, bivalves and fish. Tissue PAH concentrations indicated that these organisms were chronically exposed to high levels of PAH in an environment where they were able to survive and thrive. Their apparent ability to cope with elevated PAH concentrations may involve specially adapted and/or evolved enzyme systems.

Brooks (2000, 2004a, 2004b) and Goyette and Brooks (1998, 2002) reported the results of long-term environmental risk assessments of creosote structures located in freshwater and marine environments. None of these studies reported significant adverse effects in benthic communities resident within a few meters of the structures (see Chapter 10). Nor did the short term study of Wendt et al. (1994) document adverse biological effects in communities of invertebrates resident near creosote-treated structures.

5.1.5.7 Summary of the toxic effects associated with creosote derived PAH

The low molecular weight PAH such as naphthalene and acenaphthene produce acute toxic effects in marine animals because they are more soluble than the higher molecular weight compounds. Acute intoxication in the sensitive larval stages of marine invertebrates may occur at water column concentrations as low as 8 to 10 µg/L. For most species, the literature suggests that water column concentrations of greater that 20 µg/L are required for significant responses. The potential for accumulation of LPAH to toxic levels is small except when introduced in large quantities such as occurs in petroleum spills. Laboratory (including mesocosm) studies have demonstrated photo-enhanced toxicity associated with dissolved concentrations of anthracene as low as 1.2 to 4.0 µg/L.

Because of their decreased biological availability, sedimented PAH have a low potential to cause acute pathological responses at either the organismal or population levels in aquatic species. However, sediment levels of creosote <177µg/g have been shown to cause reductions in the abundance of sensitive taxa in field, but not in laboratory studies (Tagatz et al. 1983). Bacteria and eukaryotes

have demonstrated a remarkable ability to adapt to relatively high levels of background PAH. Chronic toxicity is more difficult to measure than acute toxicity. Chronic stress, resulting in reduced growth, but not in reduced reproductive success in mussels, was reported by Goyette and Brooks (1998) at 31 ng ∑PAH/L concentrations immediately adjacent to creosote-treated piling in Sooke Basin. Other reports of chronic stress associated with the use of creosote-treated wood products in open environments were not found.

In addition to direct physiological stress, there is a potential for the high molecular weight PAH like benzo[α] pyrene to form carcinogenic, mutagenic and teratogenic compounds during metabolism by crustaceans and vertebrates. While neplasia in mollusks is less common, Yevich and Barscz (1977) and Brooks (1991) have described carcinomas in soft shell clams (*Mya arenaria*) and mussels (*Mytilus edulis trossulus*) respectively. However, Brooks (1991) found significant correlations with the density of populations of mussels and the prevalence of hemic neoplasia, but he did not find positive correlations with anthropogenic activity. Neff (1979) summarized his section on neoplasia by noting that while carcinogenic PAH can produce cancer-like growths and cause teratogenesis and mutagenesis in some aquatic invertebrates and vertebrates, there are no reports of the induction of cancer by exposure of aquatic animals to environmentally realistic levels of carcinogenic PAH in the water, food, or sediments. More recent studies describe increases in the number of hepatic lesions and carcinomas with sediment ∑PAH burdens as low as 7 to 10 µg/g.

5.1.6 Recommended benchmarks for evaluating environmental risks associated with PAH

Numerous jurisdictions have established benchmarks for evaluating the human health and environmental risks associated with PAH in aquatic environments. Washington State Administrative Code Section 173-204 (WAC 173-204) defines marine sediment quality standards for individual PAH and for the the ∑LPAH and ∑HPAH. In addition, the EPA has proposed, but not adopted, freshwater criteria for acenaphthene, phenanthrene, and fluoranthene. This review did not reveal adopted freshwater sediment quality criteria for individual PAH compounds or their mixtures. However, there are numerous proposals based on the lowest levels at which adverse effects are observed in consolidated databases representing a broad spectrum of environments. The following discussion assumes that the toxicity of mixtures of PAH is additive. As previously discussed, it appears that toxicity associated with the mixture of PAH called creosote is less than additive—thereby adding to the conservativeness of the proposed benchmarks.

5.1.6.1 Benchmarks for assessing the risk of dissolved PAH

As previously noted, theoretical considerations and the available empirical evidence suggests that concentrations of dissolved PAH near creosote-treated wood structures are not expected to pose measurable environmental risk. However, there are conceivable applications, such as in canals, locks or impoundments lined with creosote-treated wood where dissolved PAH could reach concentrations harmful to aquatic fauna or flora. Based on the potential for photoenhanced toxicity of anthracene and its low concentration in creosote, Brooks (2005) recommended a dissolved PAH benchmark of 3.0 µg ∑PAH/L. That benchmark is considered conservative and is also recommended for use in the model presented in Chapter 9.

5.1.6.2 PAH sediment quality benchmarks

Waterloo (1999) lists freshwater and estuarine sediment quality benchmarks reviewed in Table 5.3. It should be emphasized that other than the Washington State Sediment Quality Criteria (WAC 173-204), none of the benchmarks given in Table 5.3 are enforceable sediment quality standards. They are simply guideposts for evaluating the effects of contaminants in sediments. Draft Rule Amendments to the Washington State SQS were distributed in June of 1999. The newly proposed standards include an increase in the ∑LPAH standard from 370 mg/kg organic carbon (OC) to 593 mg/kg OC and a decrease in the ∑HPAH standard from 960 mg/kg OC to 900 mg/kg OC. The sum of these two classes of PAH is proposed to increase from 1330 mg TPAH/kg OC to 1493 mg TPAH/kg OC. It should be emphasized that the Washington State SQS is for marine and estuarine environments—not for freshwater environments. Washington State (WDOE 2003) is in the process of developing freshwater sediment standards for PAH with a suggested benchmark of 6.6 mg LPAH/kg dw and 31 mg HPAH/kg dw.

Swartz (1999) provided a concise summary of the types of existing guidelines and attempted to consolidate various benchmarks into three tiers for which he claimed consensus support. These levels were, Threshold Effects Concentration (290 µg ∑PAH/g organic carbon), Median

Table 5.3 Summary of freshwater and estuarine benchmarks for polycyclic aromatic hydrocarbons. Data are from the UN Water Virtual Learning Centre website (http://bordeaux.uwaterloo.ca). All values are in µg PAH/g dry sediment.

Value (µg/g)	Type	Jurisdiction	Source
100.00 (∑PAH)	SLCA severe effects level	British Columbia	BCMOELP (1994)
110.00 (∑PAH)	SLCA severe effects level	Ontario	Persaud et al. (1992)
13.30 (∑PAH)	Recommended threshold concentration	United States	Ingersoll et al. (1996)
2.00 (∑PAH)	OMOE SQG – Lowest effect level	Ontario	Persaud et al. (1992)
22.00 (∑PAH)	AETA apparent effects threshold	British Columbia	BCMOELP (1994)
4.00 (∑PAH)	WEA effects range – Low	British Columbia	BCMOELP (1994)
3.93 (∑PAH – OC)	∑PAH threshold effects level		Swartz (1999)
21.14 (∑PAH – OC)	∑PAH mixture LC_{50}		Swartz (1999)
205.00 (∑PAH)	AET (estuarine)	Mississippi	Lytle and Lytle (1985)
4.00 (∑PAH)	Effects range – Low	NOAA	Jones et al. (1997)
44.80 (∑PAH)	Effects range – Median	NOAA	Jones et al. (1997)
13.30 ∑(LPAH + HPAH)	AET (estuarine and marine)	Washington	WAC 173-204[a]

a. Washington State Administrative Code Chapter 173-204.

Effects Concentration (1,800 µg ∑PAH/g OC) and the Extreme Effects Concentration (10,000 µg ∑PAH/g organic carbon). Swartz (1999) noted that the TEC (290 µg/g OC) is the easiest benchmark to interpret because adverse effects cannot be anticipated at values less than this. He notes that values exceeding the EEC are always associated with obvious adverse effects. Swartz (1999) councils that conclusions regarding the ecological effects of sediment contamination, which likely occur somewhere between the TEC and the MEC, should be based on site-specific analysis and weight of evidence derived from the three elements of the sediment quality triad. These *Consensus Guidelines* may resolve some of the current inconsistencies. He described a ∑PAH toxicity threshold that is consistent with the Effects Range Low (ER-L) of Long et al. (1995) and a ∑PAH mixture LC_{50} that is similar to the Effects Range Median (ER-M) described by the same authors.

Goyette and Brooks (1998, 2002) compared sediment concentrations of PAH with the ∑PAH Toxicity Threshold, the ∑PAH Mixture LC_{50} and the mean of these two values in predicting biological risk associated with creosote derived PAH in Sooke Basin. Table 5.4 summarizes the benchmarks derived from Swartz (1999). Goyette and Brooks (1998) examined the effects of creosote-treated piling in Sooke Basin. The extensive physicochemical and biological database, included infaunal community analysis and *in-situ* and laboratory bioassays using the amphipods *Rhepoxynius abronius* and *Eohaustorius estuarius*, liquid- and solid-phase Microtox™, echinoderm fertilization and *Mytilus edulis edulis* growth, survival and reproductive tests. Physicochemical analyses included a detailed description of sediment and water column concentrations of alkylated and parental polycyclic aromatic hydrocarbons. This database allowed for an examination of the efficacy of existing and proposed sediment quality benchmarks

Table 5.4 Summary of the ∑PAH toxicity threshold, ∑PAH mixture LC_{50} and the mean of these two values for 17 parental PAH. All values are µg-g^{-1} organic carbon. Values, excepting the mean, are from Swartz (1999).

	∑PAH		
PAH compound	Toxicity threshold	Mixture LC_{50}	Mean
Naphthalene	13	71	42.0
Acenaphthylene	3	15	9.0
Acenaphthene	4	23	13.5
Fluorene	17	90	48.5
Phenanthrene	29	155	92.0
Anthracene	21	114	67.5
Fluoranthene	69	371	220.0
Pyrene	90	481	285.5
Benz[α]anthracene	21	111	66.0
Chrysene	31	169	100.0
Benzo(b)fluoranthene	33	180	106.5
Benzo(k)fluoranthene	29	155	92.0
Benzo[α]pyrene	33	179	106.0
Low molecular weight PAH	87	468	277.5
High molecular weight PAH	306	1646	976.0
Total PAH	393	2114	1253.5

in predicting adverse biological response in Sooke Basin. The U.S. EPA draft sediment quality criteria for acenaphthene (130 µg/g organic carbon), phenanthrene 180 µg/g organic carbon) and fluoranthene (620 µg/g organic carbon) were found to be under-protective in that they failed to predict observed adverse biological effects when they were observed in three samples. False negative responses (adverse effects observed but not predicted by the benchmark) were not observed for any of the other benchmarks. Goyette and Brooks (1998) found 60 instances where individual PAH compounds exceeded the Threshold Effects Level (Jones et al. 1997) but where no toxicity was observed. These false positive indications associated with the TEL were observed for every PAH compound except naphthalene. The probable effects level (PEL) resulted in 21 false positive responses. The mean of the TEL and PEL ((TEL + PEL)/2) resulted in 30 false predictions of adverse effects where none were observed. The Washington State Sediment Quality Criteria (WAC 173-204) were most efficient in predicting adverse effects (12 false positive responses); these results suggested that the mean of the TEL and PEL, the PEL, or the Washington State apparent effects threshold (AET) based SQC (Table 5.5) were protective and efficient. In contrast, the TEL and the ER-L were not efficient and were considered overprotective in the Sooke Basin environment. The Washington State SQC have been adopted for assessing risk associated with creosote-treated wood in the model presented in Chapter 9.

5.1.6.3 Summary of the fate and effects of creosote derived PAH in aquatic environments

Polycyclic aromatic hydrocarbons are an integral part of earth's biosphere and there are numerous natural sources that distribute these compounds over broad areas at low concentrations. They have been a part of our environment for eons and plants and animals have developed specific enzyme systems to deal with exposure to these compounds. Numerous bacteria are capable of mineralizing PAH and except in exceptionally organic rich environments such as peat bogs and oils seeps, they do not accumulate to biologically stressful concentrations. However, anthropogenic activity, primarily associated with the extraction, transport and combustion of oil and coal can lead to locally significant concentrations in soil and sediments. Creosote is a byproduct of the steel and aluminum industry that has been used extensively as wood preservative since

Table 5.5 Summary of the sediment quality criteria (SQC) adopted by Washington State in WAC 173-204.

PAH compound	mg PAH/kg TOC	@1% TOC (mg PAH /kg dw)
Naphthalene	99	0.99
Acenaphthylene	6	0.06
Acenaphthene	16	0.16
Fluorene	23	0.23
Phenanthrene	100	1.00
Anthracene	220	2.20
2-methylnaphthalene	38	0.38
Fluoranthene	160	1.60
Pyrene	1000	10.00
Benz[α]anthracene	110	1.10
Chrysene	110	1.10
Benzo[*b&k*]fluoranthene	230	2.30
Benzo[α]pyrene	99	0.99
Ideno[1,2,3,-cd]pyrene	34	0.34
Dibenzo[*a*,*h*]anthracene	12	0.12
Benzo[*g,h,i*]perylene	31	0.31
Total low molecular weight PAH	370	3.70
Total high molecular weight PAH	960	9.60
Total PAH[a]	1330	13.30

a. Total PAH given here (∑PAH) is the sum of the LPAH and HPAH SQC adopted in WAC 173-204. However, this ∑PAH is not given in the WAC, and the value assumes additive toxicity of the two classes of PAH.

1865. Local, but significant environmental effects have been associated with historic wood treating facilities. However, these industrial processes are now highly regulated at the state and federal level to minimize pollution. It is important to discriminate between historical pollution at treated plants and current use of creosote-treated wood products, which are the focus of this chapter. The following statements summarize this background:

- The low molecular weight compounds in creosote oil (phenols, naphthalenes through fluorene) are preferentially lost during the treating process. Their concentrations in treated wood are approximately half of that found in raw creosote oil.
- The suite of PAH in creosote-treated wood is dominated by intermediate weight compounds having molecular weights from phenanthrene to benzo[*b* or *k*]fluoranthene. Creosote-treated wood contains relatively small amounts of the lighter or heavier compounds.
- Numerous microbes catabolize creosote derived PAH. Metabolism occurs in all environments under all studied physicochemical conditions. In general, the low molecular weight compounds degrade more quickly than the high molecular weight compounds; PAH degradation is faster with increasing oxygen availability; and PAH degrades more quickly in the presence of cometabolites.
- The proportion of low molecular weight compounds decreases from ca. 73% in raw oil to 60% in treated wood and to 22 to 25% in the suite of weathered sediment concentrations. Proportional increases in the intermediate weight compounds are observed at each step of the process. The proportion of high molecular weight compounds increases in weathered PAH, but remains low in sediments.
- The suite of PAH associated with creosote-treated wood is similar to that associated with highway dust. However, it contains lower concentrations of high molecular weight PAH than crankcase oil and lower concentrations of low molecular weight PAH than coal dust, diesel exhaust or atmospheric deposition.
- PAH are hydrophobic. All of the studies that have examined dissolved concentrations of PAH associated with creosote-treated wood in open aquatic environments have either failed to detect PAH or have recorded them at concentrations less than 31 ng/L. In Sooke Basin, the methodology of Swartz et al. (1995) indicated that the sum of toxic units associated with the maximum recorded concentration of dissolved PAH ($\sum TU = 0.0007$) was 250 times less than the benchmark ($\sum TU = 0.186$) recommended for protection of aquatic resources. The point is that there is no evidence in the literature suggesting that dissolved PAH associated with creosote-treated wood structures represents a threat to aquatic resources.
- PAH bioconcentrate in many aquatic organisms, but they are metabolized in all organisms studied—including mollusks and they do not biomagnify in food chains. The evidence indicates that mussels growing on creosote-treated wood structures in open aquatic environments outside other sources of PAH do not bioaccumulate PAH to tissue levels that pose a risk to the health of either human's or natural predators that might consume them.
- The photo-enhanced toxicities of individual PAH compounds have been observed at concentrations as low as 1.2 to 4.0 µg anthracene/L in the most sensitive taxa tested. Goyette and Brooks (1998) reported a maximum of 0.000708 µg anthracene/L immediately adjacent to a cluster of six creosote-treated piling in Sooke Basin. This value is 1,695 times less than the lowest concentrations at which photo-enhanced toxicity has been reported.
- Available evidence suggests that toxicity of the suite of PAH found in creosote oil is less than additive. For instance the LC_{50} for rainbow trout with respect to fluoranthene is >90.5 µg/L; whereas, the LC_{50} for whole creosote oil is 880 µg/L. In any case, the reported concentrations of dissolved PAH at which any form of toxicity has been demonstrated is hundreds to thousands of times greater than the concentration of PAH observed near creosote-treated structures in open aquatic environments and there is no evidence indicating that this route of exposure deserves additional consideration.
- Polycyclic aromatic hydrocarbons associated with creosote-treated wood can accumulate in sediments to biologically stressful concentrations. Sediment concentrations depend on the amount and age of the treated wood, hydrodynamic conditions that disperse PAH, environmental temperatures, sediment physicochemical characteristics including

percent total organic carbon, redox potential, and the availability of co-metabolites.

- Because PAH are hydrophobic, they bind to sedimented organic carbon, which reduces their bioavailability. Regulatory marine sediment quality criteria for individual PAH and suites of these compounds are available from Washington State, but they have not been widely developed. Numerous benchmarks and assessment methodologies are availability. Most of these define sediment PAH concentrations as a proportion of the sediment's total organic carbon. The literature suggests that chronic stress occurs at sediment PAH concentrations as low as 10 µg ∑PAH/g dry sediment. Reports of pre-neoplastic and neoplastic lesions associated with sediment PAH at 1.0 µg ∑PAH/g are significantly flawed because they failed to adequately assess the true exposure of demersal fish to PAH and because they failed to consider the suite of carcinogenic PCBs and metals that were also found in urban-industrial environments where the studies were undertaken. The preponderance of the literature suggests that long-term exposure of demersal fish species to ∑PAH concentrations in the range of 7 to 10 µg/g can lead to pre-neoplastic and neoplastic lesions.
- Goyette and Brooks (1998) evaluated the available benchmarks by comparing them with their extensive bioassay and macrobenthic community results. They found that the proposed U.S. EPA benchmarks for acenaphthene, phenanthrene and fluoranthene were underprotective because they failed to predict toxic effects in three samples. None of the other benchmarks failed to predict adverse effects when they occurred in any sample. The authors found that the threshold effects level (TEL) was inefficient in that it repeatedly (60 cases) predicted adverse biological effects when none were observed. The Washington State SQS, the PEL, and the mean of the TEL and PEL were protective and efficient in that their use resulted in only 12 to 30 false positive predictions (predicted toxicity that was not unobserved in any bioassay). The Washington State AET-based SQS values are similar to the Consensus Benchmarks developed by Swartz (1999) and are recommended as a benchmark for use in the risk assessment process described in Chapter 9.

5.2 Copper

Copper occurs in soft natural waters or in distilled or deionized water primarily as the divalent cupric ion. It is found as a free ion or complexed with humic acids, carbonate, or other inorganic and organic molecules in natural surface water—all of which decrease copper's toxicity. Copper is an essential element in the normal metabolism of both plants and animals. Therefore, a significant portion of the copper found in both fresh and marine systems may be taken up by biota.

5.2.1 Fate of copper in aquatic environments

The ultimate fate of much of the suspended copper is sedimentation. The model provided in Chapter 9 assumes that all of the copper leached from pressure-treated wood adsorbs to clay particles that are sedimented downcurrent from the structure. The copper is assumed to accumulate at its first point of sediment intercept. In environments subject to wave action and or current speeds fast enough to resuspend fine grained sediments and organic detritus, it is likely that adsorbed metals will be resuspended and dispersed further downstream. This makes the model overly conservative in all but depositional deep water environments. In addition, the model does not account for copper taken up by biota in the water column and dispersed by mobile animals or the copper adsorbed to POM that settles more slowly than clay particles. These pathways function to disperse copper further than predicted resulting in sediment concentrations that will be lower than predicted.

5.2.1.1 Cycling of copper from sediments as a function of redox potential

Lu and Chen (1977) examined the release of copper from sediments as a function of sediment grain size and oxygen availability. Sediment grain size was not a factor in the amount of copper released to the overlying water column. Three oxidizing conditions were examined (oxidizing, 5 to 8 mg DO/L; slightly oxidizing, ≤ 1.0 mg DO/L; and reducing, S(-II)T = 15 to 30 mg S=/L). Small amounts of bound copper were released from sediments into the overlying water (0.2 to 0.5 µg Cu/L) in reducing and slightly oxidizing environments. Copper releases in the oxidizing environment resulted in significantly higher interfacial seawater concentrations (3.2 µg/L). This effect was slightly more pronounced in the coarsest sediment tested (silty-sand).

These data imply higher copper releases from sediments in aerobic (biologically healthy) environments. There are two ways to look at these results. First, in sediments having high redox potentials, copper is more easily recycled into the water column and dispersed over greater distances. Eventually, much of the copper will be deposited in anaerobic sediments and incorporated into the lithosphere. Anaerobic environments typically support reduced infaunal and epifaunal communities (Brooks 2001). As a result, we might expect reduced environmental impacts from copper incorporated into these sediments. Alternatively, in enclosed bodies of water with coarse grained, aerobic, sediments, this study suggests that copper will cycle between sediments and interstitial and surficial waters where it is bioavailable. No data was provided on the copper species released from the sediments and therefore it is difficult to assess toxicity in this scenario. However, the biological effects associated with copper in this environmental would certainly be more significant than that associated with depauperate, anaerobic sediments. No attempt was made in the model presented in Chapter 9 to modify the risk assessment based on this discussion. These effects appear to be subtle and their exclusion should not flaw the risk assessment.

5.2.1.2 Complexing of copper in natural waters

Sciera et al. (2004) varied hardness between 10 and 40 mg $CaCO_3$/L; pH between 6.5 and 8.0 and Dissolved Organic Carbon (DOC) between 0 and 10 mg/L to demonstrate a range of 96-h LC_{50} values varying between 2.9 and 427.3 µg dissolved Cu/L in fathead minnows (*Pimephales promelas*). In general, bioassays have been conducted in ion-free water amended with Ca and Mg to achieve the specified hardness. Dissolved organic carbon has not been reported in most bioassays, but generally appeared to be low or zero in those bioassays using distilled, deionized or tap water. Sciera et al. (2004) used multifactor regression analysis to conclude that DOC was the dominant factor causing the wide spread in their LC_{50} determinations. Appendix G of U.S.EPA (2007) gives *Biotic Ligand Model* (HydroQual 2007) based instantaneous copper WQC at a range of pH, hardness and DOC values. At pH = 7.0 and 4 mg DOC/L, the WQC varies between 3.9 µg Cu/L at 40 mg $CaCO_3$/L and 12.4 µg Cu/L at 200 mg $CaCO_3$. However, when hardness is held constant at 159 mg $CaCO_3$/L in pH = 7.0 water, the WQC varies between 5.1 µg Cu/L at 2 mg DOC/L and 42.4 µg Cu/L at 16 mg DOC/L. The point being that DOC had greater effect on copper's toxicity in this these cases than did hardness.

5.2.2 Biological effects of copper in aquatic environments

The U.S. EPA (2007) provides significant additional information describing the toxicity of copper to aquatic organisms and readers are referred to that document for additional information.

5.2.2.1 Copper bioconcentration

Bioaccumulation is a general term describing the uptake of chemicals from water, sediments, food and other pathways. Bioconcentration (BCF) is defined as a process in which there is a net accumulation of a chemical directly from water across an organism's epithelium (gills, gut, skin, etc.) less the amount excreted. The National Academy of Sciences (1971) provides BCFs ranging from 100x for benthic algae to 30,000x for phytoplankton. Marine mollusks concentrate copper by a factor of 5,000 in muscle and soft tissues. Anderson (1977) reported metal bioconcentration factors in six species of freshwater clams from the Fox River in Illinois and Wisconsin. He found that soft tissues contained levels of copper equivalent to those found in sediments, which were significantly higher than water column concentrations. Anderson (1977) reported water column concentrations of copper at 0.001–0.006 µg/L, which seems low by a factor of 100 to 1000 for natural waters. Assuming that these reported water column concentrations are in error by a factor of 1,000, a comparison of the mean soft tissue copper burden (12.24 µg/g dry tissue weight) with the mean water column copper concentration 3.5 µg Cu/L) implies a BCF of 3,497, which is consistent with the NAS (1971) BCF for bivalve mollusks. Hendriks (1995) observed that dry weight corrected concentrations of copper in freshwater plants and invertebrates from the Rhine Delta were 0.2 to 0.3 times the concentrations observed in suspended sediments—suggesting that copper adsorbed to suspended sediments is not readily bioconcentrated.

Marquenie and Simmers (1987) examined metal and polycyclic aromatic hydrocarbon concentrations in sediments and earthworms (*Eisenia foetida*) at an artificial wetland site created at a confined dredged material disposal area that became a prolific wildlife habitat. At the six sites reported, they found an average copper concentration of 192.5 ± 107.6 mg Cu/kg dw in soils. At the end of ca. 49 d, *Eisenia foetida* contained an average of 36.3 ± 14.9 µg Cu/g (ash-free tissue weight) suggesting that much of the sedimented copper was not biologically available (BCF = 0.19). Control earthworms, collected outside the dredge disposal site, where soil copper concentrations

averaged 16.5 mg/kg, contained an average of 10.1 µg Cu/g giving a BCF of 0.61 (three times higher). It is possible that 49 d was an insufficient period of time for annelid tissue to come into equilibrium with the high environmental concentrations of copper. Alternately, it is also possible that *Eisenia foetida* is able to regulate copper uptake.

Rai et al. (1995) examined metal uptake from pond water amended with 1.338 µM (84 mg/L) copper in eight species of submerged macrophytes. No acute effects were observed—although several of the plant species did not increase their biomass. At the end of 15 d, the plants had removed significant quantities of metal from the pond water and the bioconcentration factors given in Table 5.6 calculated. Copper bioconcentration factors varied considerably by species (211 in *H. aristata* to 53,333 in *C. demersum*). This study demonstrated high, but variable copper BCFs in some plant species and demonstrated the potential for plants to remove copper from stormwater in retention ponds or biofiltration swales. However, it is difficult to extrapolate from this study to natural environments where copper concentrations would likely be <5 to 15 µg/L rather than 84,000 µg/L.

5.2.2.2 Copper biomagnification

Biomagnification is defined as the processes of bioconcentration and bioaccumulation leading to increased concentrations of contaminants as they pass upward through two or more trophic levels. Little information was reviewed on the biomagnification of copper by aquatic organisms. Van Eeden and Schoonbee (1993) reported copper concentrations in sediments, fennel-leaved pondweed and various organs of the red-knobbed coot associated with a metal contaminated wetland in South Africa. They found that the pondweed contained less than half the copper concentration found in sediments. Copper concentrations in the various organs of the coot were similar to those in the pondweed—except that very little copper was transferred to eggs (shell or contents). For the purposes of this paper, it will be assumed that copper accumulation in aquatic organisms is primarily a function of concentration in the ambient water. While many organisms may bioconcentrate copper, the available information suggests that copper is not biomagnified through food webs. The two processes (bioconcentration and biomagnification) are not necessarily directly related. Many materials are bioconcentrated, particularly by bivalves. However, many substances are not biomagnified because they are rapidly excreted and/or organic compounds are metabolized.

5.2.2.3 Copper toxicity in freshwater environments

Copper is an essential element for most living organisms. It is added at a concentration of 2.5 µg/L in Guillard's Medium F/2 to sea water for the optimum culture of marine algae (Strathman 1987). At concentrations slightly above those required as a micronutrient, copper can be toxic, especially to the larval stages of marine invertebrates. A single copper fitting in a seawater system may cause the death of invertebrate embryos. The EPA (1986, 2007) Ambient Water Quality Criteria reports that copper toxicity in aquatic environments is related to the concentration of cupric (Cu^{2+}) ions and perhaps copper hydroxides ($CuOH^n$). The cupric ion is highly reactive and forms various copper complexes and precipitates which are significantly less toxic than the cupric ion (Knezovich et al. 1981). Harrison et al. (1987) reported that copper discharged

Table 5.6 Metal concentration factors (dimensionless) for submerged aquatic plants (from Rai et al. 1995).

	Metal					
Plant	Cu	Cr	Fe	Mn	Cd	Pb
Hydrodictyon reticulatum	2,481	11,394	37,666	8,712	6,250	5,000
Spirodela polyrrhiza	36,500	7,920	3,878	3,107	5,750	2,521
Chara carallina	1,103	2,081	3,029	2,030	2,125	2,133
Ceratophyllum demersum	53,333	15,332	37,809	21,600	3,333	8,064
Vallisneria spiralis	2,009	1,993	1,344	333	2,375	1,777
Bacopa moonieri	18,750	2,016	2,041	2,487	29,000	366
Alternanthera sessilis	1,051	722	1,156	6,395	23,000	555
Hygrorrhiza aristata	211	652	1,138	1,955	4,600	7,174

from the San Onofre power plant cooling system was found mostly in bound forms under normal operating conditions. Their study found sufficient organic ligands available in ambient seawater to complex most of the copper, and they expected little or no impact from the discharges. Likewise, Nuria et al. (1995) and Kerrison et al. (1988) have observed that copper in freshwater lakes is generally associated with particulate organic and inorganic material rather than with dissolved organic matter (DOM). These authors concluded that natural water significantly reduces copper toxicity to aquatic organisms when compared with laboratory systems manipulated using synthetic chelators like EDTA. These reports are important for interpreting the results of laboratory bioassays using water that is deficient in the numerous organic and inorganic complexing agents found in most surface waters.

Sundra (1987) has proposed a basic mechanism explaining the observed relationship between free ion activities and the bioavailability of metals such as copper. He observed that the complexed species of copper are charged or polar and cannot pass directly across the lipid bilayer of the cell membrane. Thus, transport of copper across the membrane would require that it interact with specific metal transport proteins in the membrane. Because the free ion activity is a measure of the potential reactivity of a metal, it reflects the ability of the metal to interact with these transport proteins. The many chemical forms of copper in aquatic environments are maintained in a dynamic state of equilibrium that depends on salinity, temperature, pH, alkalinity, dissolved oxygen, sediment characteristics and the presence of other inorganic and organic ligands.

Clements et al. (1988) spiked freshwater mesocosms with 12 to 20 µg Cu/L and 15 to 27 µg Zn/L. They found significantly reduced numbers of taxa, numbers of individuals and abundance of most dominant taxa within 4 d. After 10 d, control streams were dominated by Ephemeroptera and tanytarsid chironomids, whereas treated streams were dominated by Hydropsychidae and Orthocladiini. Responses of benthic communities to metals observed at the Clinch River (Russel County, Virginia), a system impacted by copper and zinc were similar to those in the experimental streams. Copper concentrations in the Clinch River varied from not detectable at upstream controls to 105 µg/L at the point of discharge. Ephemeroptera and Tanytarsini, which comprised 46 to 48% of the macroinvertebrate community at upstream reference stations, were significantly reduced at all effluent sites. In this natural system, impacted stations were also dominated by Hydropsychidae and Orthocladiini. Interestingly, significant decreases in the number of all taxa and the abundance of individual species was observed at station (6), where 9 ± 7 (one standard deviation) µg/L Cu was observed. They found that Tricoptera and Orthoclad chironomids were tolerant of high copper concentrations. The hardness at these Clinch River (Virginia) stations averaged 169 ppm ($CaCO_3$) and the alkalinity averaged 148 µg/L. At this hardness, the EPA chronic criterion is 17.8 µg/L. However, it should be noted that this station was directly downstream from the discharge stations that had much higher concentrations (47 to 105 µg/L). Copper concentrations this high would likely have significant effects on the drift community. This is seen in a follow-up study (Clements et al. 1992) in which data from 1986 through 1989 were examined upstream and downstream from the power plant following a decrease in the copper content of the plant's effluent from 480 µg/L in 1987 to 260 µg/L in 1989. Copper concentrations were reduced at downstream Station (8) from 127 µg/L in 1987 to 52.2 µg/L in 1989. The number of taxa increased from ca. ten in 1987 to 20 in 1989. Only small decreases in both the number of taxa and the number of individuals per sample were observed in 1989 suggesting only minor effects at the observed copper concentration of 52.2 µg/L.

Gower et al. (1994) examined the relationship between invertebrate communities and a variety of metals in southwest England. Their work suggested that copper followed by aluminum, zinc, and cadmium, were the metals most responsible for influencing the observed changes in the invertebrate community. The results of Gower et al. (1994) are summarized in Table 5.7. The columns describe the response of various taxa to exceedances of the U.S. EPA chronic WQC at the reported hardness. Community information is displayed by sample for each taxonomic group.

These data are presented in some detail because they clearly demonstrate the insensitivity of at least one flatworm species (Tricladida) some caddis flies (Trichoptera) and chironomids, particularly Orthocladiinae to high water column concentrations of copper (245 x EPA standard). Oligochaetes, caseless caddis flies and stone flies (Plecoptera) are relatively insensitive at copper concentrations up to 32 times the EPA standard but the population was essentially extirpated at 245 times the EPA standard. It is certainly possible that caseless caddis flies and stone flies represent the drift community in this study and the period of their exposure to elevated copper concentrations

is unknown. This observation is supported by the reduced numbers of resident (cased) caddis flies observed in areas where the copper concentrations exceeded the EPA chronic copper standard by a factor of 5.3.

Interestingly, the Order Ephemeroptera, frequently described as very susceptible to copper intoxication, represented nearly 40% of the macroinvertebrate community at 5.3 x the EPA standard and at least one species was able to tolerate 31.6 x the EPA standard. In addition to describing general trends in copper susceptibility, these data suggest that some species in the sensitive orders Ephemeroptera, Plecoptera and Trichoptera are able to tolerate high concentrations of copper—suggesting that increasing information is provided by identification of infauna to the level of genus or species. On the other hand, it should be noted that total species richness (number of species) declines monotonically and is perhaps the best indicator of increasing copper toxicity in this study. While the numbers of Ephemeroptera, Plecoptera and Trichoptera do not follow this monotonically decreasing trend, if we consider these Orders in the aggregate, we find that species richness is inversely correlated with copper concentrations.

Kiffney and Clements (1994) examined the effects of heavy metals on a macroinvertebrate assemblage from a Rocky Mountain stream in experimental microcosms and found significant reductions in a number of taxa at their "1x" treatment of 12 µg Cu/L. The authors stated that this value was approximately equal to the U.S. EPA freshwater chronic copper standard at the measured hardness of 38.3 mg/kg ($CaCO_3$). However, at that hardness, the EPA acute criteria is approximately half the tested concentration (6.9 µg/L versus 12 µg/L) and the chronic EPA criteria is only 38% of the test concentration. The results of this study followed that of others reported herein. Significant reductions were observed in the Order Ephemeroptera, particularly in the family Heptageniidae. A large variation was observed in chironomid response to copper with significant reductions in the Tanytarsini and Tanypodinae and a small reduction in the Orthocladiinae and Chironomini.

Rutherford and Mellow (1994) examined the effects of low pH and high dissolved metal (particularly copper) content on the fish and macroinvertebrates in beaver ponds located on an abandoned ore roast yard near Sudbury, Ontario, Canada. Table 5.8 summarizes the physico-chemical properties of the water at three of the sample stations. Hardness values were not provided in this study. Dissolved copper at all of the tested stations exceeded background by factors of six at Station 1 to 200 at Station 3. Other metals were elevated, but not to the same degree as copper. It appears reasonable to suggest that most of the effects seen in the macrobenthic community were associated with copper.

Table 5.9 provides a summary of the numbers of individuals within the most copper sensitive and copper tolerant species observed in this study and suggests that the

Table 5.7 Mean number of total macroinvertebrates per sample (M), mean percentage contribution of selected major taxa to the total macroinvertebrate fauna (P), and number of species (S) observed by Gower et al. (1994) as a function of water column concentrations of copper expressed as proportional increases in the U.S. EPA freshwater copper criteria at the observed level of hardness.

	Ratio of dissolved copper to the U.S. EPA chronic freshwater criteria							
	2.0x		5.3x		31.6x		244.7x	
Taxonomic group	M	S	M	S	M	S	M	S
Macroinvertebrates	4598	39	989	21.3	2219	12.2	2378	9.2
	P	S	P	S	P	S	P	S
Tricladida (flatworm)	7.7	1.3	3.4	1.0	15.0	1.1	30.2	0.8
Oligochaeta	16.2	4.8	6.8	2.4	20.9	1.3	0.7	0.3
Ephemeroptera	17.4	3.1	38.1	2.0	16.7	0.6	0.0	0.0
Plecoptera	19.3	4.9	8.5	2.3	7.2	1.3	1.1	0.3
Coleoptera	4.0	2.4	0.6	0.8	0.7	0.4	0.6	0.6
Trichoptera (cased)	4.8	3.0	12.4	1.8	0.1	0.2	0.1	0.1
Trichoptera (caseless)	4.8	3.4	14.7	3.0	5.4	1.8	4.8	1.3
Total EPT[a]	46.3	14.4	73.7	9.1	29.4	3.9	6.0	1.7
Chironomidae	16.9	8.0	11.3	4.4	31.1	3.8	60.3	3.8
Orthocladiinae	9.1	4.7	4.8	1.8	18.4	2.1	54.5	2.4

a. EPT refers to the aggregate numbers of insects in the orders Ephemeroptera, Plecoptera, and Trichoptera.

genus *Chironomus* was very tolerant to even extraordinary concentrations of copper (600 µg/L). The Tanypodinae were tolerant of moderate (45 µg/L) copper concentrations as were several species of dragonflies (Order Odonata). In contrast, all of the mollusks in this study were intolerant of copper at 45 µg/L but survived concentrations six times that of the background of about 3 µg/L. Macroinvertebrates were sampled with a D-frame net (1 mm mesh) in near-shore vegetation, the detritus, in muck at the bottom of the pools, and in open water. The net was maneuvered for about 15 s in each of these habitats. The reported intolerance of mollusks, arthropods and some oligochaetes (Naididae) to copper is noteworthy. These data, like that of many other studies reviewed herein, suggest that copper tolerance varies widely among genera —even within the same family. These data also suggest that diverse (albeit suppressed) communities of macroinvertebrates can tolerate dissolved copper concentrations of at least 45 µg/L.

In summary, these studies demonstrate trends in the relative sensitivity of freshwater macroinvertebrates to copper intoxication. However, Gower et al. (1994) also point out that at least some EPT species can tolerate high dissolved copper concentrations. Lastly, these data suggest that species richness for all fauna, or for the aggregated Orders EPT, is better correlated with the degree of copper intoxication than is an analysis at lower levels of taxonomic structure. Ammann et al. (1997) provided an excellent review of the idea of *Taxonomic Sufficiency* for measures of impact in aquatic systems. They concluded that in at least one series of studies, identification and evaluation of infauna to the level of phylum was sufficient to document effects.

5.2.2.4 Effects of copper on salmon

Sorensen (1991) reported acute intoxication (96-h LC_{50}) in adult salmonids at dissolved copper concentrations between 60 and 680 µg Cu/L. Chapman (1978) reported effects levels for steelhead (*Oncorhynchus mykiss*) and chinook salmon (*O. tshawytscha*) alevins, swim-up fry, parr and smolts. The 200-h LC_{10} describes the concentration of copper that kills 10% of the fish in 200 h (8.3 d). He found that juvenile steelhead were more sensitive to copper than were juvenile chinook. Steelhead parr were most sensitive with a 200-h LC_{10} of 8 µg/L. Steelhead alevins (19 µg/L) and swim-up fry (9 µg/L) were slightly more robust. Swim-up fry of chinook salmon were the most sensitive life stage

Table 5.8 Water chemistry at sample stations 1 (upstream), 3 (in roast pits), and 4 (immediately downstream from roast pits) in the study of Rutherford and Mellow (1994).

Station	pH	Temp (°C)	Dissolved oxygen	Copper (µg/L)
1	6.7	22.2	8.0	18
3	3.7	22.2	8.1	600
4	6.2	23.3	7.6	45

Table 5.9 Selected macroinvertebrate taxa with significant sensitivity or tolerance to high copper concentrations at sample stations 1 (upstream), 3 (in roast pits), and 4 (immediately downstream from roast pits) in the study of Rutherford and Mellow (1994). Taxa exhibiting moderate to strong Cu tolerance are bolded; dissolved Cu concentrations (µg/L) are in parentheses after each station number.

	Sample station		
Taxon	1 (18 µg/L)	3 (600 µg/L)	4 (45 µg/L)
Total number species	40	1	25
Total number macroinvertebrates	228	105	145
Chironomidae			
Tanypodinae	10	0	14
Chironomus	13	105	23
Cladopelma	12	0	2
Microtendipes	3	0	0
Polypedilum	9	0	8
Diptera	28	0	2
Chaoborus			
Himiptera			
Corisella	7	0	3
Odonata			
Corduliidae	13	0	4
Leucorrhinia	1	0	23
Coenagrionidae	0	0	44
Trichoptera			
Oecetis	0	0	2
Banksiola	0	0	3
Amphipoda			
Hyallela azteca	3	0	0
Naididae	37	0	0
Mollusca			
Physa	8	0	0
Helisoma	29	0	0
Sphaerium	15	0	1
Hirudinea	5	0	0

of this species with a 200-h LC_{10} of 14 µg Cu/L. Finlayson and Wilson (1989) developed a chronic copper benchmark that they considered appropriate to protect all life stages of chinook salmon in the Sacramento River, California. At a hardness of 64 mg $CaCO_3$/L, their algorithm indicates adequate protection at 8.59 µg Cu/L. Acute toxicity values (96-h LC_{50} in µg Cu/L) values for two salmonids are provided in Table 5.10 together with the hardness values at which the tests were conducted.

Chronic effects of copper on salmon

Olfactory perceptions are important to fish for feeding, predator avoidance, schooling, migration, recognition of natal spawning grounds and mating (recognition of the same species). Impairment of a fish's olfactory response has potential adverse effects on individuals and populations of fish. For instance, Wisby and Hasler (1954) captured coho salmon (*Oncorhynchus kisutch*) returning to Issaquah Creek and the East Fork of Issaquah Creek in Washington State. They occluded the nares (olfactory organs) from a portion of the fish with cotton and then reintroduced them back into the environment below the confluence of the two streams. Eighty-nine percent of fish with intact nares (N = 73) chose the correct branch whereas only 60% the fish with occluded nares (N = 70) chose the correct branch. Lorz and McPherson (1976) confirmed this loss of migratory fidelity by exposing 18-mo-old coho salmon (*Oncorhynchus kisutch*) to varying copper concentrations. Exposure to 5 µg Cu/L for 165 d resulted in a 30% reduction in downstream migration. Little (1983) reported detection limits of numerous organic compounds by a variety of fishes that varied between 10^{-5} and 2.9×10^{-20} moles and that low concentrations of single amino acids are able to evoke spontaneous (unconditioned) and conditioned responses. Conditioned responses to shock reinforced introductions of single amino acids could be achieved in four or five trials. These conditioned responses have been shown to last for longer than 3 mo. This is the basis for NOAA fisheries attempts at the Manchester Research Station to condition endangered salmon stocks being maintained in their hatchery-nursery system by introducing macerated salmon in concert with water previously exposed to predators (squawfish, blue herons, etc.).

Giattina et al. (1982) reported copper avoidance in rainbow trout (*Oncorhynchus mykis*) at 4.4 to 6.4 µg Cu/L in soft water (28 mg $CaCO_3$/L). However, these same trout were attracted to 334 to 386 µg Cu/L. Drummond et al. (1973) observed reduced feeding of brook trout (*Salvelinus fontinalis*) lasting only 24 h following long-term exposure to 6 µg Cu/L. Unacclimated chinook salmon (*Oncorhynchus tshawythscha*) have been shown to significantly avoid copper concentrations as low as 0.8 µg Cu/L in tap water having hardness equivalent to 25.3 mg $CaCO_3$/L at pH = 7.5 and T = 10.2°C. (Hansen et al. 1999a). The avoidance reaction was impaired at copper concentrations ≥ 44 µg Cu/L. This last finding is important because it demonstrated that chinook salmon did not avoid lethal concentrations of copper. The authors noted that chinook salmon acclimated to water containing 2.0 µg Cu/L, did not avoid higher copper concentrations. This suggests that chinook salmon will not elicit an avoidance reaction in natural environments where the authors acknowledge that background concentrations are commonly as high as 4.0 µg Cu/L. The reasons for the lack of avoidance of copper at high concentrations was further explored by Hansen et al. (1999b) who described histological evidence demonstrating a significantly reduced number of olfactory sensors in chinook salmon exposed to ≥ 50 µg Cu/L and that the numbers of *small-dendrite* receptors was significantly decreased after a 4-h exposure to 25 µg Cu/L. The authors noted that olfactory rosette receptors have been reported to regenerate following 8 to 42 d recuperation in clean water following short-term exposure to high copper concentrations, but that the demonstrated olfactory impairment may affect important responses including homing response of anadromous salmonids, predator avoidance and feeding.

Hansen et al. (1999c) demonstrated un-acclimated rainbow and brown trout avoidance of very low concentrations of mixtures of metals (nominally 1.2 µg Cu/L, + 0.11 µg Cd/L, 0.32 µg Hg/L and 5.0 µg Zn/L). The avoidance could have resulted from additive or synergistic interaction of the metals, or it could be that the fish were responding to a single metal in the mixture. The authors did not adequately discuss the interactive effects of the mixture of

Table 5.10 Benchmarks describing acute copper toxicity in salmonids.

Species	96-h LC_{50} (µg Cu/L)	Reference
Brown trout (*Salmo trutta*)	61.5 @157.8 mg $CaCO_3$/L	Baldigo and Baudanza (2001)
Chinook salmon (*Oncorhynchus tshawytscha*)	19.0 @ 24 mg $CaCO_3$/L	Chapman (1978)

metals. Sprague (1964) observed un-acclimated Atlantic salmon avoidance to 2.3 µg Cu/L or 53.4 µg Zn/L. Important to the assessment of ACZA, which loses copper and zinc at essentially the same rate, Sprague (1964) demonstrated that unacclimated Atlantic salmon avoided Cu-Zn mixtures containing as little as 0.42 µg Cu/L and 6.1 µg Zn/L suggesting that the metals acted synergistically. These authors also found that rainbow trout acclimated to the simulated ambient metal mixture (12 µg Cu/L; 1.1 µg Cd/L; 3.2 µg Pb/L; and 50 µg Zn/L) preferred clean water when given that option and avoided higher concentrations.

Baldigo and Baudanza (2001) assessed avoidance by brown trout to water pumped from a reservoir treated with copper sulfate to control algae. This highly replicated study is one of the few reviewed studies that quantified many of water quality endpoints important for determining the toxicity of copper. The water had pH = 7.0; TOC = 134.6 µM/L; chloride = 135 µM/L; Mg = 45.7 µM/L; Ca = 112.1 µM/L and K = 13.9 µM/L. Measured copper concentrations of 9.7 to 183.3 µg Cu/L were generally higher than the designed concentrations of 0 to 152 µg Cu/L. Their results indicated that the estimated threshold for avoidance of dissolved copper was ~55 µg/L, which is only slightly less than the 61.5 µg Cu/L 96-h LC_{50} determined by these authors in the same water. The lack of response may be due to elevated copper concentration (9.7 µg Cu/L) in the rearing and control water, which was pumped from an aqueduct leaving the West-of-Hudson Catskill reservoir system. Marr et al. (1995) showed that brown trout acclimated to elevated mixtures of metals (including copper) suffered fewer mortalities than un-acclimated populations. This study is important to this review because it documents the potential for increased tolerance to metals associated with acclimation and the mediating effect that dissolved organic carbon and hardness have on chronic and acute copper toxicity.

McKim and Benoit (1971) found that 9.5 µg Cu^{2+}/L was a safe concentration for brook trout (*Salvelinus fontinalis*) in Lake Superior. Drummond et al. (1973) assessed the effects of short-term exposure to 0.7 to 24 µg Cu/L on this same species in water having total hardness of 44 to 46 mg/L and pH of 7.54 to 7.75. They found significant increases in fish activity at all increased copper concentrations above zero. Mean coughing rates were significantly ($p < 0.01$) increased at ≥ 9.0 µg Cu/L but not at 6.0 µg/L. Most importantly, they found that the addition of copper decreased feeding activity. Fish acclimated quickly at 6 µg Cu/L and 100% resumed normal feeding behavior after 24 h of exposure. Brook trout acclimated to higher concentrations of copper more slowly. Eighty-seven percent of the trout resumed feeding after 6 d exposure to 12 µg Cu/L and 100% were feeding after 14 d exposure. However, their feeding behavior was described as sluggish.

Hara et al. (1976) demonstrated reduced recognition of water containing 10^{-5} M L-serine (an amino acid associated with fish skin) when exposed to 8 µg Cu/L. The study is somewhat confusing because the authors stated that the rearing and trough water contained an average of 20 µg Cu/L. Rehnberg and Schreck (1986) conducted similar experiments with coho salmon (*Oncorhynchus kisutch*) in water having hardness equivalent to 30.5 mg $CaCO_3$/L and pH = 6.72. They observed lack of avoidance of L-serine introduced at 10^{-8} M at all tested copper concentrations. The lowest copper concentration tested was 6.35 mg Cu/L (10^{-8} M Cu). Saucier et al. (1991) exposed 14-d post-fertilization and post-hatching rainbow trout to water having a hardness of 61.8 to 64 mg $CaCO_3$/L and 22 µg Cu^{+2}/L for 41 and 37 wk, respectively, to assess the effects of long-term chronic exposure to high copper concentrations. The authors then gave the fish a choice of swimming upstream into their own rearing water or into water containing a heterospecific pheromone. Control fish significantly preferred their own rearing water that contained <5 µg Cu/L, whereas the copper exposed fish did not show a preference. Olfactory responses were not significantly different between the control and either treatment group different following 10 wk of post test conditioning of all cohorts in ambient water not spiked with copper. This long-term 22 µg Cu^{+2} /L challenge of olfactory responses suggests significant short term effects—followed by recovery after 2 to 10 wk in water containing 5 µg Cu/L. The observation of these fishes ability to discriminate the subtle pheromones in their own rearing water in the continuous presence of 5 µg Cu/L suggests that concentrations this low did not have an adverse affect on the test results. Chronic response of salmonids to dissolved copper are summarized in Table 5.11. Bjerselius et al. (1993) demonstrated that it is the cupric ion that has the greatest affect on olfactory response in salmonids. The proportion of copper in the free cupric ion state is dependent on a number of mediating factors including water hardness, alkalinity, pH, dissolved organic carbon, etc. Most State WQC are currently based on the historic U.S. EPA hardness-based recommendation. It is not possible to determine the BLM recommendation without additional data—particularly the concentration

of DOC, which unfortunately has not frequently been reported.

Hecht et al. (2007) reviewed the literature describing neurophysiological effects of copper on the olfactory response in salmon with emphasis on research by Sandahl et al. (2007) describing the results of electro-olfactograms (EOGs), in which the epithelium covering the olfactory rosette is excised to expose the organ. The rosette is then directly perfused with copper spiked water and sensory output (mV) in response to a fright/flight stimulus create using L-serine, measured using an implanted electrode. Data from Sandahl et al. (2007), describing the olfactory response of copper exposed and unexposed salmon was used in a U.S. EPA (1995) benchmark dose analysis (BMC) to estimate concentrations of dissolved copper that could be expected to affect juvenile salmon olfaction—"and, by extension, alarm response behavior involved in predator avoidance." The mean rosette output from fish that were not exposed to spiked copper was 1.2 mV. This mean was reduced by 10% (1.08 mV at 0.18 µg Cu/L and the lower 95% confidence limit for control response (0.85 mV) recommended as a benchmark concentration (0.79 µg dCu/L (dCu is dissolved copper). Sandahl et al. (2007) used dechlorinated municipal water at pH 6.6 with 120 mg/L hardness. The authors did not report DOC or other water quality parameters necessary to determine copper at the biotic ligand (rosette) using the BLM. Hecht et al. (2007) estimated BLM inputs at pH 6.6 and 120 mg $CaCO_3$/L to estimate a corresponding BLM Criterion Maximum Concentration (CMC) of 0.63 µg Cu/L, which is lower than the BMC criterion of 0.79 µg/L. Within a pH range of 6.5–7.1 and a DOC range of 0.3 to 1.5 mg/L, the BLM CMC range of 0.34 to 3.2 µg/L overlapped the BMC range of 0.18 to 2.1 µg Cu/L. Hecht et al. (2007) concluded that hardness and alkalinity were unlikely to protect juvenile salmonid olfaction in Pacific Northwest Rivers. However, USGS data suggested that 29% of West Coast Basins contained sufficient DOC to limit olfactory impairment to 50% or less at 20 µg Cu/L, but that only 6% of the river samples contained sufficient DOC (i.e., >6 mg/L) to fully protect juvenile salmon from 20 µg Cu/L. Hecht et al. (2007) did not discuss several issues that should be considered when interpreting the results of Sandahl et al. (2007).

The physiological effects of removing the nare's epithelium to expose the rosette with direct perfusion of copper spiked water was not discussed by either Sandahl et al. (2007) or by Hecht et al. (2007). Exposure of the rosette creates an unrealistic scenario that is akin to studies of PAH discussed earlier in which naked cells are exposed to PAH dissolved in DMSO. What is the response of the nare, with epithelium intact, when exposed to sudden increases in dissolved copper and how does this response correspond to the response of the exposed rossette? Does the nare partially close, like wrinkling your nose, to partially isolate the rosette? Do biotic ligands on the nare's epithelium compete with the rosette as adsorption sites for cupric ions?

The authors assumed that there is a negative correlation between electrical output from the rosette and stimulation of a fright-flight response with no threshold value. Other literature, described above, indicates that the response is inhibited at copper concentrations exceeding 4 to 5 µg Cu^{2+}/L. However, no quantitative cause and effect relationship has been established between electrical output from the rosette and inhibition of the response. Until these cause and effect relationships are established or until the studies are repeated in more physiologically and environmentally realistic conditions, the observations of Sandahl et al. (2007), while informative for generating hypotheses, must be considered effects per se.

Table 5.11 Thresholds above which salmon have been shown to avoid dissolved copper.

Species	Avoidance threshold (µg Cu/L)	Hardness (mg $CaCO_3$/L)	Reference
Rainbow trout (*Oncorhynchus mykiss*)	0.1	89.5	Folmar (1976)
Rainbow trout (*Oncorhynchus mykiss*)	6.4	23.0–27.0	Giattina et al. (1982)
Rainbow trout (*Oncorhynchus mykiss*)	70.0	112.4	Black and Birge (1980)
Rainbow trout (*Oncorhynchus mykiss*)	8.0	90.0	Hara et al. (1976)
Rainbow trout (*Oncorhynchus mykiss*)	<22 µg Cu^{2+}/L	61.8–64.0	Saucier et al. (1991)
Atlantic salmon (*Salmo salar*)	2.4	20.0	Sprague et al. (1964)
Brown trout (*Salmo trutta*)	55.0	157.8	Baldigo and Baudanza (2001)
Coho salmon (*Oncorhynchus kisutch*)	<6.4	30.5	Rehnberg and Schreck (1986)

As noted by Hecht et al. (2007), the reviewed studies did not document DOC or other mitigating constituents of natural surface waters. At this point in time, the bioavailable copper can only be assessed using the BLM, whose copper predictions depend significantly on DOC.

From an operative point of view, these issues are not as confounding as they could be. The conclusions of Hecht et al. (2007) suggest that the CMC criteria provided by the BLM are appropriate for protection of juvenile salmonid olfaction and that is the benchmark that is recommended for use in waters inhabited by listed salmon when using the model provided in Chapter 9.

Summary for copper effects on salmonids

Nearly all natural bodies of water contain copper (see Chapter 8), which constitutes about 0.006% of earth's crust and is typically found at concentrations of 2 to 100 µg Cu/g dw in sediment and 1.4 to 10.0 µg/L in water. While not discussed in this chapter, some famous salmon producing rivers, such as Alaska's Copper River, carry dissolved copper concentrations in the range of 2 to 23 µg/L. The mass of dissolved copper has been recognized as an inappropriate endpoint for assessing biological effects because its toxicity is mediated by a number of organic and inorganic constituents found in surface water. Protecting the integrity of salmon olfactions appears to represent the most sensitive issue requiring management of copper in freshwater environments. Acute toxicity has traditionally been measured by 96-h LC_{50} bioassays in laboratory water with only hardness recognized as an important parameter. The results of bioassays using juvenile salmonids give 96-h LC_{50}s between 19 and 61.5 µg dissolved Cu/L depending on hardness and perhaps on species of salmon. The review provided above indicates that the electrical output of nude juvenile salmon rosettes is deminished by short term increases of 0.79 µg Cu/L. However, none of the studies reviewed defined affecting concentrations in terms of DOC or the other organic and inorganic constituents that reduce the bioavailability of copper to biotic ligands. However, this review and that of Hecht et al. (2007) suggests that the CMC values produced by the BLM are protective of all aspects of salmon physiology.

5.2.2.5 Copper water quality criteria

Most state freshwater dissolved copper WQC (i.e., that passing a 0.45 µm filter) are based on the U.S. EPA recommendations using hardness. Table 5.12 includes definitions for acute and chronic criteria. Typical freshwater acute values range from 4.6 µg Cu/L at 25 mg $CaCO_3$/L to 18.5 at 100 mg $CaCO_3$/L and chronic values range between 3.5 and 11.4 µg Cu/L at the same hardness values. Figure 5.1 describes freshwater copper WQC over a range of hardness values.

5.2.2.6 Freshwater and the Biotic Ligand Model

In recognition of an emerging understanding that there are numerous constituents in natural bodies of water that mediate metal toxicity, U.S. EPA (2007) sponsored development of a *Biotic Ligand Model* (BLM) first published by Di Toro et al. (2001), which is now recommended for setting site specific freshwater standards for some metals, including copper. Accurate use of the BLM requires determination of pH, dissolved organic carbon (DOC), percent humic acid, temperature, Ca, Mg, Na, K, SO_4, Cl, dissolved inorganic carbon (DIC) and sulfide. These data have not routinely been collected by states, but some are available in USGS databases (see Chapter 8). The U.S. EPA (2007) has used available data meeting their data quality objectives to arrive at the following National Criteria Statements for freshwater and marine environments.

Freshwater acute

Freshwater aquatic organisms and their uses should not be affected unacceptably if the 24-h average dissolved copper concentration does not exceed the BLM-derived site-LC_{50} divided by two more than once every 3 y on average. These are the CMC values in the BLM output.

Freshwater chronic

Except where a locally important species is very sensitive, freshwater aquatic organisms and their uses should not be affected unacceptably if the 4-d average concentration of dissolved copper does not exceed the BLM-derived site-water LC_{50} divided by the Final Acute-Chronic Ratio (FACR).

Table 5.12 Historic U.S. EPA recommendations for copper WQC.

Freshwater	
	Acute[a] Cu WQC = 0.960*exp0.9422[ln(hardness)] – 1.464
	Chronic[b] Cu WQC = 0.960*exp0.8545[ln(hardness)] – 1.465
Marine	
	Acute[a] Cu WQC = 4.8 µg dissolved Cu/L
	Chronic[b] Cu WQC = 3.1 µg dissolved Cu/L

a. Acute WQC are 1-h averages not to be exceeded more than once every 3 y on average.

b. Chronic WQC are 4-d average concentrations not to be exceeded more than once every 3 y on average.

Implementation of the freshwater BLM-based standard

The U.S. EPA provides guidance and recommendations. However, States and Tribes are responsible for setting WQC within their jurisdictions. The BLM is designed to develop site-specific criteria for stream reaches. This requires site specific water quality information. However, USGS data (Chapter 8) can be used to define default values for many of the required inputs. It appears that DOC, pH and hardness are the dominant parameters most affecting BLM output. Default values for these and other parameters will be provided in Chapter 8. Existing state regulatory requirements are recommended as copper WQC in interpreting model output. Until States and Tribes adopt the recommendations made by U.S. EPA (2007), the existing hardness based WQC will remain the criteria. In all waters where there are listed salmonids, the BLM CMC is recommended as an additional benchmark for protecting salmonid olfaction.

Montgomery's Pond as an example

Montgomery's Pond (Brooks 2003) provides an example of the use of the BLM to assess copper toxicity. The water in this pond has low hardness of 15.8 mg $CaCO_3$ leading to a chronic copper WQC of 2.11 µg Cu/L using the existing hardness based standard. The BLM predicted an LC_{50} of 11.2 ± 1.9 µg Cu/L for *Daphnia magna* and 237 ± 35.5 µg Cu/L for *Pimephales promelas*. The lower value of 11.2 divided by an FACR of 3.23 gives a chronic WQC of 3.47 µg Cu/L. As seen in Chapter 10, significant decreases in the invertebrate drift community were seen at 6.34 µg Cu/L, but not at 4 µg Cu/L suggesting that the BLM provided a more realistic basis for assessing water quality than did the hardness based WQC.

5.2.2.7 Toxicity associated with dissolved copper in marine environments

Roesijadi (1978) reported that copper is normally present at relatively high concentrations in the tissues of marine animals (>1,000 µg/kg dw). Roesijadi (1987), Harrison et al. (1987) and Harrison and Lam (1985) reviewed the detoxification of copper by *Mytilus edulis, Protothaca staminea, Patella vulgata, Ostrea edulis* and *Littorina littorea*. Copper detoxification and metabolic regulation were associated with copper binding by low and high molecular weight metallothionein-like proteins in the digestive gland and the sequestering of copper in lysosomes. Costlow and Sanders (1987) used a metal-chelate buffer system to regulate the free ion concentration of copper in seawater. They exposed crab larvae to a range of free cupric-ion

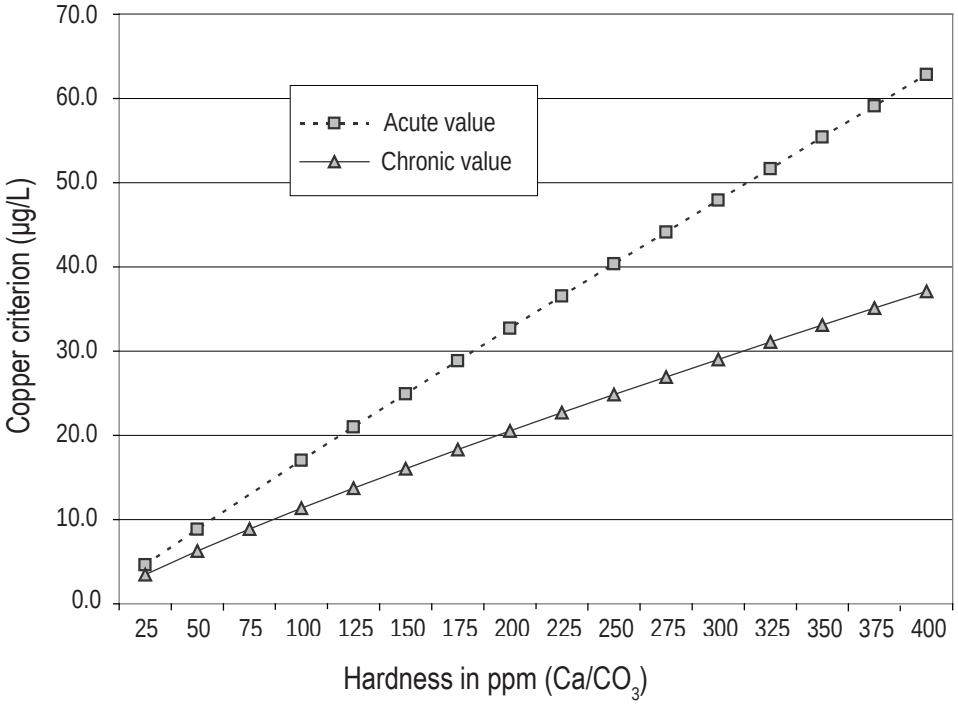

Figure 5.1 U.S. EPA chronic and acute copper criteria for freshwater. The copper standard is presented in µg/L and hardness values in mg ($CaCO_3$)/L.

concentrations and monitored survival, duration of normal development and growth. The authors reported significant reductions in growth correlated with copper accumulation and concluded that when crab larvae are exposed to cupric ion concentrations in seawater below ambient concentrations, they were able to regulate the bioconcentration of copper. At high Cu^{2+} concentrations, bioconcentration increased in an unregulated manner and larval growth was inhibited.

Harrison et al. (1987) conducted copper bioassays on a number of marine invertebrate and vertebrate species. They found that *Crassostrea gigas* embryos were most sensitive (48-h LC_{50} =10 µg/L) and larval herring the least sensitive (48-h LC_{50} was 2,000 µg Cu/L). Dinnel (1983) reported a low LC_{50} (1.9 µg/L) for the sperm of the red sea urchin (*Strongylocentrotus franciscana*). This value seems suspect because it falls within the range normally expected in unpolluted seawater. Reported values from the Dinnel (1983) study are presented in Table 5.13.

5.2.2.8 Summary for dissolved copper toxicity

Gametes and embryos of marine organisms are most sensitive to copper. Based on the previous discussion regarding the metabolic regulation of copper, it seems reasonable to suggest that the susceptibility of embryos to even low copper concentrations is associated with their inability to regulate cellular exposure to the cupric ion. Copper concentrations low enough to protect embryos are sufficient to insure that toxic effects are not imposed on larvae and adult organisms. With the exception of the sperm of the red sea urchin, concentrations <6 µg/L appear reasonable for the protection of marine life. In areas where red urchins spawn, additional restrictions might be necessary. This review revealed little copper toxicity data that included an analysis of the form of copper used in the bioassay. Most toxicity data are reported on the basis of total or dissolved copper. Because of the variety of molecular structures containing copper in aquatic environments, and a lack of definitive information about their relative toxicity, no single analytical measurement is ideal for expressing copper concentrations with respect to their potential toxicity to aquatic life. Baldwin (1989), advised that active copper (operationally defined by acidifying the aqueous sample to pH = 4 with nitric acid and measuring the concentration of copper that passes through a 0.45 micron membrane filter is probably the best available measurement.

Table 5.13 Total copper toxicity measured in controlled bioassays. Values are EC_{50} or LC_{50} in µg/L.

	Taxa	EC_{50} or LC_{50}
Sperm		
	Purple sea urchins	34.0
	Oysters	12.1
	Salmon	44.2
Embryos		
	Purple sea urchins	6.3
	Oysters	6.1
	Mussels	21.0–35.0
Larvae		
	Crab zoea	95.7
	Squid	309.0
	Cabezon	95.3
Adults		
	Sand shrimp	898.5
	Shiner perch	417.7
	Coho salmon smolt	601.0

5.2.2.9 Protocols for digesting sediments prior to metal analysis

Diks and Allen (1983) examined the bioavailability of different forms of copper associated with sediments. In their study, the distribution of copper was determined by assessing different concentrations of sedimented copper (0.0, 2.5, 5.0, 7.5 and 10.0 mg Cu/kg) in five geochemical fractions of chemically extracted sediments, and in tubificid worms. They used five chemical extraction protocols with a range of aggressiveness in liberating copper from the five geochemical compartments being considered. The least aggressive was 1.0 M $MgCl_2$, pH 7, with extraction at room temperature for 1 h. This procedure was considered appropriate for extracting only the absorbed/exchanged copper. The most aggressive procedure was 1.0 M NH_2O_2. HCl in 25% HOAc, with extraction at 96°C for 6 h. This procedure was considered sufficient to extract all copper including moderately reducible forms incorporated into the crystalline structure of iron oxides. Diks and Allen (1983) found that free ionic metals, as well as most metals ion exchanged onto fine-grained solids were biologically available. Less available forms included metals contained in solid organic materials or precipitated and coprecipitated metal oxide coatings. Metals incorporated into crystalline structures were not biologically available. Regression

analysis was used to evaluate the effects of the extraction technique and metal levels in each of the geochemical compartments on copper uptake by the tubificid worms. They found that only the copper extracted from the manganese oxide/easily reducible phase was significantly correlated ($\alpha = 0.05$) with copper uptake. They suggested that the redox potential and pH in the gut of the worm was such that manganese oxide coatings were dissolved during digestion making the copper available for uptake. This study suggests that the 0.1 M $NH_2OH.HCl$ + 0.01 M HNO_3, pH = 2 extraction, conducted at room temperature for 30 minutes (See Chao 1972) is most appropriate for determining biologically available copper in sediments. This is important because in the four sediments tested at 10 mg Cu/L (Des Plaines, Calumet, Flatfoot and Wabash), the proportion of copper biologically available in the amended sediments averaged 72%. The remaining 28% was found in geochemical phases that appeared to not be biologically available. Even more striking was the distribution of copper in the natural (unamended) sediments. In these natural sediments, only 35% of the total copper burden appeared to be biologically available while 65% was incorporated in biologically unavailable geochemical phases. The purpose of this discussion is to suggest that extraction techniques and the biological availability of copper in sediments are important parameters for determining sediment metal concentrations when conducting risk assessments. For purposes of using the model presented in Chapter 9 a strong acid digestion is recommended. This may overestimate biologically available metal concentrations, but the method is recommended by PSEP (1996) and is readily available at commercial analytical laboratories. A total sediment digestion, using hydrofluoric acid at high temperature is not available at many commercial laboratories and will overestimate bioavailable metals confounding attempts to correlate biological endpoints with sediment concentrations.

5.2.2.10 Biological response to sedimented copper

Cain et al. (1992) compared copper concentrations in the insect orders Trichoptera and Plecoptera with concentrations in mine waste contaminated sediments on the Clark Fork River in Montana. They observed sediment concentrations of 779 mg/kg in river reach 0–60; 408 mg/kg in river reach 107–164 and 129 mg/kg in reach 192–381. These levels were significantly elevated above the 18 mg/kg observed at unaffected reference sites. They found significant variability in uptake between various taxonomic and functional groups. Detritivores contained higher concentrations of copper than either omnivores or predators. This was especially true in the most contaminated reach (0–60 km). An appropriate analysis of the community structure was not presented. Flemming and Trevors (1988) dosed a calcareous, southern Ontario stream sediment with up to 10,000 mg Cu(II)sulfate/kg dw and examined its uptake of copper and the microbial response. They found that sediment uptake of copper was nearly 100% to 2,800 mg Cu/kg. At higher levels of copper, the sediment uptake capacity was diminished and at 10,000 µg Cu/g sediment, only ~60% of the copper was removed from the water column. Aerobic heterotrophic bacteria were unaffected at the end of 2 mo in sediments amended with as much as 1,000 mg Cu/kg sediment. Bacterial colony counts actually increased at higher copper concentrations. The authors attributed this to the development of a population of copper tolerant microorganisms. The bacterial community from the high copper amended sediments displayed a 500-fold increase in copper tolerance over bacteria from control sediments when plated on nutrient agar amended with excess copper. The authors suggested that 87.5% of the copper added in these studies was transformed from the toxic Cu^{2+} form to carbonate complexes (87.5%); 12% was complexed with dissolved organic matter and that only 0.5% was available as potentially toxic copper hydroxide complexes or as the toxic Cu^{+2} free ion. The point in this discussion is that calcareous sediments can significantly reduce the toxicity of very high concentrations of cupric ions. At least that statement appears true for the microbial community.

Munkittrick et al. (1991) examined the response of aquatic invertebrates to a gradient of copper and zinc contamination associated with mining activities along the Manitouwadge chain of lakes in northern Ontario. Their data are summarized in Table 5.14. The 22.7 ± 6.4 mg Cu/kg sediment at Station (3) on unaffected Loken Lake (LOK) is not significantly different from the level of 25 ± 8 mg Cu/kg sediment at Station (3) on impacted Manitouwadge Lake (MAN). However, the water column copper concentration at unaffected LOK was only 1.7 µg/L compared with 9.8 µg/L at significantly impacted MAN. Station (1) had the highest level of sediment copper (160 mg/kg) of the three MAN stations. It also had the highest abundance (12,838 invertebrates/m^2), the highest diversity (21 species/sample), and the highest number of typically sensitive cladocerans compared with the other two MAN stations,

each of which had lower sedimented copper. Munkittrick et al. (1991) also presented detailed enumeration of the chironomid species in each lake. Interestingly, for nearly all genera of chironomids, the MAN station with the highest concentration of sedimented copper also had the highest number of chironomid species. It is interesting to note that the copper-intolerant chironomid genera *Polypedilium, Cladotanytarsus,* and *Tanytarsus* were abundant in the control lake LOK and present only at Station (3) (the station with the highest sediment concentration of copper) in affected Manitouwadge Lake.

In contrast, in Loken Lake there was an apparent decrease in the number of sensitive amphipods, gastropods and oligochaetes at the station with the highest sedimented copper concentration (22.7 mg Cu/kg sediment at Station 3). In contrast, most chironomids genera were more abundant at Station (3) than at other stations having lower sediment copper concentrations. The exception was *Polypedilium simulans*, which was observed in much lower abundance at Station (3) than at the other stations. These observations suggest that the primary invertebrate response in these lakes was associated with elevated water column concentrations of copper or other documented factors and not to the sedimented copper concentrations, which spanned a broad range. This is likely because the sedimented copper was not biologically available. The point is that the elevated water column concentrations of copper in affected Manitouwadge Lake appear to be masking any effect associated with sedimented copper up to the observed level of 160 mg Cu/kg.

Miller et al. (1992) also examined the Manitouwadge chain of lakes. They reported average water column concentrations of 15 µg Cu/L in Manitouwadge Lake at a hardness of 110 mg $CaCO_3$/L. This exceeds the U.S. EPA copper criterion for freshwater (12.3 µg Cu/L). Sedimented copper in Manitouwadge Lake averaged 93 mg Cu/kg. No significant difference was observed in the standard length, weight, age or condition factor of white suckers between Manitouwadge Lake and Loken Lake. Copper concentrations in invertebrate tissues were significantly correlated (Spearman's correlation at $p \leq 0.01$) with water column concentrations of copper, but not with sediment copper concentrations over a wide range of values.

Kraft and Sypniewski (1981) studied the effects of high sediment copper concentrations on the macroinvertebrate community of the Keweenaw Waterway. They found high concentrations of copper (<589> mg Cu/kg dw) in areas where the sediment consisted of ~66% silt and clay, and lower concentrations averaging 33 mg Cu/kg dw in areas where silt-clay averaged 27% of the sediment. They observed significant differences in community structure with *Hexagenia, Tanytarsus, Peloscolex, Sphaerium* (mollusk) and *Pontoporeia* (arthropod) virtually excluded from the area with the high copper content (and fine-grained sediments). In contrast, the area with high sediment copper concentrations held more individuals in the genera

Table 5.14 Summary of copper concentrations in the water column and sediments of reference Loken Lake (LOK) and impacted Manitouwadge Lake (MAN). Significant macro-invertebrate data are included to show faunal responses. All values are in mg/kg. Data are taken from Munkittrick et al. (1991).

End point	LOK (1)	LOK (2)	LOK (3)	MAN (1)	MAN (2)	MAN (3)
Sediment copper	7.5	4.0	22.7	160.0	123.0	25.0
Water copper	0.0032	0.0013	0.0017	0.0098	0.0095	0.0098
Cladocera	1,484	1,746	5,326	437	175	87
Copepoda	172,023	1,383	4,366	1,834	1,048	262
Chironomids	11,701	20,585	13,598	9,868	5,502	4,017
Procladius	1,659	1,878	4,803	1,834	2,358	873
Cryptotendipes	262	74	0	1,048	0	87
Pagastiella	175	144	87	4,629	3,057	1,310
Polypedilium	5,852	4,544	1,572	0	0	0
Cladotanytarsus	873	6,113	1,921	1,397	0	175
Tanytarsus	1,659	5,458	2,620	262	0	175
Total abundance	47,069	36,083	25,737	12,838	8,035	5,240
Diversity	36	35	28	21	8	17

Chironomus, Atribelos, Limnodrilus, Ceratopogonidae, and *Dicrotendipes.*

Moore et al. (1979) compared sediment concentrations of arsenic, mercury, copper, lead and zinc with infauna in a series of lakes downstream from the Con Mine in the Canadian subarctic. In general, all of the metals were significantly elevated in the upstream water column and sediments, complicating the analysis. Observed metal and infauna data are summarized in Table 5.15. Sediments and the water column in Meg Lake were significantly impaired by each of the metals investigated. The most common species was the bivalve, *Pisidium casertanum*, which tolerated all of the metals. Seven chironomid and six mollusk species were observed in Keg Lake where very high concentrations of dissolved and sedimented metals were observed. Cironomids represented up to a maximum of 60% by numbers in the benthos with *Procladius culiciformis* and *Psectrocladius barbimanus* dominating. Unlike Meg Lake, *Pisidium casertanum* was rare in Keg Lake with *Physa jennessi, Valvata sincera* and *Lymnaea elodes* dominating at various times of the year. Metal levels between Meg and Keg Lakes were similar and it must be assumed that other environmental parameters were responsible for the shift in the mollusk community. Metal levels dropped significantly in Peg Lake where a total of 14 species were found (8 chironomids, 5 mollusks and one amphipod). Infaunal abundance increased significantly to 5,500/m^2 in Peg Lake—likely in response to the reduced metal concentrations.

Further reductions were observed in Great Slave Lake. Sedimented copper concentrations were only ~15% and arsenic was only 3% of the maximum found in Keg Lake. Baseline infauna and metals were not evaluated at a remote (control) site in Great Slave and it is not possible to determine whether or not conditions reported in this paper are representative of background. However, 44 species were observed in these samples with a mean abundance of ca. 3,100 infauna/m^2. These data suggest that reasonably abundant and diverse infauna can be associated with copper concentrations as high as 82 mg Cu/kg dw. Puckett et al. (1993) have shown that metals, including copper, are associated with the silt-clay fraction of sediments and that wetlands appear to be important repositories for metals adsorbed to these fine grained sediments. This finding supports the conclusion that copper adsorbs to silt and clay rather than the more coarse fractions of sediment.

Rehfeldt and Sochtig (1996) observed high metal tolerance in *Baetis rhodani*. The larvae of this species are scrapers, picking up diatoms from the surface of stones. Depending upon the developmental stage and the availability of food, *B. rhodani* can also feed on detritus. It is a polyvoltine species, occurring in different larval stages in rivers at all times of the year. Sediments in rivers studied by Rehfeldt and Sochtig (1996) contained between 30.7 and 2,917.4 mg Cu/kg dw. *Baetis rhodani* contained between 64.0 and 226.2 mg Cu/kg dw. Copper content in the larvae was highly correlated with sediment copper concentrations (Spearman rank correlation coefficient = 0.94, $P < 0.01$). Table 5.16 describes the sediment bioconcentration factor from sediments for this species. The data are from Rehfeldt and Sochtig (1996).

Water in these rivers was described as "soft" with neutral pH (7.1 to 8.5). The sediments were dried, ground to a powder, sieved to a particle size of <2 mm. Metals were extracted by boiling in 100 ml of nitrohydrochloric acid for an unspecified period of time. This aggressive extractive technique may have liberated copper from other than

Table 5.15 Comparison of metal levels and infauna at four lakes downstream from the Con Mine in the Canadian subarctic. All metal concentrations are in mg/kg (dry sediment weight) or mg/L in water.

	Meg Lake		Keg Lake		Peg Lake		Great Slave Lake	
Endpoint	Sediment	Water	Sediment	Water	Sediment	Water	Sediment	Water
Arsenic	539	2.000	349	1.900	76	0.700	12	0.020
Mercury	132		47		80		53	
Copper	477	0.200	544	0.050	106	<0.020	82	<0.020
Lead	11	0.100	8	0.100	8	<0.020	14	0.008
Total number species	9		13		14		44	
Number insect species	5		7		8		25	
Number mollusk species	4		6		5		10	
Total infaunal abundance	800		1300		5500		3100	

biologically available geochemical partitions as previously discussed. This would help explain the wide variability in sediment BCF (0.06 to 2.08) documented in Table 5.16 for a single species. Alternatively, there may be some copper regulation occurring because the copper concentration in *B. rhodani* is fairly constant, varying only by a factor of 3.5, with the lowest tissue burdens associated with the lowest sediment burdens. It is also interesting to note that dissolved copper concentrations in the River Oker were very high at 132.9 ± 53 µg Cu/L further suggesting that *B. rhodani* can regulate copper uptake. Significant differences were observed in the macrobenthic communities associated with polluted and unpolluted rivers by Rehfeldt and Sochtig (1996). They found that gammarid amphipods were particularly intolerant of copper and that mayflies of the genus *Baetus* were highly tolerant to copper. Other species in the EPT group were found in both polluted and unpolluted streams but at generally reduced numbers in polluted areas. Chironomids were found in reduced numbers in polluted streams suggesting that tolerant chironomid species were probably not present in these watersheds.

Cairns et al. (1984) spiked control sediments from the Tualatin River and Soap Creek Pond with varying levels of copper to achieve sedimented copper levels varying between 59 mg/kg dw and 10,600 mg/kg dw. Overlying water in these experiments was continually renewed until the sediments and water came into equilibrium. They then conducted sediment bioassays using sensitive species of arthropods (*Chironomus tetans, Daphnia magna, Gammarus lacustris,* and *Hyalella azteca*). A summary of the test conditions and results are provided in Table 5.17. There was little or no difference between control survival and survival of any species in sediments from Soap Creek Pond spiked to 488 to 618 mg Cu/kg dw. Nine of the ten *Chironomus tetans* survived for 10 d in sediment copper concentrations of 1080 mg/kg. Four survived at concentrations to 3,950 mg/kg. Control and treatment survival of *D. magna* was equal (9/10) at all sediment concentrations ≤ 400 mg Cu/kg dw. This experiment suggests that copper is not bioavailable in sediment rich in organic carbon and a high percentage of fines (silt and clay). This study also suggests that copper concentrations less than perhaps 600 mg/kg have little biological consequence in these "robust" sediments.

Table 5.16 Heavy metal contents of sediments and larvae of *Baetis rhodani* collected from six rivers in the German Federal Republic. Sediment bioconcentration factors are calculated for each river. All values are in mg Cu/kg dry sediment.

	Cu in		Bioconcentration factor
River	Sediment	*B. rhodani*	
Oker (Probsteib)	2917.4	169.2	0.06
Oker (Schladen 1985)	438.8	226.2	0.52
Ecker	30.7	64.0	2.08
Grane	365.7	168.2	0.46
Laute	155.5	126.5	0.81
Tolle	90.7	110.2	1.21

5.2.2.11 Toxicity summary for sedimented copper

The bioavailability of sedimented copper appears dependent on sediment physicochemical characteristics, including the proportion of fines (silt and clay), the pH of overlying and interstitial water, hardness and dissolved oxygen, and the presence of sedimented organic carbon. Background concentrations of copper reviewed in the assessment varied, as shown in Table 5.18. It appears that sedimented copper concentrations in unpolluted reference areas can vary from 2.0 mg Cu/kg dw to at least 80 mg Cu/kg dw (see Chapter 8). Diks and Allen (1983) suggest that moderately aggressive copper extraction protocols, such as that of Chao (1972) are appropriate for determining the bioavailable copper in sediments. More aggressive protocols using hot acid extraction techniques over extended periods of time will overestimate the amount of bioavailable copper by liberating copper from the lattice structure of other minerals. Given that copper is delivered to sediments from the overlying water column and that water column and sediment concentrations are generally positively correlated, it appears that it is the copper concentrations in the overlying water column and in interstitial water that are most influential on aquatic fauna and flora. Copper

Table 5.17 Summary of sediment types, test conditions, and results of copper spiked sediment bioassays reported by Cairns et al. (1984).

			Sediment 10-day LC_{50}			
Sediment	% TOC	% Silt-clay	*Chironomus tetans*	*Daphnia magna*[a]	*Gammarus lacustris*	*Hyalella azteca*
Tualatin River	1.8	59.3	2296	937	-	-
Soap Creek Pond	3.0	84.8	857	681	964	1078

a. All bioassays were based on a ten day exposure except that for *Daphnia magna* which is a 48-h LC_{50}.

does bioconcentrate, and data in Cain et al. (1992) suggests that infauna, particularly detritivores, can bioaccumulate copper from sediments. Copper does not appear to biomagnify through food webs.

This review suggests that aquatic invertebrates vary significantly in their response to sedimented copper. For instance of the seven genera of midges described in these studies, five are tolerant of sedimented copper concentrations exceeding 100 mg Cu/kg dry sediment. Only the genera *Tanytarsus* and *Polypedilium* appear intolerant at concentrations of 123 mg/kg to 160 mg/kg. It should be noted that the copper concentration in the study of Munkittrick et al. (1991) was 9.5 µg Cu/L to 9.8 µg Cu/L. Water hardness, TOC and DOC were not provided in the paper and it is not possible to assess whether the response of these species was to bioavailable copper in the water or sediments. Based on this review, it appears that *Tanytarsus, Polypedilium, Hexagenia*, *Sphaerium* and *Pontoporeia* are potentially intolerant of sedimented copper. It was not possible from the papers presented to determine whether or not their susceptibility was to sedimented copper or copper in the water column. Confirmation of the susceptibility of these taxa to sedimented copper would require sediment bioassays, such as that performed by Cairns et al. (1984). This review indicates that many species are tolerant of high concentrations of sedimented copper (Table 5.19).

5.2.2.12 Recommended benchmarks for evaluating environmental risks associated with sedimented copper

The model provided in Chapter 9 compares predicted copper accumulation in sediments with benchmarks or SQC. Copper SQC for freshwaters were not found. Based on the preceding discussion, recommended sediment benchmarks for copper are provided below.

Table 5.18 Background freshwater sediment copper levels reviewed in this assessment.

Source	Geographic location	Reported mg Cu/g dry sediment
Siipola (1991)	Lower Columbia River	18.0 to 66.0
Tetra Tech (1994)	Lower Columbia River	19.3 to 49.9
Munkittrick et al. (1989)	Loken Lake, northern ON	22.7 ± 6.4
Munkittrick et al. (1991)	Northern ON	4.0 to 23.0
Cairns et al. (1984)	Tualatin River, OR	59.0
Cairns et al. (1984)	Soap Creek Pond, University of Oregon	210.0
Cain et al. (1992)	Clark Fork River, MT	18.0
Moore et al. (1979)	Great Slave Lake, NT	82.0
Schmidt (1978)	"unpolluted sediments from nearshore areas"	2.0 to 78.0

5.2.2.13 Marine sediment benchmarks and quality criteria (SQC) for copper

Jones et al. (1997) have summarized available toxicological benchmarks for screening contaminants of potential concern for effects on sediment-associated biota. The U.S. Department of Energy accomplished this work in an effort to identify benchmark levels of contaminants that warrant further assessment or those that are at concentrations requiring no further attention. It should be emphasized that these benchmarks were not developed or intended as sediment quality criteria for surface waters. The authors note that, "Sediment benchmarks must not be used as the sole measure of sediment toxicity. Field studies and toxicity tests shall be the primary indicators of toxicity in sediments; benchmarks may be used to determine which chemicals present in the sediment are most likely causing the toxicity." The following approaches, and sediment concentrations for copper, were discussed:

Screening level concentration approach for developing the Ontario Guidelines

This approach estimates the highest concentration of a particular contaminant in sediment that can be tolerated by ~95% of benthic infauna. The screening level concentration approach (SLC) is derived from synoptic data on sediment chemical concentrations and benthic invertebrate distributions. First, the species screening level concentration (SSLC) is calculated by plotting the frequency distribution of the contaminant concentrations over all sites (at least 10) where the species is present. The 90th percentile of this distribution is taken as the SSLC for that species. Next, a large number of SSLCs are plotted as a frequency distribution to determine the contaminant concentration above which 95% of the SSLCs occur. This final concentration is the SLC. Dr. Connie Gaudat, Acting Head Soil and Sediment Section, Evaluation and Interpretation Branch, Guidelines Division of Environment Canada noted that the Ontario guidelines were developed without reference to water or sediment pH, total organic carbon, sediment grain size or any other environmental parameter affecting the fate, transport, deposition and bioavailability of the compounds under consideration (personal communication, Gaudat, 1995). In discussing copper for instance, she stated that documentation of worst cases involving metals may have occurred with very

Table 5.19 Summary of the tolerance of various freshwater taxa to sedimented copper.

Taxon	Relative tolerance	Source	Cu concentration (mg/kg dry sediment)
Cladocera			
Daphnia magna	Tolerant	e	681 to 937
Chironomids			
Chironomus	Tolerant	b,e	589 to 2296
Procladium culciformis	Tolerant	c	477 to 544
Psectrocladius barbimanus	Tolerant	c	477 to 544
Dicrotendipes	Tolerant	b	589
Pagastiella	Tolerant	a	123 to 160
Polypedilium	Intolerant	a	123 to 160
Tanytarsus	Intolerant	a,b	123 to 589
Ephemeroptera			
Hexagenia	Intolerant	b	589
Baetis rhodani	Tolerant	d	< 2,917
Diptera			
Ceratopogonidae	Tolerant	b	589
Molluska			
Sphaerium	Intolerant	b	589
Pisidium casertanum	Tolerant	c	477 to 544
Physa jennessi	Tolerant	c	477 to 544
Valvata sincera	Tolerant	c	477 to 544
Lymnaea elodes	Tolerant	c	477 to 544
Amphipoda			
Pontoporeia	Intolerant	b	589
Gammarus lacustric	Tolerant	e	964
Hyalella azteca	Tolerant	e	1078
Oligochaeta			
Limnodrilus	Tolerant	b	589

a. Munkittrick et al. (1991); b. Kraft and Sypniewski (1981); c. Moore et al. (1979); d. Rehfeldt and Sochtig (1996); e. Cairns et al. (1984).

low pH values or coarse sediments. This may be especially true in Ontario, where the guidelines were developed, because it lies on the continental shield characterized by low soil buffering capacity and low pH. She noted that many pristine areas in Canada have background concentrations that exceed the guidelines. She stated that Environment Canada is developing sediment standards, but until those standards are developed, they (and the Ontario Ministry of Environment) use site specific risk assessments to determine appropriate sediment quality criteria. She said that the site-specific standards are always greater than Ontario's guidelines because they seldom, if ever, encounter the worst case conditions associated with those guidelines. It should be noted that Jones et al. (1997) do not recommend the SLC method even for developing benchmarks against which to assess the potential biological effects of contaminants at hazardous waste sites.

National Oceanic and Atmospheric Administration approach

The National Oceanic and Atmospheric Administration (NOAA) annually collects and chemically analyzes sediment samples from sites located in coastal marine and estuarine environments throughout the United States. These data were used to evaluate three basic approaches to the establishment of effects-based criteria: the equilibrium partitioning approach (applicable to nonionic organic compounds), the spiked-sediment toxicity test approach, and various methods of evaluating synoptically collected biological and chemical data in field surveys (Long and Morgan 1991). Chemical concentrations observed or predicted by these methods to be associated with biological effects were ranked, and the lower 10th percentile [Effects Range-Low (ER-L)] and median [Effects Range-Median (ER-M)] concentrations identified. The ER-L and ER-M values were recalculated by Long et al. (1995) after omitting freshwater data and adding more recent data. Jones et al. (1997) note that the NOAA values may be used to help identify sites with the potential to cause adverse biological effects. They caution that these are not NOAA criteria or standards and are not intended for use in regulatory decisions or any other similar applications. The NOAA ER-L for copper is 34 mg Cu/kg dry sediment and the ER-M is 270 mg Cu/kg dry sediment.

Florida Department of Environmental Protection approach

The Florida Department of Environmental Protection (FDEP) methodology is similar to the NOAA approach. The updated and revised data set used by Long et al. (1995) was also used by MacDonald (1994) to calculate Threshold Effects Levels (TELs) and Probable Effects Levels (PELs). Unlike the ER-Ls and ER-Ms, the TELs and PELs also incorporate chemical concentrations observed or predicted to be associated with no adverse biological effects (no-effects data). The TEL is the geometric mean of the 15th percentile in the effects data set and the 50th percentile in the no effects data set. The PEL is the geometric mean of the 50th percentile in the effects data set and the 85th percentile in the no effects data set. The TEL represents the upper limit of the range of sediment contaminant concentrations dominated by no effects data. The PEL represents the lower limit of the range of contaminant concentrations that are

usually or always associated with adverse biological effects (MacDonald 1994). The FDEP TEL is 18.7 µg Cu/g dry sediment weight and the PEL is 108 µg Cu/g dry sediment weight.

Apparent effects threshold approach

The AET approach uses data from matched sediment chemistry and biological effects measures. Biological effects could be assessed by either benthic community surveys or sediment toxicity tests. An AET concentration is the sediment concentration of a selected chemical, above which statistically significant biological effects always occur. Jones et al. (1997) defended the use of estuarine benchmarks for evaluation of freshwater sites when freshwater benchmarks are not available.

Recommended marine sediment quality standards

Table 5.20 summarizes some of the available benchmarks. The only regulatory sediment quality standard (SQC) is Washington State's AET-based SQC for copper in marine sediments of 390 µg/g (WAC 173-204), which has been adopted for use in the risk assessment model presented in Chapter 9.

5.2.2.14 Freshwater sediment benchmarks and quality criteria (SQC) for copper

The preceding discussion describing the bioavailability and toxicity of sedimented copper suggests that TOC, percent fines (silt and clay), pH, dissolved oxygen, water hardness and alkalinity affect the bioavailability and toxicity of sedimented copper. Until appropriate freshwater sediment quality standards are developed, the benchmarks given in Table 5.21 are provided for evaluating the benthic effects associated with copper lost from preserved wood using the model provided in Chapter 9. These benchmarks are consistent with the range at which threshold effects are reported by Jones et al. (1997) and at which sediment effects were reported in the reviewed literature and they bridge the suggested SQS of 80 mg Cu/L given in WDOE (2003). In addition, they appear consistent with background levels of copper found in many parts of North America (see Chapter 8)

These values are higher than the NOEL and/or TELs described in Table 5.20. It should be emphasized that all of the values in Table 5.20 are based on worst cases and do not take into consideration all of the environmental factors that mediate copper toxicity. For instance, the Ontario screening criteria for sedimented copper (16 µg/L) is based on the worst cases in the Ontario database, an area lying on the continental shield in which worst cases likely involve very low pH, low alkalinity and hardness and coarse (rocky) substrates. As previously discussed, Environment Canada does not recognize these values as standards and cautions that background concentrations in other parts of Canada consistently exceed this screening value with no evidence of toxic effects. The benchmarks in Table 5.21 are consistent with the range (28.5–96 mg Cu/kg dw) of threshold effects levels presented by Jones et al. (1997).

5.3 Arsenic

Arsenic is a suspected human carcinogen and significant concern has been expressed in recent years regarding appropriate concentrations in drinking water. However, arsenic is less toxic to aquatic organisms than is copper. This is evidenced in Washington State's acute WQC of 360 µg As/L in freshwater and 69 µg As/L in seawater compared with copper WQCs of 4.1 µg Cu/L in seawater and 2 to 15 µg Cu/L in freshwater. The following discussion focuses on benchmarks necessary to protect aquatic life from dissolved and sedimented arsenic. Human health is not discussed and therefore the following recommended benchmarks or WQC are not appropriate for managing treated wood in surface waters that are consumed by people.

5.3.1 Fate of arsenic in aquatic environments

The model provided in Chapter 9 assumes that arsenic molecules leached from pressure-treated wood adsorb to clay particles and are sedimented downcurrent from the structure. The arsenic is assumed to accumulate at its first point of intercept with the sediments. In environments subject to wave action and or current speeds fast enough to resuspend fine-grained sediments, it is likely that ad-

Table 5.20 Summary of benchmarks for sedimented copper.

Jurisdiction	Criteria in mg Cu/kg	
Washington State Marine Sediment Quality Criteria	390	
National Oceanographic and Atmospheric Administration	ER-L = 34	ER-M 270
Florida Department of Environmental Protection	TEL = 18.7	PEL = 110
Ontario Ministry of the Environment Screening Levels	SLC Low = 16	SLC Severe = 110

Table 5.21 Recommended benchmarks for assessing the environmental risks associated with sedimented copper lost from pressure-treated wood.

Sediment and water column characteristics	Acceptable levels of sedimented copper
1. Coarse grained sediment (silt and clay <10%)	30 µg Cu/g dry sediment
Total organic carbon < 0.5%	
Moderate to low pH (5.5 to 6.5)	
Low hardness and alkalinity (25 to 35 ppm $CaCO_3$)	
2. Intermediate sediments (silt and clay between 10% and 25%)	65 µg Cu/g dry sediment
Total organic carbon between 0.5% and 1.5%	
Neutral pH (6.5 to 7.5)	
Moderate hardness and alkalinity (35 to 100 ppm $CaCO_3$)	
3. Low energy, well buffered streams and lakes (fines > 25%)	100 µg Cu/g dry sediment
Total organic carbon >1.25%	
Greater than neutral pH (pH > 7.5)	
High hardness and alkalinity (> 100 ppm $CaCO_3$)	

sorbed metals will be resuspended and disbursed. This makes the model conservative in all but depositional deep water environments.

5.3.2 Chemistry of arsenic in water

The chemistry of arsenic in water is complex and the form present in solution is dependent on such environmental parameters as pH, organic content, suspended solids and sediment characteristics. Thermodynamic considerations predict that at neutral pH, and relatively high levels of dissolved oxygen, most arsenic should be oxidized to arsenate. However, Penrose and Woolson (1974) noted that most inorganic arsenic in the sea is in the form of arsenite. Johnson (in Penrose and Woolson 1974) found that marine bacteria can reduce arsenate to arsenite. This biological transformation may be responsible for the 2:1 ratio of arsenite to arsenate observed in some marine water. In contrast, Onishi (in Andreae 1978) reported that arsenite represents only about 20% of the total arsenic found in seawater. In addition to inorganic arsenic, a number of authors (Johnson 1972, Lunde 1977, Penrose et al. 1977, Andreae 1983) reported that bacteria, phytoplankton, marine invertebrates and vertebrates can biotransform arsenic into relatively less toxic organic compounds. These reactions involve methylation and reduction to produce methylarsonic acid and dimethylarsinic acid. The low toxicity of these organic compounds allows high body burdens of arsenic which are eventually incorporated into the sediments. However, significant amounts of arsenate may be regenerated in the water column from phytoplankton that sink below the photic zone and perish. Thus, there is an arsenic cycle which involves a cycling of arsenic through its various inorganic and organic forms. The relatively high levels of arsenic found in sediments, compared to the water column, suggest that the ultimate fate of arsenic is incorporation into sediments.

5.3.4 Bioaccumulation of arsenic in aquatic organisms

Because inorganic arsenic is a potent toxicant in mammals (including man), there is considerable data describing its bioaccumulation. Sanders et al. (1994) note that while there is little direct uptake of dissolved arsenic in water by invertebrates or vertebrates, phytoplankton and macroalgae do bioconcentrate arsenic, resulting in its introduction into food webs. This is especially true for herbivores (Sanders et al. 1989). However, while bioconcentration in autotrophs may be high, bioaccumulation in higher trophic levels is reported to be low. In studies of arsenic transport from the Baltic Sea, less than 2% of the total dissolved arsenic was incorporated into biotic tissue (Blanck et al. 1989). In feeding experiments with planktonic communities from Chesapeake Bay, Sanders et al. (1989) found only 7% to 10% of the arsenic in phytoplankton and less than 1% of total arsenic was incorporated into invertebrates feeding on the phytoplankton. Penrose et al. (1977) examined the arsenic budget in a sea urchin-alga system and concluded that organic arsenic is rapidly excreted by most organisms and therefore, while there may be significant bioconcentration, there is no apparent bioaccumula-

tion in food chains. Organisms containing high concentrations of arsenic tend to be those that are prone to incidental ingestion of sediment particles while feeding. Arsenic concentration from ambient water was also reported by Schroeder and Balassa (1966), Lunde (1970, 1972) and Fowler et al. (1975). High concentrations of arsenic in marine animals were reported by USDA (1980) from around the world. Reported levels of arsenic, expressed as a proportion of wet tissue weight, for some typical marine species are provided in Table 5.22 (USDA 1980). Woolson (1977) reported that arsenic concentrations were 10 to 100 times higher in marine fish and shellfish than in fresh water species. However, as seen in previous sections, reported arsenic concentrations in marine waters are typically lower (1.5 µg As/L) than in fresh water (see Chapter 8). No plausible explanation for this apparent contradiction was found.

Table 5.22 Arsenic content of aquatic animal life (in µg/kg ww).

Marine	
Crab	27,000–52,500
Clams (all species)	900–12,720
Oysters (*Crassostrea virginica*)	600–42,750
Lobster (*Panulirus borealis*)	3,200–9,600
Tuna	710–4,600
Freshwater	
Trout (*Oncorhynchus sp.*)	69–149
Perch (*Perca fluviatilis*)	600
Bass (*Micropterus salmoides*)	70–930
Channel catfish (*Ictalurus punctatus*)	0–3,100

Penrose and Woolson (1974) reviewed studies by Fernandez del Riego, Seydel, and Lunde suggesting that arsenic is not bioaccumulated in food chains. Work by Boothe and Knauer and by Black and Penrose (both cited in Penrose and Woolson 1974) suggested that arsenic ingested in food is rapidly excreted by marine organisms. Woolson (1975) summarizes his review of arsenical bioaccumulation by noting that

> Arsenic is bioconcentrated by aquatic organisms but not biomagnified. Plants usually accumulate more arsenic than fish, and crustacea accumulate intermediate amounts. Marine organisms normally contain more arsenic than their fresh water counterparts. However, the arsenic contained in the organisms is apparently not toxic to animals or humans, and is readily excreted.

The available evidence indicates that arsenic does not bioaccumulate in food chains. It appears that arsenic ingested at lower levels of the food web is converted to organic molecules which may be rapidly excreted at the next trophic level. This analysis assumes that concentrations of arsenic in aquatic animals are dependent on ambient water concentrations and that they are not bioaccumulated in higher trophic levels.

5.3.5 Arsenic toxicity to aquatic biota

The toxic properties of arsenic have been known for centuries. The toxicology of arsenic may be divided into three general areas: direct inhibition of cellular respiration, mutagenic effects and hemolysis. Baroni et al. (1963) and Penrose and Woolson (1974) noted that controlled attempts to attribute carcinogenic properties to the arsenicals have failed. Ferm (1977) has demonstrated the teratogenic nature of sodium arsenate injected into a variety of experimental animals. Arsenic toxicity is dependent on the oxidation state, chemical form and route of exposure. In general, arsenic acids are least toxic, followed by inorganic arsenate, arsenoxides and inorganic arsenite. The trivalent organic and inorganic arsines are the most toxic. Sorensen (1991) concluded that two to ten higher concentrations of arsenate (compared with arsenite) are required to achieve the same level of toxicity. Acute arsenic exposure to concentrations exceeding the reported LC_{50} values can result in immediate death of fish because of arsenic induced increases in mucus production, resulting in suffocation, or degeneration of gill epithelium. Chronic exposures can result in the accumulation of arsenic to toxic levels. The detoxification role of the liver places this organ at risk. The liver is considered the site of greatest damage associated with acute arsenic intoxication. Renal and splenic tissues also concentrate elevated levels of arsenic because of their roles in urinary and erythrocyte filtration.

Eisler (1988) reported acute toxicity concentrations for a variety of fresh water and marine plants and animals associated with several species of arsenic. Lethal Concentrations which killed 50% of the invertebrate test organisms (LC_{50}) are provided in Table 5.23 (data from Eisler 1988, and Sorensen 1991). In marine water, it appears that arsenic concentrations in excess of 200 µg/L may

result in the mortality of juvenile Dungeness crab and an unspecified species of red algae. NTIS (1986) reported acute concentrations of arsenic (III) for twelve saltwater animals ranging from 232 to 16,030 µg As/L. Chronic stress is observed at about half these values or 116 µg/L. Arsenic (V) was less toxic for the two invertebrates examined with acute values of 2,000 and 3,000 µg/L. None of these animals were as sensitive to arsenic as were algae which showed toxic responses to either arsenic (III) or (V) at concentrations as low as 19 µg/L.

Eisler's (1988) data and that of Sorensen (1991) suggest that in fresh water, arsenic concentrations associated with acute toxicity appear to be a somewhat higher, in the neighborhood of 900 µg As/L. NTIS (1986) reported acute toxicity associated with arsenic (III) in 16 species of freshwater animals. An acute concentration of 812 µg/L was found for cladocerans. At the other end of the range, the acute concentration for a midge was 97,000 µg/L. From these papers it appears reasonable to assume an LC_{50} of 800 µg/L for the more sensitive freshwater species. NTIS (1986) indicated that chronic stress is encountered by all freshwater species at about 21% of their acute values. For the most sensitive species, this value would be 168 µg As/L. This review indicates that arsenite can cause chronic stress at levels as low as 168 µg As/L in fresh water systems and 230 µg As/L in marine systems. For most aquatic organisms, arsenate is less toxic. However, for the most sensitive marine algae, this review indicates no difference in toxicity thresholds between the two primary valence states of arsenic (+3 and +5) and toxicity thresholds as low as 19 µg/L are necessary to protect the most sensitive species of marine algae.

Table 5.23 Lethal levels of arsenite in freshwater plants and animals. Unless otherwise specified, values are for the LC_{50} expressed as µg As/L.

Freshwater	
Taxa	Arsenite (As^{+3})
Algae	1,700-4,000 (LC_{100})
Cladocerans	1,300
Amphipod	960 (28-d LC_{100})
Goldfish	490 (7-d LC_{50})
Salmo gairdneri	25,600 (96-h LC_{50})
Ictalurus punctatus	15,000 (72-h LC_{50})
Lepomis microchirus	35,000 (96-h LC_{50})
Marine water	
Taxa	Total Arsenic (As)
Red algae	300 (LC_{100})
Copepods	510
Dungeness crab	230
Oyster (eggs)	7,500 (48 h)
Blue mussels	16,000 (LC_{100})
Pink salmon	3,800 (10-d LC_{54})

Table 5.24 Marine and freshwater arsenic WQC in µg As/L.

	Fresh		Marine	
Contaminant	Acute	Chronic	Acute	Chronic
Arsenic	360	190	69	36

5.3.5 Recommended benchmarks for evaluating environmental risks associated with arsenic

Marine and freshwater WQC based on the recommendations made by the U.S. EPA (1998) for arsenic are summarized in Table 5.24.

5.3.6 Arsenic sediment quality standards

Washington State has published SQS for metals in WAC 173-204-320. The marine SQS standard of 57 mg As/kg dry sediment weight (dw) is based on an AET. Washington State is developing freshwater SQS (WDOE 2002, 2003). As an example WDOE 2003 suggests a freshwater SQS of 20 mg As/kg dw but the state has not yet adopted a standard. A range of published benchmarks for freshwaters is provided in Table 5.25. None of these benchmarks have currently been adopted as SQS. In each case the mean of the lower and upper benchmark is provided as guidance for adopting an interim standard. It appears that the suggested Washington State freshwater sediment SQS of 20 mg As/kg dw is within the range of the means of the lower and moderate benchmarks and this value is adopted as an interim standard for use in the model provided in Chapter 9. Arsenic losses from ACZA and CCA-C pressure-treated wood are generally low and as shown in Chapter 10, large increases in sedimented arsenic have not been seen near pressure-treated wood structures in either freshwater or marine environments.

Table 5.25 Published arsenic freshwater sediment benchmarks.

Benchmark	µg As/L
Apparent effects threshold (AET)	40
Effects range low (ERL)	12
Effects range moderate (ERM)	33
Mean of ERL and ERM	22.5
Threshold effects level (TEL)	5.9
Probable effects level (PEL)	17
Mean of TEL and PEL	11.5

5.3.7 Summary of the fate and effects of arsenic in aquatic environments

Dissolved arsenic is less toxic than dissolved copper and in general, if copper is managed in association with pressure-treated wood structures, then arsenic will not reach concentrations in either water or sediments that adversely affect biota. States have adopted U.S. EPA recommended acute arsenic WQC of 360 µg As/L in freshwater and 69 µg As/L in seawater. Washington State's marine SQS of 57 mg As/kg dw sediment is the only adopted SQS found in this review. Several freshwater sediment benchmarks were reviewed, but none have been adopted. WDOE (2003) has suggested a freshwater SQS of 20 mg As/kg dw and this is adopted as an interim benchmark until regulatory SQS are published.

5.4 Chromium

CCA-C is the only preservative currently using chromium. The metal is added to facilitate complexation and binding of copper and arsenic within the cellular structure of the wood. Chromium loss from wood treated with CCA-C using WWPI/CITW (1996) BMP have been measured (Chapter 7) at 0.11 µg Cr/cm^2-d in freshwater and 0.03 µg Cr/cm^2-d in 30 PSU seawater during the first day of immersion. Long-term loss rates in either environment were <0.000. If CCA-C preserved wood projects are managed to maintain copper below biological effects concentrations, then no effects associated with chromium should be anticipated. This statement is supported by the very low chromium concentrations in water and sediments near CCA-C preserved wood bridges reported by Brooks (2000a and 2000b) and reviewed in Chapter 10. The model provided in Chapter 9 assumes that chromium leached from pressure-treated wood adsorbs to clay particles and is sedimented downcurrent from the structure where it is assumed to accumulate.

5.4.1 Fate of chromium in aquatic environments

The ultimate fate of chromium VI appears to be incorporation into fine grained sediments with high organic and iron content. Adsorption is dependent on salinity and is greatest at salinities of 0.1 to 1.0 PSU (Mayer and Schick 1981). Chromate, hydrochromate and dichromate are somewhat soluble in water, allowing migration into and out of the water column over aerobic sediments but less so from anaerobic sediments. Observed concentrations in European estuaries ranged from 3.9 mg Cr/kg dw in intertidal sands to 162 mg Cr/kg in anaerobic muds (Rehm et al. 1984). Chromium(III) forms stable complexes with negatively charged inorganic and organic compounds. It is rarely found in waters with decaying plant or animal tissues or silt and clay particles. Precipitated chromium(III) hydroxides remain in the sediments under aerobic conditions. With low pH and anoxic conditions, chromium(III) hydroxides may solubilize. However, Lu and Chen (1977) found that chromium(III) was not significantly released from sediments into seawater under all redox conditions.

5.4.2 Chromium bioaccumulation

Eisler (1986) reported that algae and higher plants accumulate chromium from seawater by factors of up to 8,600 and from solutions containing 50,000 µg Cr/L chromium by a factor of 18 in 48 h. Although chromium is abundant in primary producers, there is little evidence of bioaccumulation through marine food chains. Baptist and Lewis (1969; cited in Eisler 1986) followed the transfer of chromium through an experimental food chain and observed a decline in the concentration of chromium through each of four trophic levels. Comparison of the results of this food chain study with measurements of direct chromium uptake from seawater suggest that direct uptake is a far more important pathway than assimilation through the food chain. Bioconcentration factors (BCF) for numerous aquatic species are given in U.S. EPA (1983). The reported BCF for chromium (VI) in fish muscle is less than 1.0. Values of 125 and 192 were found for chromium (VI) in oysters and blue mussels. The EPA document also gives values for chromium (III) and concludes that they are similar to chromium (VI). The EPA conclusion was that mean BCF values of 0.5 and 130 are appropriate for fish muscle and bivalve mollusks respectively. Both values are relatively low. It appears that chromium is not bioaccumulated in the food chain and that chromium concentra-

tions at all trophic levels are primarily a function of background concentrations in water.

5.4.3 Acute toxicity associated with dissolved chromium in aquatic environments

The toxicity of chromium to aquatic species can vary by an order of magnitude, or more, depending on a variety of biological and physicochemical factors. These include differences associated with species, age, developmental state, temperature, pH, salinity, length of exposure and interaction with other contaminants. Chromium(III) is less toxic than chromium(VI). Most of the chromium(VI) found in nature is a result of domestic and industrial emissions (Steven et al. 1976). Interaction of chromium(VI) with organic compounds can result in reduction to chromium(III). However, in aerobic marine environments, chromium(VI) is the more abundant species. It is most toxic to the developmental stages of aquatic species in soft, fresh water, with low pH. Eisler (1986) reports a 96 h-LC_{50} of 200 µg Cr/L for salmon fingerlings and 495 µg Cr/L for rainbow trout (*Oncorhynchus mykis*) eggs. Most species of fish tolerate >10,000 µg Cr/L and bluegills (*Lepomis macrochirus*) demonstrated a very high toxic threshold at 213,000 µg Cr/L in water with 120 ppm $CaCO_3$. In marine water, chromium VI toxicity also varies by orders of magnitude, depending on the taxa. Polychaetes and larval crabs (*Callinectes sapidus*) are the most susceptible organisms at 200 and 320 µg Cr/L respectively.

5.4.4 Recommended benchmarks for evaluating environmental risks associated with chromium

5.4.4.1 Water quality criteria

Most jurisdictional water quality criteria (WQC) are based on recommendations made by the U.S. EPA (1998). Washington State, in Chapter 173-201A WAC, defines water quality standards for surface waters. The WAC states that toxic substances shall not be introduced above natural background levels in waters of the state at concentrations which have the potential either singularly or cumulatively to adversely affect characteristic water uses, cause acute or chronic toxicity to the most sensitive biota dependent upon those waters, or adversely affect public health, as determined by the Department of Ecology. Table 5.26 lists criteria established for the protection of aquatic life in Washington State.

Table 5.26 U.S. EPA recommended and Washington State adopter chromium WQC for surface waters. A hardness of 60 mg $CaCO_3$/L was used in Equations 3 and 4 for values requiring computation.

	µg Cr/L			
	Fresh		Marine	
Contaminant	Acute	Chronic	Acute	Chronic
Chromium(VI)	16.0	11.0	1,100.0	50.0
Chromium(III)	361.1	130.87	—	—

The U.S. EPA/Washington State WQC for chromium(III) in freshwater is based on hardness (Equation [5.1] for acute criterion and Equation [5.2] for the chronic criterion).

Criteria for chromium VI and in marine environments are summarized in Table 5.26. The acute criterion is a 1-h average concentration and the chronic criterion is a 4-d average neither of which is to be exceeded more than once every 3 y. These WQC, applied at site specific hardness are used in the model presented in Chapter 9 to assess the environmental response to the use of CCA-C pressure-treated wood. However, due to its relatively low toxicity and very low loss rate from BMP produced CCA-C preserved wood, chromium is not expected to be a deciding factor in risk assessments.

$$\text{Acute chromium(III) WQC} = 0.316*\exp^{(0.8190[\ln(\text{hardness})] + 3.688)}\ \mu g/Cr_{III}/L \quad [5.1]$$

$$\text{Chronic chromium(III)WQC} = 0.860*\exp^{(0.8190[\ln(\text{hardness})] + 1.561)}\ \mu g/Cr_{III}/L \quad [5.2]$$

5.4.4.2 Sediment quality standards (SQS)

Washington State has developed marine SQS in WAC 173-204-320. The chromium standard is 260 mg Cr/kg dw sediment. Freshwater SQS are not presently available. However, Washington State (WDOE 2002, 2003) is in the process of developing these standards and the suggested freshwater chromium SQS is 95 mg Cr/kg dw. These values are recommended for use in evaluating the environmental response to proposed CCA-C structures using the model presented in Chapter 9.

5.4.5 Summary of the fate and effects of chromium in aquatic environments

Chromium losses from CCA-C pressure-treated wood produced using WWPI/CITW (1996) Best Management

Practices have been found to be very low. If copper concentrations are managed to avoid biological effects at proposed structures, then biological effects associated with chromium should not be anticipated. However, in some instances, such as when high concentrations of Cr VI are present due to natural or other anthropogenic inputs, the additional chromium added by CCA-C preserved wood needs to be evaluated and therefore these losses are considered in Chapter 9.

5.5 Zinc

Chemonite (ACZA) is the only preservative currently using zinc. Chemonite has AWPA Standards for both marine and freshwater uses and is frequently used to treat Douglas-fir piling and timbers, which are refractory to CCA preservative. Zinc loss rates (see Chapter 7) from ACZA preserved piling are similar to copper loss rates. However, zinc is less toxic in aquatic environments and therefore, management of ACZA treated structures typically focuses on environmental concentrations of copper. Zinc concentrations in pristine sediments are typically less than 100 mg Zn/kg dw but can be greater than 700 mg/kg in industrialized areas like Elliott Bay, Washington State. Lu and Chen (1977) reported zinc concentrations of 30 to 35 mg Zn/kg dw from reference sediments in San Pedro Bay, California. The U.S. EPA (1985) reports typical zinc concentrations of 0.50 µg Zn/L in the marine waters of Puget Sound. Dissolved zinc concentrations in freshwater are somewhat higher and more variable. Johnson (1994) reported median concentrations of 1.0 to 1.8 µg Zn/L in four Washington State rivers. Highest concentrations occurred during winter and were well correlated with Total Suspended Solids (TSS) and flow rate. In contrast, Pelletier (1994) reported concentrations of 80.9 to 164 µg Zn/L in the Spokane River in eastern Washington. These values exceed Zn water quality standards (24.1 to 79 µg Zn/L at the reported hardness) during all seasons. The major sources of the zinc were found to be nonpoint from historical mining practices. The point being that although zinc is less toxic than copper, there are instances in which other inputs of this metal require special consideration with respect to managing ACZA treated structures in freshwater.

5.5.1 Zinc bioconcentration and bioaccumulation

Accumulation of zinc by fish is a factor of exposure time, exposure concentration, size of the organism, water hardness, acclimation, feeding level and trophic level. Sorensen (1991) reviewed zinc accumulation in fish and reported bioconcentration factors of 2.8 to 5.1. Unlike copper, which is sequestered primarily in the liver, zinc accumulation is greatest in the eye, followed in order by decreasing concentrations in the kidney, bone, skin, gill and liver. Very little zinc was found in muscle or gonad. Sauer and Watabe (1984) observed significant zinc deposition in the scales of mummichogs (*Fundulus heteroclitus*) exposed to varying concentrations (210 to 7,880 µg Zn/L). Their study suggested that scales take up zinc actively, rather than passively. Baptist and Lewis (1967) examined zinc bioaccumulation in a four level system composed of phytoplankton, brine shrimp, post-larval fish (*Eucinostomus* sp. or *Micropogon undulatus*) and mummichogs *(Fundulus heteroclitus)*. They observed bioaccumulation factors (relative to the amount of zinc in the phytoplankton) of 40 in the post-larval fish fed to satiation on brine shrimp and of 5 in the mummichogs fed a reduced diet of post-larval fish.

5.5.2 Acute toxicity associated with dissolved zinc in freshwater environments

Zinc was not included in this discussion because levels at which toxic responses are observed are an order of magnitude greater than those associated with the use of ACZA treated wood.

5.5.3 Water column standards

Washington State, in Chapter 173-201A WAC, defines water quality standards for surface waters based on recommendations in U.S. EPA (1998). Table 5.27 lists the criteria for copper and zinc at 50 mg $CaCO_3$/L hardness.

5.5.4 Sediment quality standards for zinc

Washington State has published a marine SQS of 410 mg Zn/kg dw for zinc in WAC 173-204-320. This standard is

Table 5.27 Water quality standards based on U.S. EPA (1998) recommendations at a hardness of 50 mg $CaCO_3$/L.

	µg metal/L			
	Fresh		Marine	
Contaminant	Acute	Chronic	Acute	Chronic
Copper	8.0	5.6	3.1	1.9
Zinc	58.0	52.5	84.6	76.6

based on the metal's AET. Different jurisdictions may develop more, or less, stringent standards depending on a number of factors. For purposes of the risk assessment process described in Chapter 9, the Washington State criteria will be used as a standard for marine sediments. WDOE (2002, 2003) has suggested a freshwater SQS of 140 mg Zn/kg dw and this is recommended as an interim standard until freshwater regulatory SQS are published.

5.6 Copper Naphthenate

The fate and effects of copper were discussed in section 5.2 of this chapter and the following discussion will focus on the fate of naphthenate and the combined effects of copper and naphthenate. Copper naphthenate (CuN) has been used as an industrial biocide since the beginning of the 20th century (Minich and Goll 1948). The active ingredient in CuN is a combination of copper salts and naphthenic acid, which is found in crude oils at concentrations of 0.5% to 2.0%. These acids are known as cyclopentane or cyclohexane carboxylic acids. Naphthenic acids are represented by a family of compounds with typical molecular weights ranging from 210 to 330 grams/mole (Brient et al. 1995). Buchanan and Solomon (1990) reported that CuN is relatively insoluble in water (<1.5 mg/L). For purposes of pressure treating wood for use in freshwater, the preservative is dissolved in oils meeting the requirements of AWPA Standard P-9 (AWPA 1996). Copper naphthenate is an unrestricted pesticide registered by the U.S. EPA for general use in treating wood (40 CFR Section 152.170). The unrestricted status of copper naphthenate means that an applicator's license is not required by end users. For this reason, CuN is typically used to treat field cuts and borings during the construction of treated wood projects. All of the other common wood preservatives (CCA, ACZA, ACQ-B, pentachlorophenol and creosote) are restricted pesticides and end users must have a pesticide applicator's license to apply the preservative but not to use the preserved wood. This unrestricted status is given because

- Copper naphthenate wood preservatives are not Toxicity Category I acute toxicants;
- This preservative does not cause significant subchronic, chronic or delayed toxic effects
- Copper naphthenate does not pose a serious hazard to man or the environment
- Copper naphthenate treated wood is not characterized as a hazardous waste (40 CFR Part 261). This means that CuN treated wood waste is not subject to TCLP testing This greatly simplifies the disposal of CuN treated wood waste.

5.6.1 Fate of copper naphthenate in freshwater environments

Herman et al. (1993, 1994) showed that natural microflora, including *Acinetobacter calcoaceticus, Pseudomonas fluorescens* and *Kurthia* sp., were capable of degrading both commercial mixtures of naphthenic acids and to a lesser extent more complex mixtures of extractable organic acids found in oil sands tailings. The naphthenic acids were rendered non-toxic following 3 d of microbial remediation. Cosmacini (1972) examined the biodegradation of sodium naphthenate in fresh water and reported that initial concentrations of ca. 4.5 mg/L were reduced to concentrations indistinguishable from zero in his published graph within 9 d. Chim et al. (1974) found that naphthenic acids did not inhibit microbial degradation in activated sludge at concentrations less than 500 mg/L. Herman et al. (1993, 1994) reported that indigenous bacterial cultures present in oil sand tailings could utilize commercial mixtures of naphthenic acids as a sole source of carbon converting approximately 50% of the carbon into CO_2. Assuming that the environmental fate of copper naphthenate is similar to that found by Cosmacini (1972) for sodium naphthenate suggests a half-life of approximately 3 d. Because of their slow loss rates from pressure-treated wood (Chapter 7), sediment accumulation of the active ingredients in wood preservatives occurs over periods of years. These time frames are long in comparison with a half-life of 3 d for naphthenate. This assumption leads to the conclusion that the naphthenate component will degrade leaving the copper behind in sediments. The risk assessment provided in Chapter 9 is based on the accumulation of copper in water and sediments.

5.6.2 Toxicity of copper naphthenate in freshwater

Product Chemistry Guideline 63-10: Dissociation Constant, from the U.S. EPA Office of Prevention, Pesticides and Toxic Substances, requires that the active ingredient be sufficiently soluble in water to test for dissociation constants. Because copper naphthenate has such a low dissociation constant, the EPA has not required this data. Based on this information, it is assumed that the migrating preservative will remain in the associated (CuN) form while in the water column. Toxicity data for copper naphthenate dissolved

in a dimethylformamide solvent, developed by the U.S. Army Environmental Hygiene Agency (AEHA 1988), is summarized in Table 5.28. The lowest 96-h LC_{50} was 250 µg CuN/L for rainbow trout (*Oncorhynchus mykiss*). The 96-h NOEC for this species was 56 µg CuN/L. For purposes of this risk assessment, a dissolved CuN benchmark equal to 10% of the lowest 96-h LC_{50} or 25 µg CuN/L will be used. This value is less than half of the NOEC for the compound. This preliminary benchmark is considered conservative because the bioavailability of CuN was significantly increased in the bioassay tests due to being dissolved in dimethylformamide. Copper naphthenate has very low water solubility and is expected to quickly complex with dissolved and/or particulate organic matter in aquatic environments. The anticipated reduction in toxicity associated with these complexes has not been studied and cannot be quantified. Therefore, no allowance for the anticipated reduced toxicity in biologically active environments is included in the benchmark.

5.6.3 Toxicity of sedimented copper naphthenate

No direct information describing the toxicity of sedimented copper naphthenate was available in completing this assessment. However, Herman et al. (1994) studied microbial remediation of oil sands tailings and observed a Microtox EC_{50} of 30% (v/v) for a commercial naphthenic acid (NAS) mixture (100 mg NAS/L).

5.6.4 Water and sediment quality benchmark for assessing CuN

The data provided in Table 5.28 combined with the discussion provided above suggests that CuN is less toxic than uncomplexed copper. Therefore, the water and sediment quality benchmarks described for copper in section 5.2 will be applied in the risk assessment process described in Chapter 9 for copper naphthenate preserved wood.

Table 5.28 Toxicity data for copper naphthenate dissolved in dimethylformamide to a variety of aquatic organisms. All values are in micrograms copper naphthenate per liter (µg CuN/L). Data from AEHA (1988).

		Mean		0.1 x mean LC_{50} or NOEC
Test species	Type test	mg/L	µg/L	
Rainbow trout	96 h LC_{50}	0.25	250	25
Rainbow trout	96 h NOEC	0.056	56	56
Bluegill sunfish	96 h LC_{50}	1.50	1500	150
Bluegill sunfish	96 h NOEC	0.56	560	560
Daphnia magna	48 h LC_{50}	1.10	1100	110
Daphnia magna	48 h NOEC	0.56	560	560

5.7 Pentachlorophenol

Pentachlorophenol or "penta" was described in the scientific literature as early as 1841 and has been commercially produced since 1936. Products using pentachlorophenol as an active ingredient have been used as herbicides, insecticides, fungicides, molluscicides and bactericides (U.S. EPA 1980). In 1994, industry production of wood treated with oil-borne preservatives (primarily pentachlorophenol with some copper naphthenate) was 41,297,000 ft^3 representing 6.5% of the total volume of treated wood produced in the United States. Utility pole preservation accounted for 71% of the total (AWPI 1994). The EPA-approved Consumer Information Sheet (CIS) for pentachlorophenol contains the following permitted and restricted uses:

Permitted uses for penta

1. Ground-contact areas of farm buildings where domestic animals or livestock are unlikely to crib (bite) or lick the wood.
2. Penta-treated wood can be incidentally used in the construction of bridges and docks in direct or indirect contact with either human or livestock drinking water.
3. Penta-treated wood can be used in building interiors (including farm buildings) where the wood is in ground contact providing that two coats of an appropriate sealer are applied.
4. Penta is typically used for the preservation of utility poles, crossarms, bridges, fenceposts, and other uses requiring that the strength of the wood be retained.

Restricted uses of penta

1. Penta-treated logs should not be used in constructing homes.
2. Penta-treated wood should not be used in applications where prolonged contact with the skin may occur such as outdoor furniture unless a sealer is applied.
3. Penta should not be used in areas where domestic animals are likely to crib (bite) or lick the wood. In

addition, Penta should not be used for farrowing or brooding facilities.

4. For animal or human food storage.
5. For cutting-boards or countertops.

Because wood that has been preserved with penta does not become brittle and retains its strength, it can be the material of choice in bridge construction. In addition, aquatic environments can be exposed to pentachlorophenol when preserved utility poles are used to cross streams and wetlands.

5.7.1 Environmental chemistry of pentachlorophenol

Pentachlorophenol (C_6Cl_5OH) has a relative molecular mass of 266.34, a melting point of 190°C, a boiling point of 310°C and a density of 1.98 g/cm^3 at 22°C. The vapor pressure is 0.00415 Pa at 20°C. Pentachlorophenol has a pH dependent octanol-water partition coefficient (log K_{ow}) of 3.3 at neutral pH, 5.1 at pH = 4 and 1.9 at pH = 8 and readily dissolves in most organic solvents. Its solubility in water is also pH dependent and varies between 10 mg/L at pH 6 to 20 mg/L at pH 8 (Mackay et al. 1995).

Penta is not dissociated from its hydroxyl ion in aqueous solutions with pH lower than 5.0. However, as the pH increases, the bioavailability and toxicity of pentachlorophenol decreases because less of the compound is found in the undissociated form. The solubility of penta and potential for adsorption to suspended inorganic particulate matter (particularly clay) is positively correlated with the dissociated fraction which increases above pH = 6.5.

5.7.2 Fate of pentachlorophenol in aquatic environments

Pentachlorophenol may be dissolved in water or sorbed to suspended matter or bottom sediments. In addition, penta is taken up by fauna and flora that metabolize the compound at varying rates. Penta readily degrades in the environment by chemical, microbiological, and photochemical processes (USDA 1980, Eisler 1989). Photochemical degradation is a function of the spectrum and intensity of incident light and appears to proceed rapidly in natural environments with half-lives of 0.15 to 15 d (Smith et al. 1987). Half-life in sediments can range from days to years depending on environmental conditions. The ultimate fate of penta appears to be burial in anaerobic sediments under infrequently encountered conditions or mineralization to carbon dioxide and water.

5.7.2.1 Fate of dissolved penta

Penta that is dissolved in water may be removed by volatilization, photodegradation, absorption, or biodegradation. Penta is subject to rapid photodegradation under laboratory conditions. Boyle et al. (1980) examined the degradation of pentachlorophenol in a two by two array of microcosms that did and did not contain natural lake sediments held under aerobic and anaerobic conditions. At the end of the 131-d experiment, the authors determined the pentachlorophenol half-life under each of the four conditions. Their results are summarized in Table 5.29. Fisher (1990) concluded that the half-life of dissolved and sedimented pentachlorophenol in aerobic and organically rich environments would be on the order of one week. Middaugh et al. (1993) reported that the gram-negative bacterium, *Pseudomonas* sp. (strain SR3) was able to degrade pentachlorophenol and provided an adequate sole carbon source sustaining growth. Nearly complete degradation of 39,000 to 40,000 µg penta/L was accomplished by acclimated *Pseudomonas* sp. Pentachlorophenol half-lives in freshwater streams reported in McAllister et al. (1996) varied between 40 and 120 h.

Boyle et al. (1980) also partitioned the ^{14}C at the end of the experiment. The bulk (99%) of the sedimented ^{14}C was observed in the non-biogenic clay fraction. They also found ^{14}C in algae, floating flocculent material and other biogenic material in the water column. Minimal ^{14}C was observed elsewhere in the microcosms (including the aquarium sides and cover). The authors concluded that pentachlorophenol degradation was positively correlated with incident light levels, pH, oxygen and the presence of sediment. They concluded that pentachlorophenol is likely most persistent in the deoxygenated hypolimnion water of lakes. A number of environmental factors affect the rate at which pentachlorophenol is degraded in natural aquatic environments. The following paragraphs discuss these factors.

5.7.2.2 Effects of pH and water temperature on the degradation of pentachlorophenol

Valo et al. (1985) reported that the metabolism of pentachlorophenol was inhibited at temperatures less than 8°C or greater than 50°C. Optimum degradation occurred at 28°C. Jarvinen and Puhakka (1994) and Jarvinen et al. (1994) found that 99% of the pentachlorophenol present

Table 5.29 Pentachlorophenol remaining in water and sediments at the end of 131 d in each of four aquaria. Residues are mg pentachlorophenol for each compartment. One hundred mg of pentachlorophenol were originally added to the aquaria. The water column half-life also provided.

Test conditions	Total penta in water (mg/L)	Water half-life (days)	Penta in sediments (mg/kg)	Total penta
Aerobic without mud (lighted)	0.95	18.6		0.950
Aerobic with mud (lighted)	0.21	13.9	0.03	0.240
Anaerobic without mud (dark)	16.00	79.8		16.000
Anaerobic with mud (dark)	0.005	12.8	0.04	0.045

in contaminated groundwater was degraded at temperatures of 5 to 10°C. Trevors (1982) documented no penta degradation by acclimated *Pseudomonas* at 0°C. Pentachlorophenol degradation rates at 4°C were dependent on the specific *Pseudomonas* strain used. However, an average of 28.2% of the initial 50,000 µg penta/L substrate was metabolized in 80 d at 4°C while 50.2% of the same concentration was metabolized in 8 d at 20°C. Valo et al. (1985) observed pentachlorophenol degradation at pH values between 5.6 and 8.0. A neutral or slightly acidic pH was found to be optimum. Wong and Crosby (1981) observed pentachlorophenol half-lives of approximately 100 h at pH 3.3 and 3.5 h at pH 7.3 in sterile solutions containing 100,000 µg penta/L. No pentachlorophenol degradation was observed in flasks maintained in the dark. In summary, it appears that pentachlorophenol degradation is optimum at pH values between ca. 6.5 and 8.0 and at temperatures between 10°C and 30°C. These are conditions expected in much of North America during all seasons excepting winter when low temperatures at northern latitudes can be expected to decrease pentachlorophenol degradation rates. In addition, pentachlorophenol is expected to degrade more slowly in areas subjected to low pH.

5.7.2.3 Adaptation to pentachlorophenol by microbial communities

Larsson et al. (1988) and Larsson and Lemkemeier (1989) observed significantly higher degradation of pentachlorophenol by unacclimated microbial communities inhabiting brown water lakes containing high levels of humic acid when compared with clear water lakes. These authors concluded that the microbial communities inhabiting brown water lakes had adapted to the higher phenol levels naturally present in the water and therefore were preacclimated to metabolize pentachlorophenol. McAllister et al. (1996) reviewed the literature pertaining to the microbial degradation of pentachlorophenol. They confirmed that sediment pentachlorophenol concentrations exceeding 300 µg/kg (dw) inhibit microbial degradation until a period of acclimation has passed. Once acclimation had begun, it appears that the higher the initial concentration of PCP, the longer the maximum number of viable cells, capable of degrading PCP, was maintained. Gonzalez and Hu (1991) observed that lag phases of 10 h occurred at 10 mg/L, 30 h at 20 mg/L, 55 h at 44 mg/L, 80 h at 80 mg/L and 200 h at 200 mg/L. It appeared that microbial communities are not generally preadapted to metabolize pentachlorophenol and exposure of naïve communities to penta concentrations as low as 300 µg penta/L can initially reduce their growth. An adaptation period of several h to perhaps 2 wk is necessary for community adaptation to pentachlorophenol. Following this time, adapted communities can tolerate much higher concentrations of pentachlorophenol and rapidly metabolize penta.

5.7.2.4 Effects of additional sources of carbon on pentachlorophenol metabolism

Topp et al. (1988) studied the response of pentachlorophenol-degrading *Flavobacterium* sp. to high levels of penta with and without the addition of sodium glutamate as a cometabolite. They found that the specific activity of penta-degrading cells in the absence of supplementary carbon was 1.51×10^{-13} g penta/cell-hour. They showed that the form and amount of alternate substrates was important in determining the metabolism of pentachlorophenol. For instance, optimal stimulation of pentachlorophenol removal required the addition of 3.0 g sodium glutamate/L. However, glutamate in combination with glucose or cellobiose partially repressed penta metabolism. *Flavobacterium* removed 2.5% of the penta from a 25,000 µg /L initial concentration. The addition of 4 g sodium glutamate/L increased metabolism resulting in the removal

of 61.9% of the penta in 3 h. When the mixture was amended with 4 g sodium glutamate and 5 g/L glucose, penta metabolism was reduced to 15.5% in 3 h. The combination of the two substrates that reduced penta metabolism because in a separate experiment, the authors found that the addition of 0.5 g of glucose to the medium in the absence of sodium glutamate resulted in the complete degradation of an initial 61,000 µg penta /L solution by *Flavobacterium* sp. The amount of penta removed decreased when incremental amounts of sodium glutamate were added to the glucose. Topp et al. (1988) did not discuss the possibility that *Flavobacterium* sp. acclimated to and degraded pentachlorophenol when it represented a sole carbon source—but preferentially shifted to alternate substrates when available. Perhaps rather than acting antagonistically, the combination of sodium glutamate and glucose acted synergistically reducing the dependence of the bacteria on pentachlorophenol. Yu and Ward (1995) observed maximum pentachlorophenol degradation in media supplemented with glucose and peptone. Topp et al. (1988) found that amendment with supplementary source of carbon reduced the lag time required before significant penta metabolism commenced. These authors noted that penta concentrations greater than 20,000 µg/L inhibit *Escherichia coli* and that *Pseudomonas* was inhibited at concentrations above 500,000 µg/L. They also reported that 50,000 µg penta/L was degraded as a sole source of carbon after a lag phase of 90 h.

Pentachlorophenol half-lives in freshwater streams reported in McAllister et al. (1996) varied between 40 and 120 h. Consistent with Liu et al. (1981), McAllister et al. (1996) reported that additional substrates tended to reduce penta degradation rates and hypothesized that adsorbed penta was less bioavailable. McAllister et al. (1996) also noted that the most widely studied pentachlorophenol degrading microorganisms are the pure-culture bacterial strains, *Flavobacterium* spp. and *Rhodococcus chlorophenolicus*. The enzymes responsible for initiating the catabolism of pentachlorophenol by *Flavobacterium* sp. have been isolated and characterized. Furthermore the genes encoding these enzymes have been characterized and cloned into *E. coli,* which then demonstrated the ability to degrade pentachlorophenol. These reports suggest that pentachlorophenol will be degraded more rapidly in organically rich environments. However, some caution is necessary because there is evidence that some combinations of organic substrates appear to result in reduced degradation rates.

5.7.2.5 Effects of water hardness on the fate of dissolved pentachlorophenol

Brockway et al. (1984) studied the fate of pentachlorophenol in static and continuous-flow hard and soft water mesocosms. They observed no significant effect on the fate or effects of penta associated with water hardness.

5.7.3 Fate of sedimented pentachlorophenol

Fisher (1990) constructed microcosms with water at pH 4, 6 and 8 and sediments with 0.0% and 3.0% total organic carbon. She found that more penta was partitioned to the sediments at lower pH than at higher pH. In addition, she reported a positive correlation between sediment TOC and penta concentration. The organisms in the high TOC microcosms accumulated significantly less penta than did those in the 0.0% TOC systems. This work is consistent with Eisler's (1989) observation that pentachlorophenol is fully protonated and lipophilic at low pH, whereas it is ionized and unlikely to adsorb to organic ligands at high pH. Shimizu et al. (1992) reported adsorption coefficients of pentachlorophenol in aquatic environments with varying organic carbon (0.72% to 2.38%) and varying clay content (10.1% to 60.8%). They concluded that at pH values between 6 and 8, the adsorption coefficient was not influenced significantly by organic carbon content (Correlation coefficient = 0.12) but was positively correlated with clay content (Correlation coefficient = 0.94). This work suggests that clay particles (which frequently carry an electrical charge), rather than particulate or dissolved organic carbon form a more likely adsorption nucleus in aquatic environments. It appears that the potential for sedimentation of pentachlorophenol is a complex problem driven by at least the following parameters:

- Water column pH. At reduced pH values, pentachlorophenol tends to be in the fully protonated and more lipophilic. Therefore, it is expected to adsorb to dissolved or particulate organic matter. On the other hand, at higher values of pH, more of the pentachlorophenol is ionized with a higher potential for binding to particulate inorganic matter (silt and clay).
- It appears that more pentachlorophenol will be partitioned from the water column to sediments having increased organic carbon content.

Bryant and Rogers (1990) described the degradation of pentachlorophenol in anaerobic sediments from diverse locations around the world. They observed that dechlorination did not occur for at least the first 15 d of exposure in unadapted sediments. However, following that adaptation period, pentachlorophenol was completely degraded by 33 d. A second addition of 70 mg pentachlorophenol/kg sediment on d 33 was rapidly dechlorinated to about 25 mg/kg in 2 d. In contrast, they observed no biotransformation within a 40-d period of penta added to unadapted Cherokee Pond sediments. The point made in this study is that not all microbes have the ability to dechlorinate penta as a first degradative step. Their study is consistent with other reports indicating that unacclimated, but biological rich sediments, require a period of up to 2 wk for development of suitable microbial communities before aerobic or anaerobic degradation begins. However, when established, these communities rapidly catabolize penta. Interestingly, these authors found no degradation of pentachlorophenol in autoclaved sediments—further emphasizing the microbial nature of the observed degradation. Van Gestel and Ma (1988) determined pentachlorophenol half-lives in low pH and organically rich Holten (pH = 5.6; 6.1% organic matter) and Kooyenburg (pH ~ 5.0; 3.7% organic matter) soils of 23.2 to 47.9 d respectively. There was no significant difference in the half-lives of pentachlorophenol in these two sediments. Smith and Novak (1987) found that penta concentrations as high as 25,000 µg/L in saturated soils were degraded to non-detectable concentrations in less than 3 mo. They found that chlorophenol degradation rates were linearly related to the initial concentration and varied between 100 µg/L-d at 200 µg/L initial concentration to >10,000 µg/L-d at an initial concentration of ca 800,000 µg/L.

5.7.3.1 Effects of sediment oxidation-reduction potential

Delaune et al. (1983) examined the degradation of pentachlorophenol in estuarine sediments following a major accidental spill in a Louisiana Gulf Coast estuary. They completed a series of laboratory experiments in which pH was manipulated between 5.0 and 9.0 and sediment redox potential between -250 mV and +500 mV. They observed maximum degradation at pH = 8.0 with declines at either lower or higher values. Pentachlorophenol was observed to degrade at all values of redox. However, significantly higher degradation rates were observed in aerobic (+250 and +500 mV) than in reducing (0.0 and –250 mV) conditions. A half-life of ca. 24 d was apparent at pH 6.5 and a redox potential of +500 mV. However, at this pH, minimal degradation was observed at any other value of redox. At a pH of 8.0, significant degradation was observed at +250 and +500 mV redox potentials and a half-life of 26.5 d was apparent at +500 mV. The authors found that pentachlorophenol was more tightly bound to oxidized sediment solids than to reduced sediments. Therefore, they concluded that there was a tendency for pentachlorophenol to become preferentially associated with the thin oxidized surface sediment horizon, as well as with suspended colloidal particulates, which would also tend to be oxidized. Under either condition, the penta would be retained in the photic zone of shallow estuaries where the potential for photodegradation is enhanced. The authors concluded that although tidal transport and photodegradation in the water column could play a role in the removal of residual pentachlorophenol from a spill area, their laboratory studies suggested that degradation under either aerobic or anaerobic conditions could account for the disappearance of residual penta left in the immediate vicinity of the spill or that which was transported from the spill site and deposited in adjacent waterbodies.

5.7.3.2 Anaerobic degradation of pentachlorophenol

Guthrie et al. (1984) studied the anaerobic degradation of pentachlorophenol as a component of sewage sludge during treatment. They found methanogenic bacteria were unaffected by pentachlorophenol concentrations <200 µg/L. Acclimation of the bacterial flora to penta raised the inhibition threshold to ca. 600 µg/L. They found that pentachlorophenol was biodegradable anaerobically and that removal was so complete that the soluble concentrations in the Phase II reactors were below detection limits of 5 µg/L. Sorption appeared a minor mechanism of penta removal and volatilization was considered insignificant. The authors concluded that pentachlorophenol undergoes extensive anaerobic biodegradation—especially by acclimated microbial communities. Anaerobic degradation of pentachlorophenol has been confirmed by Kudo (1989). Liu et al. (1981) observed similar results in their comparison of aerobic and anaerobic degradation of sedimented pentachlorophenol at pH = 7.0. They observed an increase in the aerobic half-life of 0.36 d to 190 d in anaerobic conditions. They also found that the inclusion of either sodium chlorophenate or glucose as a second substrate inhibited, rather than enhanced the anaerobic degradation

of pentachlorophenol. In contrast, Valo et al. (1985) found that pentachlorophenol degradation was enhanced by the addition of 0.4 or 40 mM NH_4Cl. Bryant and Rogers (1990) examined the degradation of pentachlorophenol in anaerobic sediments from Georgia, Florida, New York and the Soviet Union. They observed an adaptation period of ca. 15 to 20 d during which little penta degradation occurred. Degradation in anaerobic sediments was rapid with unstated apparent half-lives of 2 to 7 d. The data described in the preceding paragraphs is summarized in Table 5.30.

Data provided in Baker et al. (1980), DeLaune et al. (1983) and Boyle et al. (1980) was interpreted to produce half-lives as a function of initial pentachlorophenol concentration, reduction oxidation potential, pH and temperature (Table 5.31). Sedimented half-life was estimated assuming that microbes require 15 d to adapt to pentachlorophenol and that degradation rates remain linear at all times. This is a small database and the methodology is not precise. However, the results are consistent with the remainder of the literature and appear to reasonably predict sedimented pentachlorophenol half-lives that are important in understanding the accumulation in sediments.

These data were analyzed using linear and non-linear regression analysis. Within the range of the data at hand, sedimented pentachlorophenol half-life was not a function of the initial concentration ($p = 0.10$) or temperature ($p = 0.40$). In addition, the constant term was not significant ($p = 0.42$). Redox potential and pH were significant factors ($p = 0.001$ and $p < 0.000$ respectively). The final regression was highly significant ($P < 0.00015$) and it explained 76% of the variation in the database. The underlying assumptions requiring normally distributed residuals and homoscedasticity were met. The resulting predictive equation is:

$$\text{Sedimented penta half-life} = 18.19448 * \text{pH} - 0.29284 * \text{redox (mV)} \quad [5.3]$$

At a pH of 7.5, Equation [5.3] predicts a pentachlorophenol half-life of 63 d in well-oxygenated (+250 mV) sediments. In reducing sediments (−100 mV), the half-life is significantly increased to 165.7 d. Figure 5.2 provides a graphical representation constructed using a quadratic smoothing routine in the Statistica™ software package. Under normal environmental conditions, the redox potential in surface sediments will vary between −100 and +400 mV and the pH is expected to vary between perhaps 6 and 8.5. Figure 5.2 suggests that under these conditions, sedimented pentachlorophenol half-lives will vary significantly between 12.7 d and 176.4 d. The linear equation used to make predictions in this model predicts a half-life of 24.8 d at pH = 7.8 and +400 mV and 156.6 d at pH = 7.0 and −100 mV.

5.7.4 Case studies describing the fate of pentachlorophenol

Seidler et al. (1986) examined the transport and fate of pentachlorophenol contaminated waste water as it passed through two estuarine ponds being used to grow shrimp in Florida. Pentachlorophenol was added to the test ponds by broadcasting 500 mg of sodium-pentachlorophenate in acetone on day zero followed by the addition of 250 mg on alternate days thereafter to provide a theoretical concentration of 10 µg penta/L. Pentachlorophenol concentrations of 3 to 5 µg/L were observed in the treatment ponds during the first 22 d of the study. At the pond pH of 8.0, the authors predicted that 99.9% of the added penta would be dissolved in the phenolate form, suggesting that photolysis would be the most significant degradation process. They calculated a penta half-life of 2 d for the ponds being treated.

The authors reported that sediments contained 54 times more pentachlorophenol than was found in the water column. However, the data presented in their paper suggest that sediment levels mimicked water column levels with a concentration factor of one to perhaps ten. Sediment concentrations of pentachlorophenol averaged about 5 µg/kg with a maximum of ca. 18 µg/kg. The authors suggested that a chronic influx of pentachlorophenol at a concentration of 10 µg/L resulted in elevated concentrations in the water, sediment, and shrimp. Penta concentrations in the shrimp (maximum of ca. 18 µg/g on d 20) were positively correlated with water column concentrations rather than with sediment concentrations. This suggests direct uptake from the water rather than biomagnification from infaunal and epifaunal prey. Once the source of pentachlorophenol was removed, the compound rapidly disappeared from the water, shrimp and sediments—apparently in a matter of hours or days.

Crossland and Wolff (1985) determined the half-life of pentachlorophenol in outdoor ponds repeatedly dosed to maintain a concentration of 50 to 100 µg penta/L. The authors hypothesized that evaporation, sorption, hydro-

Table 5.30 Summary of pentachlorophenol half-lives, principle reasons for degradation and the effects of various environmental parameters on degradation rates.

a. Reported pentachlorophenol half-life in water

Author	Half-life (d)	Conditions
Boyle et al. (1980)	18.6	Aerobic in the laboratory
Boyle et al. (1980)	79.8	Anaerobic in the laboratory
Crossland and Wolff (1985)	2.0 to 4.7	Outdoor mesocosms
Liu et al. (1981)	0.36	Aerobic in the laboratory
Liu et al. (1981)	190.0	Anaerobic in laboratory
Wong and Crosby (1981)	2.0	pH 7.3 (natural sunlight)
Yu and Ward (1995)	~1.5	Mixed bacterial cultures

b. Effects of pH on pentachlorophenol half-life in water

Author	pH	Half-life (h)	Conditions
Wong and Crosby (1981)	7.3	3.5	Laboratory F40BL lamps
Wong and Crosby (1981)	3.3	100.0	Laboratory F40 BL lamps
Wong and Crosby (1981)	7.3	48.0	Natural sunlight

c. Effects of Ambient temperature on pentachlorophenol half-life

Author	T°C	Half-life (d)	Conditions
Topp et al. (1988)	4	>80	*Pseudomonas* cultures
Topp et al. (1988)	20	<12	*Pseudomonas* cultures

d. Anticipated half-life of pentachlorophenol in soils or sediments

Author	Half-life (d)	Conditions
DeLaune et al. (1983)	24 to 26	Natural estuarine sediments
McAllister et al. (1996)	10 to 70	Flooded soils
McAllister et al. (1996)	< 2 to >5	Freshwater streams
Neary et al. (1990)	Average 30	Southern ecosystems
Smith and Novak (1987)	<5	9 to 13 mg PCP/L unsaturated soil
Smith and Novak (1987)	~15	Average 3 mg PCP/L unsaturated soil

e. Effects of sediment reduction – oxidation potential

Author	Half-life aerobic (d)	Half-life anaerobic (d)
Boyle *et al.* (1980)	13.9	12.8
Bryant and Rogers (1990)		2 to 5
McAllister *et al.* (1996)		144

f. Pentachlorophnol half life in plants and animals.

Author	Species	Penta half-life (h)
Benner and Tjeerdema (1993)	*Atherinops affinis*	52.7
Glickman *et al.* (1977)	*Onchorhynchus mykiss*	6.2 to 6.9

lysis, biodegradation and indirect phototransformation of penta would be of minor importance under the environmental conditions in the ponds. The partition coefficient for penta between water and sediments was predicted to be near unity. The authors used the SOLAR model to calculate a direct photodegradation rate constant for the transformation of penta in the ponds whose pH varied between 7.3 and 10.3 with a mean of 8.3. A series of bioassays were conducted and pond invertebrates were enumerated at levels of taxonomy exceeding Order. The results appeared consistent with the general body of literature describing the toxicity of pentachlorophenol. The observed half-lives of pentachlorophenol in the three treatment ponds varied between 2.0 and 4.7 d and agreed with the predicted half-lives based on results of the SOLAR analysis. The authors concluded that direct phototransformation

Table 5.31 Estimated sedimented pentachlorophenol half-lives as a function of the initial pentachlorophenol concentration, reduction-oxidation potential, pH and temperature. Sedimented penta half-lives were estimated from data in Baker et al. (1980), DeLaune et al. (1983) and Boyle et al. (1980).

Source	Initial penta concentration (µg/kg)	Redox potential (mV)	pH	Temp °C	Half-life (d)
Baker	100.0	250	7.1	0.0	47.5
	100.0	250	6.9	20.0	36.7
Boyle	1400.0	0	4.5	15.0	13.0
	1400.0	250	6.8	15.0	7.5
DeLaune	20.0	500	6.5	33.1	6.0
	20.0	250	6.5	33.1	103.5
	20.0	0	6.5	33.1	178.8
	20.0	500	8.0	33.1	12.0
	20.0	250	8.0	33.1	32.5
	20.0	0	8.0	33.1	81.0
	20.0	–250.0	8.0	33.1	290.0
	20.0	500.0	9.0	33.1	50.0
	20.0	250.0	9.0	33.1	50.0

was responsible for nearly all of the degradation observed in their study. In addition, they noted that at the end of the study, sediment concentrations of pentachlorophenol were very similar to water column concentrations. They hypothesized that an insufficient period of time had elapsed for development of a microbial community capable of efficiently metabolizing pentachlorophenol in the sediments.

Robinson et al. (1983) examined the degradation of pentachlorophenol in a series of experimental ponds contaminated by a single high dose of pentachlorophenol (1,000 µg/L) followed by a series of small doses (0.2, 0.2, 0.4, and 0.4 mg/L) at monthly intervals. Two of the replicated sets of ponds held only phytoplankton whereas the third set of ponds contained rooted macrophytes. The authors found higher metabolism of pentachlorophenol in the ponds containing rooted macrophytes. The increased degradation of penta in the macrophyte ponds resulted in lower body burdens in channel catfish, bluegill and largemouth bass. These fish species survived the highest dose of penta (1,000 µg/L) in the pond containing macrophytes but succumbed in the ponds containing only phytoplankton. The authors suggested three hypotheses

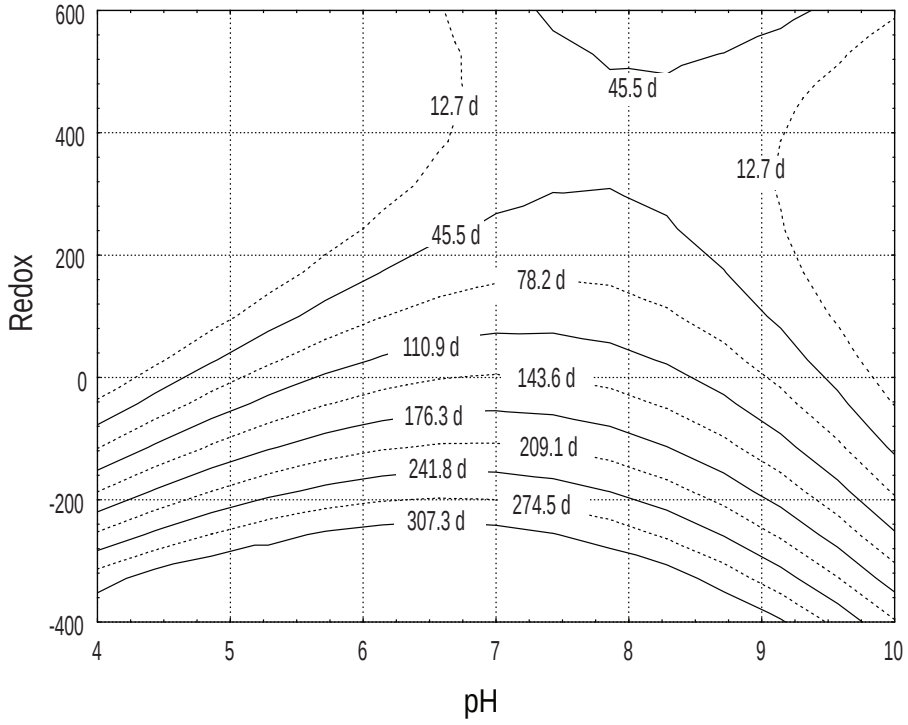

*Figure 5.2 Quadratic solution to the pentachlorophenol half-lives in Table 5.31 with sediment reduction-oxidation potential (mV) and pH as independent factors. 3D Contour Plot (PENTA.STA 22v*126c). z = -715.685+243.79*x-1.042*y-17.252*x*x+0.073*x*y+0.001*y*y*

as possible explanations for these results. However, only two of those hypotheses appear different from each other: (1) the presence of the macrophytes resulted in a different chemical or physical environment in the ponds that increased penta degradation; and (2) the macrophytes or aufwuchs community associated with the macrophytes were incorporating penta as a conjugate within their cell structures.

5.7.5 Metabolism of pentachlorophenol by biota in aquatic environments

Fisher (1990) noted that algae, invertebrates and vertebrates rapidly metabolized pentachlorophenol**.** Biotransformation rates (BTI) were significantly higher in algae, especially at higher pH levels and in association with high TOC sediments. Biotransformation rates in snails and fish varied between 0.52 and 3.88 with no significantly different rates between the two phyla. Kukkonen and Oikari (1988) examined the metabolism of pentachlorophenol in the cladoceran *Daphnia magna* and reported that neonate (<24 h old) and adult daphnia were able to equally metabolize pentachlorophenol and that humic content in the aquaria (DOC up to 23.5 mg/L) had no effect on the metabolic rate. The concentration of pentachlorophenol in these experiments was 20 µg penta/L and the pH was 5.5. The authors did not determine a metabolic half-life. However, their data indicated that the concentration of free penta in the water column declined to 50% of the initial value in ca. 10 h and that 50% of the penta taken up by *Daphnia magna* was metabolized in approximately 24 h. They concluded that the principle metabolic pathway in *D. magna* involved sulfate conjugation. Trujillo et al. (1982) found the half-life of pentachlorophenol in midges to be 4.7 d. However, data in Lydy et al. (1994) suggests that midge tissue concentrations of pentachlorophenol peaked at ca. 24 to 14 h and then declined rapidly to 50% of the maximum in another 24 h. The authors' calculated a half-life of 15 h for pentachlorophenol in the midge.

Glickman et al. (1977) determined pentachlorophenol half-lives in a variety of rainbow trout (*Oncorhynchus mykiss*) tissues. The values varied from 6.2 h in blood to 23.7 h in fat. Pentachlorophenol is lipophilic and these results suggest that there is short-term sequestration in body lipids. The authors found high penta levels in the bile of these fish and concluded that the pentachlorophenol was being conjugated with bile and excreted.

Stehly and Hayton (1989) examined the metabolism of pentachlorophenol in rainbow trout, fathead minnows, sheepshead minnow, firemouth and goldfish exposed to penta for 64 h. They found that penta metabolism was species specific. Consistent with other studies, these authors found that biliary excretion accounted for less than 30% of the total pentachlorophenol metabolites and that 76% of the penta metabolites in bile consisted of pentachlorophenol-sulfate or pentachlorophenol-glucuronide. All of the metabolites excreted into the water were sulfate conjugates while bile was enriched in glucuronide conjugates. Similar results were demonstrated by Cravedi et al. (1995) in Arctic char (*Salvelinus alpinus*) eleutheroembryos (end of yolk sac resorption or 50 to 100 mg wet weight). Test pH was 7.9 and duration was 48 h. They observed that pentachlorophenol-glucuronide accounted for 24.2% of the ^{14}C found in water at the end of 48 h. The parent pentachlorophenol accounted for 29.5% and pentachlorophenylsulfate represented 49.4% of the ^{14}C present in the water column. Benner and Tjeerdema (1993) studied the toxicokinetics and biotransformation of pentachlorophenol in a marine species of fish (*Atherinops affinis*). The fish were exposed to 50 µg penta/L for 24 h to determine the bioconcentration factor, elimination rate constant and the elimination rate half-life. The absorption rate constant was 0.012/h leading to a bioconcentration factor of 278. The elimination rate constant was higher at 0.014/h and an elimination half-life of 52.7 h was determined. During 24 h of exposure to clean seawater, topsmelt depurated 32.9% of the retained pentachlorophenol and residues. Most (64.9%) of the penta was excreted unaltered. However, pentachlorophenol metabolites pentachlorophenylsulfate (18.9%) and pentachloro-β-D-glucuronide (16.2%) were also observed. The authors noted that these same compounds have been identified as intermediates in the metabolism of pentachlorophenol by goldfish, fathead minnows, rainbow trout, firemouths (*Cichlasoma meeki*), sheepshead minnows and striped bass. The authors concluded that topsmelt rapidly absorb and more slowly depurate pentachlorophenol through excretion and/or detoxification by sulfation and glucuronidation.

5.7.6 Bioconcentration, bioaccumulation and biomagnification of pentachlorophenol

Bioconcentration and bioaccumulation of contaminants is of special importance because some aquatic species, most notably bivalves, have demonstrated an ability to rapidly bioconcentrate contaminants in water to high tissue levels. The concern is that persistent contaminants

may move up the food chain, biomagnifying to higher concentrations in each trophic level, until contaminants found at non-toxic levels in the ambient environment reach concentrations where they do cause stress and disease. For effective biomagnification and movement of contaminants through the food chain, several conditions must be met. First, organisms must have the ability to bioconcentrate low levels of contaminants from the water column or their food. Second, these contaminants, or their toxic metabolic intermediates, must be retained, unaltered, in the tissues of the organism until it falls prey to an animal at a higher trophic level. There are a number of factors that mitigate against biomagnification. If contaminants are not absorbed, they cannot accumulate. Numerous organisms, particularly vertebrates, have the ability to either metabolize or excrete organic contaminants. The gut, liver, kidney and gall bladder are common sites of pentachlorophenol concentration, metabolism and excretion in vertebrates. If the contaminants are either rapidly excreted, or they are metabolized to non toxic compounds, then the chain is broken and biomagnification is not effective in passing contaminants through the food chain.

5.7.6.1 Bioconcentration of pentachlorophenol from the water column

The uptake of pentachlorophenol is a function pH (Fisher 1990, Fisher and Wadleigh 1986) and not so much of water concentration. At pH 4.0, for example, penta is fully protonated and therefore highly lipophilic resulting in higher bioconcentration potential. Conversely, penta is completely ionized at pH 9.0 with lower bioconcentration potential and significantly reduced toxicity. In general, Fisher (1990) observed a negative correlation between the uptake of pentachlorophenol by algae, snails or fish and pH. Highest bioconcentration factors were found at pH 4 (BCF = 117.2 to 681.9). Bioconcentration factors at environmentally realistic pH values of 6.0 and 8.0 ranged from 3.8 (algae at pH = 8.0) to 271.1 (fish at pH = 6.0).

Maekelae and Oikari (1990, 1995) determined pentachlorophenol BCFs in adult (55 mm valve length) freshwater mussels (*Anadonta anatina*) of 145 to 342. Tests were conducted at a pH of 6.5 in penta concentrations of 7 and 14 µg/L. However, an equilibrium penta concentration of only 1.8 ± 0.1 µg/L was reached in 4 and 16 h in the two experiments. They found higher penta concentrations in the digestive gland and kidney when compared with whole body soft tissues. In a more recent experiment using (^{14}C)-pentachlorophenol, these same authors (Maekelae and Oikari 1995) found steady state bioconcentration factors averaging 100 in *Anadonta anatina* and 73 in *Pseudanodonta complanata*. The pH in these experiments was 6.5 and the pentachlorophenol concentration 9.7 µg/L. The bioconcentration factors were determined as the body burden of penta (measured in a number of ways) divided by the final concentration of pentachlorophenol. It must be recognized that when determining bioconcentration factors for labile chemicals, such as pentachlorophenol, the average exposure affecting uptake is likely much higher than the final concentration, which tends to inflate the BCF. It would appear that the development of biologically meaningful bioconcentration factors should use some intermediate concentration of the contaminant. An appropriate protocol would necessarily require consideration of the dynamics of degradation in the ambient water integrated over time compared with the depuration and metabolism of the contaminant in the organism.

Niimi and Cho (1983) suggested that pentachlorophenol is rapidly accumulated and eliminated by trout in the natural environment. Uptake from water appeared to be the most important pathway, and any accumulation through food was thought to represent only a minor contribution. Consistent with Glickman et al. (1977) and Rogers et al. (1990), these authors observed the highest penta concentrations in the liver and bile of fish fed penta contaminated feed to satiation for 40 d. Biliary excretion appeared to be a major route of depuration. They noted that the residence time of pentachlorophenol in water was short and suggested that its impact on fish would be most evident in localized areas that receive a continuous input of pentachlorophenol from a point source. Furthermore, Niimi and Cho (1983) concluded that biomagnification of pentachlorophenol is minimal based on the results of their study and the observation that penta concentrations in smelt and alewive, the primary forage species for many Lake Ontario salmonids, were similar to concentrations found in the predators.

5.7.6.2 Bioconcentration of pentachlorophenol from sediments

The ultimate fate of pentachlorophenol deposited in aquatic environments is either decomposition in the water column or sedimentation. Fisher (1990) concluded that, "Thus, for the organic sediment system, bioaccumulation will be determined by interactions between pH, available sorption sites, degree of ionization of PCP and levels of sediment ingestion." Midges (chironomids) are deposit

feeders and are known to rework sediments by feeding and burrowing. Therefore, midges are not only exposed to pentachlorophenol in interstitial water, but they also ingest contaminated particles. Fry and Fisher (1990) compared the bioaccumulation of pentachlorophenol by *Chironomus riparius* allowed to burrow in pentachlorophenol contaminated sediments with similar midges held in suspension directly above the sediments. A third experiment exposed dead midge larvae to contaminated sediments to evaluate the passive uptake of penta. The authors reported BCFs of 229 from water and 7.3 from sediment. The bodies of dead midges exposed to contaminated sediment yielded a BCF of 13.3. Fry and Fisher (1990) noted that Trujillo et al. (1982) found the half-life of pentachlorophenol in midges to be 4.7 d and hypothesized that lack of metabolic degradation in the passive uptake experiment contributed to the higher ultimate pentachlorophenol body burden in the dead chironomids. However, they noted that ionized pentachlorophenol appeared to have a significant affinity for the body wall of the dead larvae and concluded that passive uptake from pore water appeared to be important to the accumulation of pentachlorophenol from sediments. Fry and Fisher (1990) noted that pentachlorophenol has a log K_{ow} of 5.01 and concluded that penta does not behave like a neutral lipophilic compound and therefore its activity and fate were not predictable from its octanol-water partition coefficient. In contrast, Lydy et al. (1994) found that the BCF in *Chironomus riparius* (458) was reasonably well predicted by a much lower octanol-water partition coefficient (K_{ow} = 758) when the water concentration was 9 µg penta/L. Haque and Ebing (1988) determined BCFs from soil of 6.3 for *Allolobophora caliginosa* and 22.2 for *Lumbricus terrestris*. They found that pentachlorophenol was tightly bound to soil and concluded that ingestion of contaminated particles was a significant pathway.

Van-Gestel and Ma (1988) investigated the toxicity and bioaccumulation of pentachlorophenol in the earthworms, *Eisenia fetida andrei* and *Lumbricus rubellus*, in organically rich sandy soils (3.7 to 6.1% organic matter) of low pH (5.0 to 5.6). The LC_{50} values for *Lumbricus rubellus* were 883 mg penta/ kg dw in Holten soil (pH = 5.6 and 6.1% organic matter) and 1,094 mg penta/kg dw in Kooyenburg soil (pH ~ 5.0 and 3.7% organic matter). The differences in these LC_{50} values were not significant. Bioconcentration factors were based on the average of the sediment values on days zero and 14—this appears a more reasonable approach than using the values on the last day as done by Maekelae and Oikari (1990). The BCF based on bulk sediment penta concentration varied between 3.4 and 8.0. In contrast, the BCFs varied between 426 and 996 when based on porewater concentrations, which the authors suggested was consistent with the BCF of 475 observed in fish.

5.7.6.3 Biomagnification of pentachlorophenol from food

Schuytema et al. (1993) fed mealworms contaminated with between 64.8 and 2,604 µg penta/g to African clawed frogs (*Xenopus laevis*) for 27 d. They observed no mortality in the frogs and no significant bioaccumulation of pentachlorophenol. Highest concentrations were found in the liver of frogs. However, these levels were inversely proportional to the level of penta in the mealworms. Penta was found in frog liver at <4.6 µg/g in frogs fed mealworms contaminated to 64.8 µg penta/g, whereas, the liver of frogs fed mealworms with 2,604.6 µg penta/g contained < 0.6 µg penta/g. Niimi and Cho (1983) determined the uptake from food and half-life of pentachlorophenol in rainbow trout (*Oncorhynchus mykiss*). Trout fed diets containing 40 µg penta/kg food attained whole body concentrations of 2 µg/kg over the 3-mo study. Trout fed penta contaminated feeds that were 75 times higher (3,000 µg/kg) accumulated 40 µg penta/kg at the end of 40 d. The body burden declined to 20 µg/kg by the end of the study. The biological half-life of penta in trout was estimated to be approximately 7 d. This observation is consistent with the literature describing the depuration and metabolism of pentachlorophenol in vertebrates and invertebrates. It appears that penta clearance in all tested organisms is fast enough to minimize any potential for biomagnification in food chains.

5.7.7 Pentachlorophenol toxicity in aquatic environments

Pentachlorophenol is known to uncouple oxidative phosphorylation, inhibiting ATP pathways important to respiration in both animal and plant cells. In addition, Moreland and Hilton ((1976) described penta as a more general inhibitory uncoupler, suggesting that it has several sites of action, including photophosphorylation, protein synthesis and lipid biosynthesis (Morrod 1976). All of the mechanisms of penta's toxicity have not been precisely defined, but may generally involve the disruption of cellular membranes (Jayaweera et al. 1982, Senger and Ruhl 1980, Smejtek et al. 1983).

5.7.7.1 Acute toxicity of pentachlorophenol in aquatic environments

Penta interferes with oxidative phosphorylation by uncoupling the production of adenosine triphosphate from adenosine diphosphate. Because this process provides the energy source for cellular metabolism, pentachlorophenol is a broad-spectrum biocide. Eisler (1989) summarized 96-h LC_{50} penta concentrations for aquatic organisms. For most freshwater species, the 96-h LC_{50} varied between 100 and 2,000 µg penta/L. In general, Eisler's (1989) data suggest that freshwater vertebrates (fish) are more sensitive than invertebrates. Table 5.32 summarizes the lower LC_{50} values provided by Eisler (1989) for salmonids and centrarchids. Salmonids of the genus *Oncorhynchus* appear most sensitive. In contrast, invertebrate LC_{50} values are typically above 100 µg/L.

5.7.7.2 Chronic toxicity of pentachlorophenol to aquatic organisms

ENVIRON (1996) summarized a number of chronic endpoints for aquatic fauna and flora exposed to pentachlorophenol. Their data is further summarized to include only those data that included test pH in Table 5.33. The U.S. EPA freshwater chronic standard for pentachlorophenol is included for comparison in the table.

Roszell and Anderson (1994) examined the effect of pentachlorophenol on non-specific immune function in two phagocytic cell populations isolated from the estuarine fish, *Fundulus heteroclitus*. They found that phagocytosis of yeast particles was significantly inhibited at penta concentrations greater than 5,000 µg/L. A comparison of phagocytic response between controls and a pentachlorophenol level of 1,000 µg/L did not reveal significant differences. Brown et al. (1985) observed a reduced number of feeding acts in the young of largemouth bass exposed to pentachlorophenol concentrations of 67 and 88 µg/L but not at concentrations less than 67 µg/L. Endpoints measured included the number of feeding attempts, and the numbers of misses and mistakes. The No Observed Effect Level was 45 µg penta/L. Hardness (65 mg/L as $CaCO_3$) and pH (7.7) were not measured directly in this experiment, but were assumed equal to that observed in a 1985 study using the same water supply.

Table 5.32 Acute toxicity of freshwater fish to pentachlorophenol (96-h LC_{50}) reported in Eisler (1989).

Species	Concentration (µg penta/L)
Oncorhynchus mykiss (rainbow trout)	34 to 121
Oncorhynchus nerka (sockeye salmon)	63 to 68
Oncorhynchus tshawytscha (chinook salmon)	68 to 78
Salmo salar (Atlantic salmon)	500
Salvelinus fontinalis (brook trout)	128
Lepomis macrochirus (bluegill)	120 to 350
Micropterus salmoides (largemouth bass)	136 to 287

Keller (1993) noted that in 1993 there were over 40 species of freshwater unionid mussels listed as endangered or threatened under the Endangered Species Act (PL 100-707). Keller determined a 48-h LC_{50} value in juvenile *Anadonta imbecilis* at pH 7.0 and compared the results with concurrent bioassays on *Daphnia magna* and *Lepomis macrochirus*. His results indicated that the 48-h LC_{50} for the juvenile mussels (610 µg penta/L) was greater than the 48-h LC_{50} for the daphnid (330 µg/L) or the 96-h LC_{50} for bluegills (240 µg/L). At pH = 7.0, the U.S. EPA chronic pentachlorophenol water quality standard is 5.73 µg/L, giving a safety factor of 106.

5.7.7.3 Carcinogenicity

Jorens and Schepens (1993) noted that pentachlorophenol is not classified as a human carcinogen, but suggested that historical contaminants in commercial penta products, such as chlorodibenzo-p-dioxins are known carcinogens. The U.S. EPA has classified pentachlorophenol as a B2 carcinogen (i.e., no evidence of carcinogenic response in humans, but the evidence in animals is sufficient to cause the compound to be suspect). Likewise, Health Canada (1994) has classified penta as a Group III.B compound that is possibly carcinogenic to humans.

5.7.7.4 Mutagenicity and teratogenic effects

Venegas et al. (1993) examined the teratogenic effects of pentachlorophenol in an amphibian (*Caudiverbera caudiverbera*). They examined embryonic development in water containing 15, 150, 300 and 1,500 µg/L pentachlorophenol. The results of the Micronucleus Test on premetamorphic larvae indicated higher micronucleus formation rates in the controls than in the various penta treatments. Chromosomal aberration tests in anaphase and telophase provided a second endpoint. The authors concluded that the highest penta dose resulted in inhibition and delays in development of normal growth of embryos of this amphibian. However, the study found no clear mutagenic effects. CCME (1997) concluded that while chlorophenols

Table 5.33 No observed effects level (NOEL) and lowest observed effects level (LOEL) associated with pentachlorophenol in freshwater environments. All pentachlorophenol values are in µg penta/L. Data from ENVIRON (1996).

			µg penta/L				
Endpoint	Duration (h)	Species	NOEL	LOEL	pH	Temperature (°C)	EPA*
Reproduction; number viable eggs	16	*Lymnaea stagnalis* (snail)	50	NR	8.0	18.3	46.5
Larval survival and reproduction	10–28	American flagfish	55	102	6.95	25	5.4
Biomass and mortality, eggs @ 10°C; alevins @ 15°C; fry @ 20°C	>28	*Oncorhynchus mykiss*	10.9	25	8.0	10–20	15.6
Hatchability, survival and growth	32	*Pimephales promelas*	16.5	34.6	6.5	25	3.5
Survival and growth (fry and juveniles)	90	*Pimephales promelas*	6	13	7.4	25	8.6
Survival and growth (fry and juveniles)	90	*Pimephales promelas*	36	85	7.4	25	8.6
Survival and growth (fry and juveniles)	90	*Pimephales promelas*	>130	>130	9.4	25	63.9
Early life stage hatchability, survival, and growth	32	*Pimephales promelas*	44.9	73	7.55	25	10.0
Hatchability survival and growth	32	*Pimephales promelas*	63.7	125	8.5	25	25.9
Hatchability survival and growth	32	*Pimephales promelas*	27.6	58.2	7.5		9.5
Hatchability survival and growth	32	*Pimephales promelas*	32	75	8.0		15.6
Growth	56	*Chaetogammarus marinus*	100	NR	8.0	NR	15.6
Number of viable oocytes	18	*Oncorhynchus mykiss*	11	19	7.4	12	8.6
Number of viable oocytes	18	*Oncorhynchus mykiss*	12	22	7.5	12.5	9.5
Reproduction	21	*Daphnia magna*	180	320	8.0	20	15.6
Survival and reproduction	7	*Daphnia magna*	100	500	8	20	15.6
Inhibition of cell growth	5	*Skeletonema costatum*	11	20	8.1	19–22	17.3
Inhibition of cell growth	5	*Selenastrum capricornutum*	12	17	7.5	24–25	9.5
Inhibited cell growth	5	*Anabaena flos-aquae*	7.8	18	7.5	24–25	9.5
Growth, reduction; biomass	21	*Elodea canadensis*	230	380	7.95	22	14.9
Inhibited cell growth	5	*Inavicula pelliculosa*	40	77	7.5	25	9.5
Inhibited cell growth	5	*Anabaena flos-aquae*	7.8	18	7.50	24–25	9.5
Frond density and biomass	14	*Lemna gibba*	32	72	5.0	23–27	0.8

* EPA chronic standard.

may have reproductive and fetotoxic effects, they do not appear to be teratogenic or mutagenic.

5.7.7.5 Effects of temperature on pentachlorophenol toxicity

Fisher and Wadleigh (1986) examined the toxicity of pentachlorophenol to the midge *Chironomus riparius* at 15, 25 and 35°C. The endpoint they examined was a flight response following stimulation with a pair of forceps during exposure to PCP in soft water at pH 7.0. Fisher observed that EC_{50} values increased from 1,176 µg/L at 15°C to 1,556 at 25°C and then declined to 631 µg PCP/L at 35°C. They concluded that midge metabolism was increased at the higher temperatures and that penta's interference with respiration (phosphorylation of ADP to ATP) at the higher metabolic rate was responsible for the increased EC_{50} at 35°C. Similar results were reported by Fisher (1991) in an experiment conducted at pH values of 4, 6 and 8. The EC_{50} values for penta in this study varied between 253 µg/L at a pH of 4 and temperature of 35°C to 2,052 µg penta/L at pH 8 and 25°C. Eisler (1989) cites similar results from Hedtke et al. (1986) and Hedtke and Arthur 1985) who reported positive correlations between EC_{50} and temperature for fathead minnows (*Pimephales promelas*), the isopod *Asellus racovitzai* and the snail *Physa gyrina*. These reports indicate that the toxicity of pentachlorophenol to aquatic species increases with increasing temperature. Similar increases in the toxicity of pentachlorophenol to *Notopterus notopterus* with increasing temperature (16°C, 23°C, and 36°C) were observed by Gupta et al. (1983).

5.7.7.6 Effects of pH on pentachlorophenol toxicity

Fisher (1991) reported that the toxicity of penta-chlorophenol is inversely related to pH. The effective concentration (EC_{50}) of pentachlorophenol resulting in failure of *Chironomus riparius* to execute an appropriate flight response increased from 384 µg/L at 25°C and pH = 4 to 2,052 µg penta/L at the same temperature and pH = 8.0. Smith et al. (1987) examined the toxicity of pentachlorophenol to *Selenastrum capricornutum* and found that culture media equilibrium pH and 96-h EC_{50} were very strongly correlated (r = 1.00) between pH values of 7.3 and 8.5. The authors concluded that the toxicity of pentachlorophenol is due primarily to the concentration of the undissociated compound. The dependence of pentachlorophenol toxicity on pH was further elucidated by Spehar et al. (1985) in fish and amphipods. They found that acute exposures in all three species showed that pentachlorophenol toxicity was decreased with increasing pH. Kaila and Saarikoski (1977) observed a similar response in the crayfish (*Astacus fluviatilis*). The 8-d LC_{50} decreased from 53 mg/L at pH 7.5 to 9 mg penta/L at pH 6.5.

Stehly and Hayton (1990) reported reduced uptake and clearance of penta in goldfish (*Carassius auratus*) with increasing pH in the range 7.0 to 9.0. They concluded that pH-related changes in the pharmacokinetics of penta resulted in a decrease in BCF with increasing pH and suggested that this could account for both the decreased capacity of the fish to accumulate penta and its reduced toxicity at higher pH values. Early life stage exposures of fathead minnows showed that chronic penta toxicity and bioaccumulation were similarly decreased when pH values were increased. They developed a relationship describing bioconcentration as a function of pH. Reported BCFs ranged from 1,066 at pH 6.5 to 281 at pH 8.5. The authors concluded that the decrease in chronic pentachlorophenol toxicity appeared to be due to reduced bioaccumulation and toxicity as a direct result of the increased dissociation at higher pH values (Equation [5.4]).

$$\text{Fathead minnow bioconcentration factor} = 10^{(4.80 - 0.28*\text{pH})} \quad (R^2 = 0.94) \qquad [5.4]$$

5.7.7.7 Effects of water hardness on pentachlorophenol toxicity

Inglis and Davis (1972) examined the effects of water hardness (13.0, 52.2, 208.7 and 365.2 mg $CaCO_3$/L) on six species of fish, including rainbow trout. Reported values of pH ranged from 7.8 to 8.0. The authors concluded that water hardness had no significant effect on pentachlorophenol toxicity to any of the tested species.

5.7.7.8 Effects of dissolved organic carbon on the toxicity of pentachlorophenol

Lee et al. (1993) examined the acute toxicity of pentachlorophenol to zebrafish (*Brachydanio rerio*) and the cladoceran, *Daphnia magna*, at total organic carbon concentrations varying between 0.0 and 50 mg/L. They observed no significant difference in the 96-h EC_{50} (zebrafish bioassay) or 48-h EC_{50} (*Daphnia magna*) as a function of TOC at any of the tested values.

5.7.7.9 Pentachlorophenol toxicity to aquatic plants

Smith et al. (1987) investigated the toxicity of pentachlorophenol to *Selenastrum capricornutum* as a function of pH and found that the 96-h EC_{50} was given by this relationship (Equation [5.5]):

$$\text{96-h } EC_{50} \text{ (}\textit{Selenastrum capricornutum}\text{)} = \exp^{(0.847*\text{pH} - 4.28)} \qquad [5.5]$$

The regression coefficients in this relationship are similar to the current U.S. EPA pentachlorophenol standard and demonstrates reduced toxicity at elevated pH where more pentachlorophenol is in the dissociated form (i.e., having lost the OH radical). The authors suggest that the toxicity of pentachlorophenol is primarily associated with the undissociated compound.

5.7.8 Potential for contamination of groundwater by pentachlorophenol

Goerlitz et al. (1985) examined the potential for pentachlorophenol contamination of groundwater at a wood preserving facility near Pensacola, Florida. They did not find pentachlorophenol in groundwater and concluded that the low solubility of PCP at the groundwater pH of 6.0 was responsible. LaFrance et al. (1994) examined the adsorption and mobility of pentachlorophenol as a function of dissolved organic carbon and pH and concluded that dissolved organic matter does not promote nor diminish pentachlorophenol adsorption and transport in the soil column under conditions representative of an environmentally relevant groundwater-soil system exposed to penta concentrations less than 100 µg/L. At high

penta concentrations ~ 800 µg/L, dissolved organic matter may increase the adsorption of penta. Warith (1993) and Warith et al. (1993) examined the mobility of pentachlorophenol in soils containing high levels of total organic carbon (f_{oc} = 16%). Similar soils might be found in a wetland or in an organically rich estuarine environment. Based on their data, the authors concluded that the migration potential of pentachlorophenol in the environment is minimal. In summary, it appears that penta is strongly sorbed to soil; hence, leaching through the soil profile and contamination of groundwater is unlikely. The physicochemical properties of pentachlorophenol and these reports indicated that there is little potential for the contamination of groundwater associated with the use of pentachlorophenol treated wood.

5.7.9 Regulatory criteria for pentachlorophenol in aquatic environments

Section 304 (a) (1) of the Clean Water Act (33 U.S.C. 1314 (a) (1)) required the U.S. Environmental Protection Agency to publish and update ambient water quality criteria. These criteria reflect the latest scientific knowledge of the identifiable effects on the health and welfare of aquatic resources including fauna, flora and human uses. The biological effects review provided herein is consistent with EPA (1980, 1998) in predicting that pentachlorophenol's acute and chronic toxicity are inversely correlated with water pH and dissolved oxygen and directly correlated with temperature. At pH = 6.5, the EPA found that acute values ranged from 4.4 to > 43,920 µg penta/L. Chronic values ranged from <1.835 to 79.66 µg penta/L. The mean acute-chronic ratios ranged from 0.89 to 15.79 µg/L. Freshwater algae were affected by concentrations as low as 7.5 µg/L, whereas vascular plants were adversely affected at 189 µg/L and above. Bioconcentration factors ranged from 7.3 to 1,066 in three species of fish. Procedures described in the "Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and their Uses" indicate that excepting where a locally important species is highly sensitive to pentachlorophenol, freshwater aquatic organisms and their uses should not be affected unacceptably if the average short term concentrations (µg penta/L) do not exceed the numerical values given by Equations [5.6] and [5.7]:

$$\text{Acute criterion: 1-h average concentration} < \exp(1.005*\text{pH} - 4.830)\ (\mu g/L) \quad [5.6]$$

$$\text{Chronic criterion: 4-d average (once every 3 y)} < \exp(1.005*\text{pH} - 5.290)\ (\mu g/L) \quad [5.7]$$

It should be noted that these are not appropriate concentrations for continuous exposure and they are not No Observed Effect Levels or Threshold Effects Levels. For instance, at a pH of 6.8, the chronic criterion for penta is 4.68 µg/L. At this pH, the EPA (1986) notes that a pentachlorophenol concentration of 1.74 µg penta/L caused a 50% reduction in the growth of yearling sockeye salmon in a 56-d test. This seems inconsistent with the chronic EPA criterion. However, it is important to remember that the EPA criterion is for a maximum 4-d exposure—not for a 56-d exposure. These criteria are not rules and they do not have regulatory impact. However, a number of states have used EPA (1986) as the basis for setting regulatory standards for potentially toxic compounds, including pentachlorophenol: Washington State (WAC 173-201), Texas (30 TAC 307.6), Utah (UAC R317-2-14), Florida (FAC 62-302.530), Indiana (327 IAC 2-1-6) and Oregon (OAR, 340 41). Different criteria have been proposed or adopted by other jurisdictions. The data for continuously distributed criteria provided in ENVIRON (1996) were subjected to non-linear regression analysis. The results are presented in Table 5.34 and Figure 5.3.

The Draft 1995 CCME/BC criteria and the 1995 Ontario guidelines do not adequately reflect the effect of pH on the toxicity of pentachlorophenol. These criteria are either under protective at low pH or over protective at high pH. The 1994 Aquatic Dialogue Group Level of Concern Approach (see Environ 1996 for a review) produces two sets of criteria. The lower levels are sufficient to protect 99% of the species and the upper values are sufficient to protect 95% of the species. These are continuous criteria and therefore it is inappropriate to compare their recommendations with those of the 1- h and 96 h EPA criteria.

In response to recommendations made by a US EPA task force in 1992, the Aquatic Risk Assessment and Mitigation Dialogue Group (ADG) was sponsored by the EPA and the North American Chemicals Association in 1993. The ADG included representatives of EPA, agrochemical companies, academia, and environmental and agricultural interest groups. The Society of Environmental Toxicology and Chemistry (SETAC) acted as a facilitator for meetings of the group and published a final report with recommendations (SETAC 1994). The ADG recommended an "integrated probabilistic risk assessment approach that

included both the probability of exposure and effects." ENVIRON (1996) applied the ADG methodology to derive pentachlorophenol chronic toxicity guidelines sufficient to protect 90%, 95% and 99% of species. The results are included in Table 5.34 and in Figure 5.3 for the protection of 95% and 99% of species.

5.7.9.1 Summary

The risk assessment presented in Chapter 9 will use the US EPA chronic criteria as a benchmark against which to compare levels of pentachlorophenol migrating into the water column during the first 4 d. The 1994 Aquatic Dialogue Group Level of Concern necessary to protect 99% of species will be used as a second benchmark against which to assess long term water column concentrations of pentachlorophenol within a few cm of the immersed wood on days ≥ 5.0 following immersion of the treated wood.

5.7.9.2 Pentachlorophenol contamination of drinking water supplies

Table 5.35 lists limits for pentachlorophenol in drinking water established by a number of jurisdictions. It appears that most jurisdictions have invoked a maximum continu-

Table 5.34 Comparison of various jurisdictional criteria for pentachlorophenol. The EPA and Great Lakes chronic criteria are for four day exposures. Others listed below are continuous criteria. All values are in µg/L.

Jurisdiction	Criterion	Basis for criterion
CCME (1997)	0.02 at pH 6.5; 0.1 at pH = 7.0 or 7.5; 0.30 at pH ≥ 8.0	Extrapolation from acute guppy data; ACR = 24.39; safety factor = 0.1
ENVIRON (1996)	Chronic criterion = exp(1.002*pH – 7.90)	Application of 0.1 safety factor to lowest chronic LOEC in database
Ontario 1995	0.5 at all values of pH	Application factor of 0.01 applied to the lowest mean species-specific acute LC_{50}
U.S. EPA (1986)	4-d chronic = exp(1.004*pH – 5.28)	Statistical approach using all relevant acute toxicity data; set to protect 95% of species; ACR = 3.166
Great Lakes Water Quality Initiative (GLWQI 1995) in EPA (1996)	4-d chronic = exp(1.004*pH – 5.12)	Similar to US EPA approach except the ACR = 2.608
1994 Aquatic Dialogue Group Level of Concern	Continuous chronic = exp(1.005*pH – 7.26)	Graphical approach using entire database of primary chronic NOECs

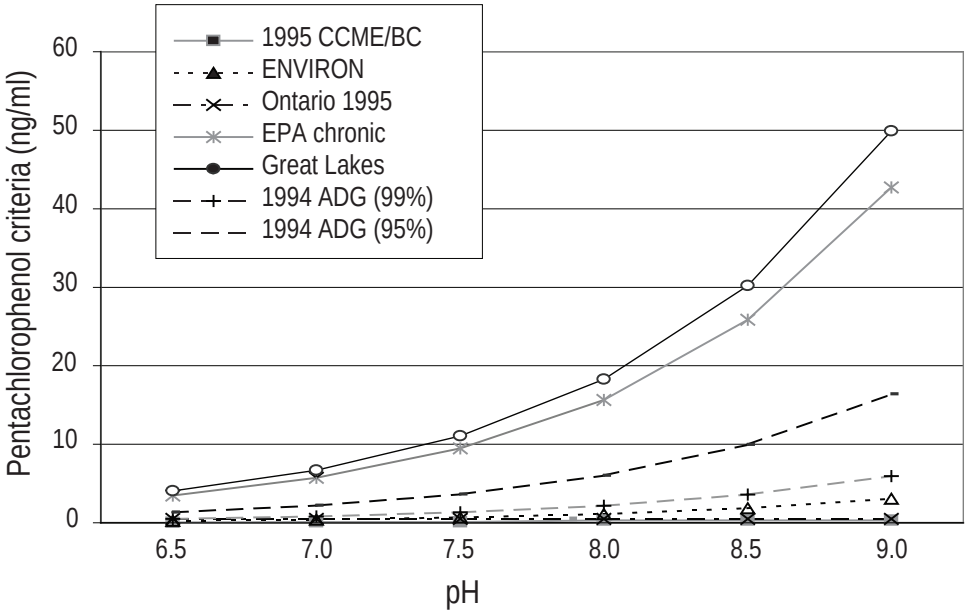

Figure 5.3 Graphical representation of various jurisdictional criteria defining allowable concentrations of pentachlorophenol in freshwater as a function of average pH.

Table 5.35 Regulatory limits for pentachlorophenol concentrations in drinking water and/or groundwater.

Authority	Maximum permissible level (µg penta/L)
U.S. EPA maximum contaminant level (MCL)	1.0
Australia and New Zealand	10.0
Florida (FAC 17-302.530)	1.0; not to exceed an annual average of 0.28µg/L
Indiana (327 IAC 8-2-5)	1.0
Oregon (OAR Chapter 340, Division 41, DEQ)	1.0
Wyoming (WCWR 020-080-018 Appendix A	1.0

ous pentachlorophenol concentration of 1.0 µg/L in drinking water. This value will be used in assessing the appropriateness of pentachlorophenol structures proposed for construction in drinking water supplies.

5.7.10 Sediment quality benchmarks

Washington State (Washington Administrative Code, Chapter 173-204-320) has developed an AET-based pentachlorophenol standard for marine sediments of 360 µg penta/kg dw. The AET is the lowest concentration of a chemical above which adverse effects are always observed in Puget Sound Sediments. Adverse effects are determined from laboratory bioassays on a variety of test animals and on the paired analysis of infaunal communities and sediment chemistry. These standards are considered sufficient to protect most marine organisms. The New York State Department of Environmental Conservation (NYSDEC 1993) has established a freshwater sediment criterion for pentachlorophenol of 100 µg penta/g sedimented organic carbon for acute toxicity and 40 µg penta/g sedimented organic carbon to prevent chronic toxicity. These criteria are based on an equilibrium partitioning model and state water quality criteria for pentachlorophenol. They are intended for use in screening contaminated sediments. The NYSDEC criteria are expressed as a concentration in sediment by prescribing an organic carbon content. For example, in a sediment with 1% organic carbon, the corresponding chronic criterion value is 400 µg penta/kg dry sediment (40 µg/g TOC x 0.01 TOC = 0.4 µg/g or 400 µg/kg), or 800 µg/kg at 2% TOC and 1,200 µg/kg at 3% TOC, etc.).

The Washington State marine standard of 360 µg penta/kg dry sediment weight will be used as benchmark in assessing marine environmental risks associated with the use of pentachlorophenol treated wood and the NYSDEC criteria will be used for freshwater sediments.

5.8 Didecyldimethylammonium Chloride

Didecyldimethylammonium chloride (DDAC) is a member of the quaternary ammonium compounds (QAC). Quaternary ammonium compoundswere first synthesized in the late 1800s and their bactericidal properties were reported two decades later. These compounds are well known for their germicidal, fungicidal, and algicidal properties when the alkyl fractions contain fewer than 8 to 14 carbon atoms. Formulations containing between 0.01 and 1.0% QACs are used extensively as antiseptics, bactericides, fungicides, sanitizers and deodorants (Gosselin et al. 1984). Quaternary ammonia compounds (QACs) with carbon chain lengths exceeding 14 are used extensively as softeners in laundry applications. DDAC does not occur naturally and its presence in aquatic environments results from spills and wastewater or stormwater discharges. Huber (1984) reported that 39,000 tons of QACs were marketed in the USA in 1978 and he measured QAC concentrations of 5 to 20 µg/L in the Main River of West Germany. The point is that this general class of compounds is used extensively in modern society and they are finding their way into aquatic ecosystems in detectable quantities. Therefore, their ecological effects are of interest and they require management to insure that adverse effects are not associated with their use.

5.8.1 Fate of DDAC in aquatic environments

Didecyldimethylammonium chloride is a nonvolatile, photolytically stable salt, which is highly soluble in water (Henderson 1992). Its octanol/water partition coefficient is near zero (Huber 1984, Henderson 1992). Brooks et al. (1996) reviewed the available literature and reported the properties given in Table 5.36.

Table 5.36 Summary of environmental fate properties of didecyldimethylammonium chloride (DDAC).

Photolysis	Solubility in water	Hydrolysis	Sediment half-life (complexed with clay)	Half-life in aerobic sediments	Soil sorption
No	High	No	45.6 days	11.2 days	Strong

Based on these properties, the environmental fate of QACs, including DDAC, is likely adsorption to suspended inorganic material, most likely clay and subsequent sedimentation (Fernandez et al. 1991, Huber 1984). Daly (1989) examined the adsorption/desorption of DDAC at 25°C in the dark in sand, sandy loam, silty clay loam, and silt loam at 0.25, 0.90, 2.05 and 2.1% organic carbon, respectively. The study was conducted at a 1:200 soil to water ratio using four nominal concentrations of 0.70, 3.50, 5.25 and 7.00 mg/L DDAC. The study concluded that DDAC is essentially immobile in soil. Adsorption/desorption mechanics and the effects of pH, interstitial water hardness, salinity, etc. were not addressed. While DDAC appears stable under the test conditions, it is not possible to determine from this paper, the potential for desorption and recycling back into the water column. The high water solubility and low K_{ow} associated with DDAC suggest that this may occur under some physico-chemical conditions. Huber (1984) described the ionic binding of cationic QACs (including DDAC) as irreversible and it appears reasonable to conclude that DDAC is not easily recycled back into the water column in a dissolved form following adsorption to clay and sedimentation.

The literature describing the degradation of QACs and DDAC, in particular, is equivocal. It should be remembered that QACs with relatively short carbon chains (<14) are commonly used as bactericides. Above threshold concentrations toxic to bacteria, these compounds may adversely affect aquatic microflora that are important factors in their degradation. Biodegradation is expected to be the main route of dissipation of DDAC in the environment (Agriculture Canada et al. 1988). Gawel and Huddleston (1972) reported complete degradation of DDAC within 48 h at concentrations of 10 mg/L by mixed bacterial cultures obtained from soil and sewage. Gerike and Gode (1990) found significant degradation at 5 mg/kg DDAC but poor ultimate degradation at 15 mg/kg. In contrast, Cranor (1991) observed little degradation of ^{14}C dosed DDAC at 10 mg/kg added to aerobic soils and monitored for one year. At the end of 365 d, 72.9% of the dosed radioactivity remained as parent compound and the estimated anaerobic half-life was 1,048 d. Similar studies reported by Cranor (1991) estimated an aerobic aquatic half-life of 8,365 d and an anaerobic aquatic (microbially active water and sediment dosed with 10 mg/kg DDAC) half-life of 6,218 d. The refractory nature of QACs in anaerobic conditions is further supported by Huber (1984). In contrast, Wildlife International (1996) reported a half-life of 11.2 d in aerobic sediments obtained from three sites on the Saint Clair River in Canada. When DDAC was complexed with clay, the half-life increased to 45.6 d. Taken altogether, this review suggests that the sedimented half-life of DDAC varies with its concentration in sediments, availability of oxygen and the resident microbial community. No information was obtained as to whether or not DDAC provided a suitable sole carbon source for some species of bacteria, or if it was catabolized only in the presence of other organic compounds. Some refractory compounds, like high molecular weight PAH, are not efficiently catabolized in the absence of more labile and complex carbon substrates. If this is true, then the half-life of DDAC would vary with the amount of sedimented TOC. However, the Wildlife International (1996) study used unamended, natural sediments, and the observed half-life of between 11.2 and 45.6 d is likely an appropriate value—at least at sediment concentratons <10 mg DDAC/kg.

DDAC's strong affinity for soils suggests that it will adsorb to clay particles and be sedimented. Based on this discussion, it appears that DDAC is stable in sediments at concentrations exceeding (10 mg/kg), but is degraded with a half-life of 11.2 to 45.6 d at lower concentrations. Additionally, it appears that DDAC degradation is dependent on positive redox potentials and longer half-lives can be expected in anaerobic conditions or where microflora are compromised.

5.8.2 Biological effects of DDAC in aquatic environments

5.8.2.1 DDAC bioconcentration

Huber (1984) concluded that DDAC (or other QACs) are not significantly bioconcentrated with BCFs of 5 to 32 for related QACs. Henderson (1992) reported a whole body BCF of 81 for DDAC in the bluegill sunfish (*Lepomis mac-*

rochirus) and concluded that because DDAC is highly ionic, it is not expected to easily cross the gastrointestinal epithelium. The results of excretion studies in rats support this conclusion. Following oral dosing with ^{14}C-DDAC, 89% to 99% of the radioactivity was found in the feces and less than 2.5% in the urine. Absorbed DDAC was metabolized by oxidation of the decyl side chain to a variety of oxidative products. Evidence seemed to favor initial hydroxylation of the carbon next to the terminal carbon, followed by formation of a hydroxyketone. The four major metabolites found in their study were more polar and presumed to be less toxic than the parent compound, although the specific chemical structures were not determined. Depuration experiments reported by Henderson (1992) indicated that 67% of the DDAC accumulated in whole fish tissues was depurated within 14 d. The available literature suggests that QACs, including DDAC do not significantly bioconcentrate in aquatic organisms.

5.8.2.2 DDAC biomagnification

No direct evidence examining this question was reviewed. However, based on the low observed bioconcentration factors, rapid depuration and observed metabolism, biomagnification through food chains appears unlikely.

5.8.2.3 Acute toxicity associated with DDAC in freshwater environments

Brooks et al. (1996) conducted a review of the toxicological database for DDAC and reported the LC_{50} values summarized in Table 5.37. It should be noted that the sturgeon bioassay conducted by Farrell used yolk-sac and 30–40-d-old larvae. Anecdotal evidence provided by Dr. Gerald Schoenig (personal communication) suggested that Dr. Farrel's sturgeon larvae were the survivors of two highly stressful events occurring just prior to the bioassays. These stresses were created during shipping in which half the larvae were lost and by the inadvertent introduction of lethal quantities of chlorine into the holding tank the day prior to the bioassays resulting in the loss of an additional 50% of the larvae. Subsequent laboratory bioassays (*AQUA-Science* 1997a) on 78-d-old sturgeon larvae indicated a 96-h LC_{50} of 416 µg DDAC/L, which is consistent with other fish bioassays. Test conditions were: temperature = 16°C to 17°C; dissolved oxygen = 5.4 to 9.8 mg/L; pH = 7.05 to 7.79; hardness = 94 to 135 mg/L and alkalinity = 77 to 130 mg/L. This study also reported a 96-h LC_{25} of 225 µg/L and a 96-h NOEC of 100 µg/L. A second larval sturgeon bioassay was conducted with the addition of ca. 1/16" of sediment from the Fraser River in British Columbia to the

Table 5.37 Summary of didecyldimethylammonium chloride LC_{50} data for aquatic species from Brooks et al. (1996); values are in µg DDAC/L.

Species	96-h LC_{50}
Sturgeon larvae (*Acipenser transmontanus*)[a]	0.001 to 0.010
Sturgeon larvae (*Acipenser transmontanus*)[e]	0.415
Zebra fish[a]	0.172
Fathead minnow (80% DDAC)[a]	0.195 (0.122 to 0.224)
Bluegill sunfish (*Lepomis macrochirus*)[d]	0.320
Bluegill sunfish (*Lepomis macrochirus*)[d]	0.295
Bluegill sunfish (*Lepomis macrochirus*)[d]	0.270
Rainbow trout (*Oncorhynchus mykiss*)[a]	0.466 ± 0.1
Rainbow trout (*Oncorhynchus mykiss*)[d]	1.24 (range = 0.52 to 2.81)
Rainbow trout (*Oncorhynchus mykiss*)[c]	1.700 (1.4 to 2.0)
Coho salmon (*Oncorhynchus kisutch*)[d]	0.840 (range from 0.67 to 1.00)
Catfish[a]	0.590
Chinook salmon (*Oncorhynchus tshawytscha*)[a]	0.705 ± 0.015
Chinook salmon (*Oncorhynchus tshawytscha*)[d]	0.360
Guppies[d]	0.600
Sheepshead minnow (80% DDAC)[a]	0.940 (0.770 to 1.200)
Flounder[a]	2.050
Catfish (96-hr LC_{50})[d]	1.300
Daphnia magna[a]	0.052
Daphnia magna (48-h EC_{50})[b]	0.060 (0.051 to 0.072)
Mysid shrimp	0.069

a. Farrel cited in Brooks et al. (1996); b. Handley et al. (1994c); c. Handley et al. (1994b); d. Henderson (1992); e. Schoenig cited in Brooks et al. (1996) and Brooks (1998).

bottom of the test chambers (*AQUA-Science* 1997b). This bioassay was conducted under similar physicochemical conditions, but at reduced alkalinity (57 to 89 mg/L) and hardness (43 to 110 mg/L). Higher toxicity thresholds were determined in the presence of sediment: For purposes of this report, the sturgeon bioassay data reported by *Aqua-Science* (1997a) will be used (96-h LC_{50} = 416 µg DDAC/L). These data suggest that sturgeon larvae (*Acipenser transmontanus*) are not particularly sensitive to DDAC.

5.8.2.4 Chronic toxicity associated with DDAC

Limited data were available describing the chronic effects of DDAC for aquatic species. Twenty-four hour exposures of rainbow trout to 400 µg/L have been shown to affect blood plasma glucose levels, plasma corticosteroid hormones, and lactate levels. Reduced swimming performance in rainbow trout was observed by Wood et al. (in preparation, cited in Brooks et al. 1996) at 200 µg DDAC/L. Table 5.38 summarizes the available NOECs for DDAC.

5.8.2.5 Effects of dissolved organic carbon

Wood leachate contains high levels of organic extractives in addition to preservative. Taylor et al. (1996) reported high biological oxygen demand (BOD) of 1,660 to >2,600 mg/L in wood leachate and DDAC has the potential to bind to these organic compounds. In general, sorption of compounds to DOC reduces their bioavailability and thus their toxicity (Mills 1993). As an example, Springborn Laboratories (1994) reported decreased acute toxicity of DDAC to fathead minnows with increasing DOC. The results of the study are summarized in Table 5.39. The data shows that DDAC behaves like other organic molecules (PAH, etc.) in that toxicity is mediated by the availability of organic material in the water column and in sediments. Ultimately, the availability of organic carbon (DOC, POC, sedimented TOC, etc.) should be taken into account in developing either water or sediment standards for DDAC.

5.8.2.6 DDAC toxicity to aquatic plants

Handley et al. (1994a) reported a DDAC NOEC of 1,000 µg/l with a 96-h EC_{50} of 3,000 µg/L for *Scenedesmus subspicatus*. The end points examined in different tests were 50% reductions in growth rate and biomass. Henderson (1992) cited Walker and Evans (1978) in noting that QACs suppress growth in *Chlorella* and duckweed (*Spirodela oligorhiza*) at concentrations above 3,000 to 5,000 µg/L. In duckweed, sublethal levels of QACs caused a yellowing or browning of the frond margins and the production of smaller sized fronds. In *Chlorella,* the size, shape, and internal organization of the cells were affected; death appeared to be due to disruption of chloroplast structures.

5.8.2.7 Carcinogenic, mutagenic, and teratogenic effects

Henderson (1992) reviewed several studies describing each of these issues and found no evidence of adverse effects. His review suggested that Didecyldimethylammonium chloride does not have carcinogenic, mutagenic or teratogenic effects in mammals.

5.8.2.8 Recommended DDAC water column benchmarks

Based on the available toxicological database described above, it appears that the geometric mean of the two values of the NOEC for *Daphnia magna* (49 µg DDAC/L) provides a conservative benchmark against which to judge environmental risk. This value is considered conservative because non-linear regression analysis of the NOEC versus DOC data in Table 5.39 suggests an increase in the NOEC of $\exp^{0.116 \times DOC\ (mg/L)}$ as DOC increased. This simple analysis suggests that NOEC would be 12% higher at 1.0 mg/L DOC

Table 5.38 Summary of no-observed effect concentrations (NOEC) measured for DDAC.

Species	NOEL (Mg/L)
Rainbow trout[b] (*Oncorhynchus mykiss*)	1,000
Bluegill sunfish (*Lepomis macrochirus*)	100
Coho salmon (*Oncorhynchus kisutch*)	590
Algae (*Scenedesmus subspicatus*)[a]	1,000
Daphnia magna[c]	32
Daphnia magna[d]	74
Mysid shrimp	52

a. Handley et al. (1994a); b. Handley et al. (1994b); c. Handley et al. (1994c); d. Henderson (1992).

Table 5.39 The effect of dissolved organic carbon (DOC) on toxicity of DDAC to fathead minnows in a 96-h static renewal acute toxicity test.

Amount of humic acid added to DDAC solution (mg/L)	96-h LC_{50} (µg DDAC/L) with 95% confidence limits	NOEC (µg DDAC/L)
0.0	190 (160–270)	92
10.0	770 (650–1,000)	400
20.0	1,200 (940–1,600)	940

and 42% higher at 3.0% DOC, both of which are realistic concentrations in natural surface waters. A summary of the DOC data and regression analysis is provided in Figure 5.4. The physicochemical interactions resulting in the observed decrease in toxicity are unknown. Based on the very low (near zero) octanol-water partition coefficient, it would not appear that DDAC readily complexes with DOC.

It was the conclusion of the Antisapstain Review Committee in British Columbia that insufficient evidence existed for lowering the stormwater discharge criteria from 700 µg DDAC/L to 395 µg DDAC/L. It should be noted that applying a safety factor of ten to the current discharge limit of 700 µg DDAC/L results in an allowable receiving water concentration of 70 µg DDAC/L, which is higher that the 49 µg DDAC/L benchmark chosen for the risk assessment presented in Chapter 9.

5.8.2.9 Toxicity associated with sedimented DDAC

No empirical evidence describing the fate and transport of DDAC in open aquatic environments was found. The high water solubility and low K_{OW} of DDAC suggests that it will likely bind ironically to finely divide inorganic particulate material, particularly clay, in the water column. The strong sorption to soils reported in Brooks et al. (1996) suggests little propensity to desorb and re-enter the water column from sediments. No information was available describing the potential for infaunal detritivores to ingest DDAC sorbed to clay particles or for the movement of this material across the gut epithelia. However, Henderson (1992) observed poor absorption of DDAC across the gut of fish, very low bioconcentration factors and a small potential for biomagnification through food chains. He also reported that fish rapidly catabolize and/or excrete DDAC. Based on this evidence, it appears unlikely that significant biological risks to eukaryotes will be associated with DDAC at sedimented values less than 500 to 700 mg DDAC/kg dw. There is evidence that sediment concentrations above 10 mg DDAC/kg could affect microbial flora resulting in unknown changes in nutrient fluxes and decomposition of organic matter in sediments. ABC Laboratories (1995) examined the toxicity of DDAC incorporated into sediments to a midge (*Chironomus tetans*). Test chambers were maintained in a temperature-controlled waterbath with temperatures ranging from 21°C to 22°C. Dissolved oxygen ranged from 3.3 to 8.1 mg/L throughout the test. Water pH varied from 6.9 to 8.3. Measured endpoints included survival and growth at the end of 14 d as well as survival and emergence success at the end of 28 d exposure. The 14-d and 28-d LC_{50} values were calculated at >1,000 and 2,085 mg DDAC/kg sediment respectively. A 14-d EC_{50} value was calculated at 1,287 mg DDAC/kg based on total observed adverse effects (decreased size and mortality). The NOEC, based on larval weight and time to emergence was 530 mg DDAC/kg. The geometric mean maximum acceptable toxicant concentration (MATC) calculated from the above data was 728 mg DDAC/kg dw

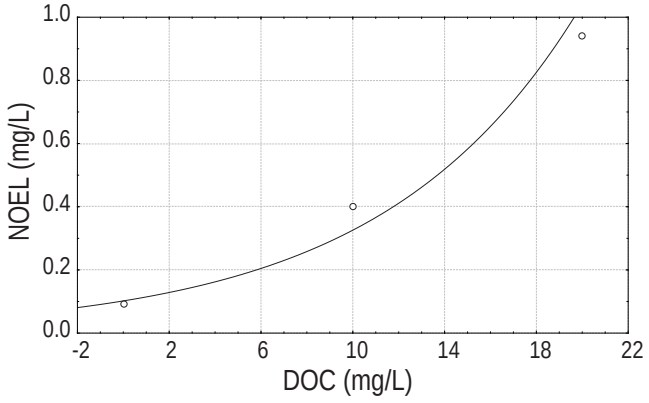

*Figure 5.4 Effect of dissolved organic carbon (DOC) on the no observed effect level (NOEL) of didecyldimethylammonium chloride (DDAC) to fathead minnows in a 96-h static renewal acute toxicity test (data from Springborn Laboratories, 1994). Scatterplot (DDACTOC.STA 10v*10c) y = 0.102*exp(0.116*x) + eps.*

5.8.2.10 Recommended sediment benchmark for DDAC

For purposes of the risk assessment presented in Chapter 9, it is assumed that DDAC dissolved in the water column adsorbs to clay particles followed by sedimentation. A sediment benchmark of 10 mg DDAC/kg dw is adopted to protect microbial communities. The data for larval sturgeon and midges discussed above suggests that this benchmark is very conservative when applied to eukaryotes.

5.8.2.11 Summary of recommended benchmarks for dissolved and sedimented DDAC

Regulatory WQC or SQS are not available for DDAC. DDAC does not appear to bioconcentrate or biomagnify. The No-Observed-Effect-Level (NOEL) for *Daphnia magna* (49 µg DDAC/L) is adopted as a benchmark. It appears that DDAC is not bioavailable in sediments. However, it exhibits anti-microbial activity at concentrations above 10 mg

DDAC/kg dry sediment weight and this value has been chosen as a sediment benchmark.

5.9 Tebuconazole

Tebuconazole is a xylem systemic fungicide. It has a water solubility of 32 mg/L and a low vapor pressure of 1.3 x $^{10-8}$ mm Hg. Based on these chemical characteristics, it is not expected to volatilize from surface water to the atmosphere. Tebuconazole has a low potential for bioconcentration or biomagnification with a predicted BCF of 78 to 141 and low K_{ow} = 2.99 to 3.7. It is resistant to biodegradation (i.e., primary biodegradation will take days to weeks and ultimate biodegradation may take months) and does not appear to be significantly metabolized by fish (LanXess 2005). Tebuconazole's relatively low water solubility suggests that it will adsorb to sediment or soil particles thereby reducing migration through soil to groundwater. These model predictions are confirmed by the U.S. EPA (1994), which also noted that tebuconazole was resistant to hydrolysis and photolysis.

5.9.1 Fate of tebuconazole (TEB) in the environment

Bayer (2005) reported that tebuconazole does not readily degrade by photolysis, hydrolysis or biodegration. However, mesocosm studies with water and sediments indicated tebuconazole half-lives of 43 d at a concentration of 3.2 µg TEB/L and 49 d at 32 µg TEB/L. The mean half-life was reduced to 31 d when restricted to the water phase only.

5.9.1.1 Acute toxicity associated with dissolved tebuconazole in freshwater environments

Toxicity to aquatic plants and animals is summarized in Table 5.40.

5.9.1.2 Chronic toxicity associated with dissolved tebuconazole in freshwater environments

The results of bioassays conducted to assess chronic toxicity are summarized in Table 5.41 using data from Bayer (2005). For surface water, the dynamic tests are more appropriate and the means of the LOEC and LOEC were 18.5 µg/L for fish and 175 µg/L for *Daphnia magna*. The tested algae tolerated higher concentrations of tebuconazole (1,000 to 1,190 µg/L) with no observed effect on either growth rate or biomass. The bacterial community in activated sludge was not affected by tebuconazole.

5.9.1.3 Mutagenicity and genotoxicity of tebuconazole

Long-term mamalian studies with rats, mice and dogs suggest that tebuconazole is not mutagenic or genotoxic (WHO 1994).

5.9.2 Recommended benchmarks for evaluating environmental risks associated with tebuconazole

Regulatory water and sediment quality criteria (WQC and SQC) were not found for tebuconazole.

5.9.2.1 Tebuconazole water quality benchmark

The limited data provided in Table 5.40 suggests an acute (2-d) benchmark of 320/10 = 32 µg TEB/L. Based on the limited bioassay data reviewed in Table 5.41, it appears that the mean of the dynamic rainbow trout LOEC and NOEC (18.5 µg TEB/L) is an appropriate interim chronic benchmark that should be assessed against TEB losses from pressure-treated wood on d 30. Data for development of marine benchmarks were not found.

Table 5.40 Bioassay data describing the acute toxicity of tebuconazole to aquatic biota.

Species	End point	Concentration (µg/L)
Americamysis bahia (opossum shrimp)	96-h LC_{50}	320 – 660
Cyprinodon variegates (sheepshead minnow)	96-h LC_{50}	5,900[a]
Pseudokirchneriella subcapitata (algae)	5-d EC_{50}	1,450 – 2,830[a]
Daphnia magna (water flea)	48-h EC_{50}	4,000[a]
Daphnia magna (water flea)	96-h EC_{50}	490[a]
Lepomis macrochirus (bluegill sunfish)	96-h LC_{50}	4,200 – 6,400
Oncorhynchus mykiss (rainbow trout)	96-h LC_{50}	5,700[a]
Leuciscus idus melanotus (golden orfe)	96-h LC_{50}	8,700

a. U.S. EPA ECOTOX database.

Table 5.41 Bioassay data describing the chronic toxicity of tebuconazole to aquatic biota.

Species	End point	Concentration (µg/L)
Fish		
Oncorhynchus mykiss (rainbow trout)	21-d LOEC[a]	32.0
Oncorhynchus mykiss (rainbow trout)	21-d NOEC[a]	10.0
Oncorhynchus mykiss (rainbow trout)	60-d LOEC[b]	25.0
Oncorhynchus mykiss (rainbow trout)	60-d NOEC[b]	12.0
Invertebrates		
Daphnia magna	21-d reproduction LOEC[c]	30.0
Daphnia magna	21-d reproduction NOEC[c]	10.0
Daphnia magna	21-d reproduction LOEC[d]	230.0
Daphnia magna	21-d reproduction NOEC[d]	120.0
Algae		
Desmodesmus subspicatus[e]	96-h growth inhibition NOEC	1,000
Pseudokirchneriella subcapitata[e]	96-h growth inhibition NOEC	1,190
Lemna gibba (duckweed)	14-d EC_{50}	1511
Bacteria		
Respiration inhibition test with activated sludge (OECD 209) resulted in an EC_{50} of >10,000,000 µg/L		

a. Static test using method OECD 204.

b. Dynamic (flow-through) using method U.S. EPA 72-4.

c. Static test using method OECD 211.

d. Dynamic (flow-through) using method U.S. EPA 72-4.

e. Static test using OECD 201.

5.9.2.2 Tebuconazole sediment quality benchmark

No data were found allowing an assessment of the toxicity of tebuconazole to macrofauna in aquatic environments. However, Bayer (1998) noted an aerobic soil half-life >365 d and an anaerobic aquatic half-life that was also >365 d but less than the aerobic half-life. Tebuconazole appears to be persistent in soil and sediments with long half-lives. For purposes of determining its accumulation in sediments, a half-life of 46 d is assumed based on information in Bayer AG (2005). No interim sediment standard can be recommended for this fungicide.

5.10 Propiconazole

Propiconazole is a fungicide used in combination with copper or other organic pesticides for preserving wood. It has a water solubility of 150 mg/L at 20°C, a low vapor pressure of 5.6×10^{-5} Pa at 25°C, and a log K_{ow} of 3.72 at pH = 6.6 and 25°C. The EXTOXNET chemical profile notes that the migration of propiconazole in alkaline, low organic soil is restricted to the top 25.4 cm of soil. Vertical movement in soils with high clay and TOC content will likely be restricted to 7.5 cm.

5.10.1 Fate of propiconazole in aquatic environments

The half-life of propiconazole in natural freshwater environments is estimated at 85 d, with hydrolysis being the primary route of degradation (Arch 2005). The compound is relatively photostable with a variable photolytic half-life of 47–984 d at latitudes between 30° and 50°N. The Log_{Kow} of 3.72 suggests that propiconazole is moderately hydrophobic and will likely adsorb to particulate organic and inorganic matter suspended in the water column followed by sedimentation.

Arch (2005) reported that 96.5% to 98.1% of propiconazole was initially found in mesocosm water. At the end of 175 d, 83% to 87.5% of the compound was found in sediments and only 2% remained in the water. No information was found describing the half-life of propiconazole in sediments or water–sediment environments. The compound's half-life in aerobic soil is reported by Arch (2005) to vary between 17 and 430 d. The average half-life for 26 field and laboratory measurements of half-life in soil was 111 d. Half-lives in anaerobic soils were not provided. However, 20.2% of the active ingredient was reported remaining at the end of 119 d.

5.10.1.1 Acute toxicity associated with dissolved propiconazole in freshwater environments

This is a systemic triazole fungicide that acts by inhibiting the 14-demethylase enzyme, common in all plants, animals, protists, fungi, and some bacteria sterol synthesis pathways. Table 5.42 lists bioassay data cited by Egstrom (DRAFT) provided from Arch (2005) or extracted from the U.S. EPA ECOTOX (ECOTOXicology) Database, available at http://cfpub.epa.gov/ecotox/ and the Extension Toxicology Network Database (EXTOCNET) available at http://pmep.cce.cornell.edu/profiles/extoxnet/. The lowest acute 96-h LC_{50} is 670 for rainbow trout. Application of a safety factor of ten suggests an acute (4-d average) benchmark of 67 µg propiconazole/L. This appears overprotective for other species and rainbow trout.

5.10.1.2 Chronic toxicity associated with dissolved propiconazole in freshwater environments

EXTOCNET lists a BCF for bluegill sunfish of 116 and a 98% depuration time of 14 d. This limited data suggests that propiconazole will not significantly bioconcentrate, bioaccumulate or biomagnify in food chains—mitigating the potential for chronic effects. However, insufficient information was obtained to make an unequivocal statement in this regard. The 21-d NOEC for *Daphnia magnia* of 310 µg/L and the 28-d NOEC for *Chironomus riparius* of 8,000 µg/L are the only chronic animal data found (Table 5.43). The single data point for *Navicula seminulum* of 93 µg/L suggests that plants may be more sensitive to propiconazole than animals. For purposes of the risk assessment discussed in Chapter 9, a chronic value of 93/10 = 9.3 µg

Table 5.42 Bioassay data describing the acute toxicity of propiconazole to aquatic biota.

Fish species	End point	Concentration (µg/L)[a]
Lepomis macrochirus (bluegill sunfish)	96-hr LC_{50}	1,300 – 9,800
Oncorhynchus mykiss (rainbow trout)	96-h LC_{50}	830 – 506,000
Cyprinus carpio (common carp)	72- to 96-h LC_{50}	5,700 to 46,000
Ictalurus punctatus (channel catfish)	96-h LC_{50}	12,000
Phoxinus phoxinus (minnow)	96-h LC_{50}	1,800
Rutilus rutilus (roach)	96-h LC_{50}	1,800
Salmo trutta (brown trout)	96-h LC_{50}	1,200 – 3,390
Invertebrate species		
Gammarus lacustris (amphipod)	96-h LC_{50}	1,300
Heptagenia sulphurea (mayfly)	96-h LC_{50}	1,000
Procambarus sp. (crayfish)	96-h LC_{50}	49,000
Daphnia magna (water flea)	48-h EC_{50}	3,200 – 11,300
Americamysis bahia (opossum shrimp)	96-h EC_{50}	510 – 1,420
Baetis rhodani (mayfly)	96-h LC_{50}	900
Heptagenia sulphurea (mayfly)	96-h LC_{50}	1,000
Hydropsyche siltalai (caddisfly)	96-h LC_{50}	1,200
Plants		
The U.S. EPA ECOTOX Database[1] contains effects data for 23 species of freshwater and marine algae. Acute effects on these plants were assessed using short-term (i.e., ≤ 96-h) EC_{50} tests. Reported effects concentrations ranged from 83 µg/L for 24-h tests of 10 marine microalgae species to 32,000 µg/L for *Pseudokirchneriella subcapitata*. Most values were in the range of 2,200 to 6,500 µg/L. However, a 72-h EC_{50} of 0.8 µg/L was reported for *Chlamydomonas noctigama* and a mean 48-h EC_{50} of 55 µg/L was reported for *Rhodomonas lacustris*.		
Marine species		
Invertebrates		
Mysidopsis bahia (mysid shrimp)	96-h LC_{50}	510[b]
Litopenaeus vannamei (white shrimp)	24 to 96-h LC_{50}	950 – 1,167
Crassostrea virginica (Virginia oyster)	96-h EC_{50}	1,700[b]
Fish		
Leiostomus xanthurus (spot)	96-h LC_{50}	2,200 – 3,900
Cyprinodon variegatus (sheepshead minnow)	96-h LC_{50}	68[b]

a. U.S. EPA ECOTOX Database, except where indicated; b. Bayer AG (2005).

propiconazole/L will be adopted for assessment 5 d after construction.

5.10.1.3 Acute toxicity associated with dissolved propiconazole in marine environments

Five data points are included in Table 5.42 for marine vertebrates and invertebrates. The lowest of these is 68 µg propiconazole/L for *Cyprinodon variegatus.* A safety factor of ten would imply a benchmark value of 6.8 µg propiconazole/L of seawater.

5.10.2 Biological response to sedimented propiconazole.

LanXess (2001) listed a sediment 28-d NOEC for *Chironomus riparius* of 25.0 mg propiconazole/kg. No other information was found describing the toxicity of sedimented propiconazole. However, the Extension Toxicology Network notes an earthworm LC_{50} (unknown test duration) of 686 mg propiconazole/kg. For purposes of the risk assessment described in Chapter 9, a preliminary freshwater sediment benchmark equal to the 28-d NOEC for *Chironomus riparius* (25.0 mg/kg) will be used. However, it is emphasized that this is based on a single test and the benchmark carries a low level of confidence.

5.10.3 Preliminary benchmarks for managing propiconazole in aquatic environments

Regulatory WQC or SQC were not found for propiconazole and a limited amount of information was found in support of this analysis. Preliminary propiconazole benchmarks for purposes of interpreting output from the risk assessment model are summarized in Table 5.44. These benchmarks may need to be superceeded as new information becomes available or regulatory criteria are published.

Table 5.43 Bioassay data describing the chronic toxicity of propiconazole to aquatic biota.

		Chronic toxicity (µg/L)
Invertebrates		
Daphnia magna (water flea)	21-d NOEC	310
Chironomus riparius (midge)	28-d NOEC	8,000
Algae		
Navicula seminulum (algae)	11-d EC_{50}	93

Table 5.44 Benchmarks for managing the use of propiconazole in aquatic environments.

Propiconazole benchmark	Value
Day 2.0 acute freshwater benchmark	510/10 = 51.0 µg/L
Day 10.5 chronic freshwater benchmark	93/10 = 9.3 µg/L
Day 2.0 acute seawater benchmark	68/10 = 6.8 µg/L
Chronic seawater benchmark	no data
Freshwater imidacloprid sediment benchmark	25 mg/kg dry sediment

5.11 Imidacloprid

Imidacloprid is a commercial insecticide with a water solubility of 510 mg/L at 20°C, a low vapor pressure of 1.5x 10^{-9} mm Hg, and a log K_{ow} of 0.57 LanXess (MSDS). Edstrom (DRAFT) reported a predicted BCF of 3 using EPI Suite.

5.11.1 Fate of imidacloprid in aquatic environments

Imidacloprid is not expected to bioaccumulate to a significant level in aquatic organisms and should not volatilize from surface water to the atmosphere. Its moderate water solubility and low K_{ow} suggests that it will not adsorb to sediment or soil particles to a significant degree. EXTOXNET reports primary degradation in the field, DT50, to range from 7 to 146 d and notes that imidacloprid has a moderate affinity for organic ligands. Imidacloprid is hydrolytically stable across pH values of 5 to 11. However, the compound is reported by LanXess (2005) to readily absorb UV light and a high rate of photolysis is expected in water. This information suggests that imidacloprid dissolved in rainwater will not readily settle to sediments, but should remain dissolved in the water column for an extended period where it will be photolytically degraded or will adsorb to particulate organic matter followed by sedimentation. For purposes of the risk assessment in Chapter 9, a degradation half-life of 146 d will be assumed for sedimented imidacloprid.

5.11.2 Biological effects of imidacloprid in aquatic environments

LanXess (MSDS) reports no evidence of carcinogenicity or teratogenicity at the doses tested. The NOEL for neurotoxicity was 3,027,000 µg/L.

5.11.2.1 Acute toxicity associated with dissolved imidacloprid in freshwater environments

Imidacloprid is a systemic chloronicotinyl insecticide used for control of sucking insects. The chemical acts as an acetylcholinesterase inhibitor causing a buildup of acetylcholine and blocking the transmission of neural impulses. The U.S. EPA ECOTOX Aquatic Database contains 179 entries for imidacloprid (CAS # 138261413-1). Reported LOECs and 96-h LC_{50}s for imidacloprid are summarized in Table 5.45 for development of an acute benchmark.

5.11.2.1 Chronic toxicity associated with dissolved imidacloprid in freshwater environments

Long-term LOEC and NOEC results for imidacloprid recorded in the U.S. EPA ECOTOX Database are in Table 5.46.

5.11.3 Biological response to sedimented imidacloprid

No information was obtained.

5.11.4 Preliminary benchmarks for managing imidacloprid in aquatic environments

Fish appear to tolerate relatively high concentrations of imidacloprid for short periods of time (96-h LC_{50} >85,000 µg/L) as do the tested frogs (145,000 to 296,000 µg/L).

5.11.4.1 Recommended freshwater acute benchmark

The most sensitive species listed is *Epeorus longimanus*, a mayfly in the Order Ephemeroptera, with calculated 24-96 h LC_{50}s between 0.7 and 2.1 µg/L and a 24-h LOEC of 5.8 µg/L. *Epeorus* sp. are widespread clingers found in lotic

Table 5.45 Short-term bioassay data describing the acute toxicity of imidacloprid to aquatic biota. Data from U.S. EPA ECOTOX and the EXTOXNET databases.

Test organism	Test endpoint	Concentration (µg/L)
Rana limnocharis (bog frog)	96-h LC_{50}	296,000[a]
Rana nigromaculata (black spotted frog)	96-h LC_{50}	115,000–145,000[a]
Cypridopsis vidua (ostracod)	48-h LC_{50}	273–715[a]
Daphnia magna (water flea)	48-h LC_{50}	10,440–64,873[a]
Ilyocypris dentifera (ostracod)	48-h LC_{50}	214–517[a]
Aedes aegypti (yellow fever mosquito)	48–72-h LC_{50}	44 - >6,300[a]
Aedes albopictus (mosquito)	24-h LC_{50}	300–800[a]
Aedes taeniohynchus (mosquito	24–48-h LC_{50}	13–21[a]
Culex quinquefasciatus (mosquito)	24-h LC_{50}	40–400[a]
Epeorus longimanus (mayfly)	24–96 h LC_{50}	0.7–2.1[a]
Cypretta seurati (ostracod)	48-h EC_{50}	1–16[a]
Cypridopsis vidua (ostracod)	48-h EC_{50}	3–16[a]
Daphnia magna (water flea)	48-h EC_{50}	6,029–85,200[a]
Ilyocypris dentifera (ostracod)	48-h EC_{50}	6–24[a]
Chydorus sphaericus (water flea)	48-h EC_{50}	301–132,673[a]
Epeorus longimanus (mayfly)	24-h LOEC	5.8[a]
Fish		
Oncorhynchus mykiss (rainbow trout)	96-h LC_{50}	83,000–229,100[a,b]
Cyprinis carpio (common carp)	96-h LC_{50}	280,000[b]
Leuciscus idus melanotus (golden ofre)	96-h LC_{50}	85,000[b]
Lepomis macrochirus (bluegill)	96-h LC_{50}	>105,000[a]
Marine species		
Artemia sp. (brine shrimp)	48-h LC_{50}	361,230[a]
Palaemonetes pugio (glass shrimp)	96-h LC_{50}	308.8–563,5[a]
Cyprinodon varigatus (sheepshead minnow)	96-h LC_{50}	163,000[a]

a. U.S. EPA ECOTOX Database.
b. EXTOXNET Database

Table 5.46 Bioassay data describing the chronic toxicity of imidacloprid to aquatic biota. Data from the U.S. EPA ECOTOX Database.

Test organism	Test endpoint	Concentration (µg/L)
Invertebrates		
Daphnia magna (water flea)	21-d LOEC	1,250–40,000
Daphnia magna (water flea	21-d NOEC	625–20,000
Pteronarcys dorsata (stonefly)	14-d LOEC	47–38,600
Tipula sp. (cranefly)	14-d LOEC	135–81,300
Invertebrate community abundance	127-d NOEC	120–240
Fish		
Oryzias latipes (Japanese medaka)	30-d NOEC	120

(erosional) environments associated with high current speeds (Merrit and Cummins 1978) where contaminants from treated wood are significantly diluted. Ostracods are also sensitive with 48-h LC_{50} values ranging between 214 and 715 µg/L. Ostracods, including *Cypridopsis* sp. are widespread and found in a variety of environments including depositional areas associated with slow moving water. When current speeds are <10 cm/s, the recommended benchmark for freshwater systems is based on the lowest 48-h LC_{50} reported for *Ilyocypris dentifera* (214 µg/L) with a safety factor of 10 applied or 21.4 µg/L. When current speeds are >10 cm/s, a benchmark based on the LOEC for *Epeorus longimanus* (5.8 µg/L) is recommended with a safety factor of 10 applied giving a recommended benchmark of 0.58 µg/L.

5.11.4.2 Recommended freshwater chronic benchmark

Given the available bioassay data, an appropriate chronic benchmark likely lies between the LOEC and the NOEC. For all species tested, the three NOECs listed in Table 5.46 are higher in concentration than the lowest LOEC and the few data make this determination problematic. The lowest LOEC is for the stonefly (*Pteronarcys dorsata*). The Order Plecoptera inhabits erosional environments characterized by high current speeds. In addition, the range of recorded 14-d LOECs is extreme (47 to 38,600 µg/L). The chosen benchmark is equal to the lowest 127-d NOEC for invertebrate community abundance (120 µg/L) with a safety factor of 10 applied giving a benchmark of 12 µg/L.

5.11.4.3 Recommended marine acute and chronic benchmarks

Data for three species were found in the ECOTOX Database (Table 5.47). An acute benchmark based on the lowest recorded 96-h LC_{50} for glass shrimp (*Palaemonetes pugio*) with a safety factor of ten is recommended (30.8 µg/L). The recommended chronic benchmark is based on the same 96-h LC_{50} bioassay data, but with a safety factor of 20 applied (15.5 µg/L).

5.11.4.4 Long-term freshwater imidacloprid sediment benchmark

No information was obtained. However, based on the low potential for imidacloprid to adsorb to particulate inorganic matter and short photolytic half-life in water (4 h from EXTOXNET), imidacloprid is not expected to accumulate in sediments to a significant degree.

5.12 Bioassays for Assessing the Combined Effects of Active Ingredients

The latest generation of wood preservatives, including ACQ-B, ACQ-C, CAB, Wolman AG™, and micronized copper products include several fungicides, bacteriocides and

Table 5.47 Proposed benchmarks for managing the use of imidacloprid in aquatic environments.

Imidacloprid benchmark	Value (µg/L)
Day 1.0 acute freshwater benchmark (current speed <10 cm/s)	27.30
Day 1.0 acute freshwater benchmark (current speed >10 cm/s)	0.58
Day 10.5 chronic freshwater benchmark	120.0/10 = 12.0
Day 2.0 acute seawater benchmark	308.8/10 = 30.8
Chronic seawater benchmark	308.8/20 = 15.4

Figure 5.5 Experimental decks with installed Cubitainers™ ready to collect the first 1.5 cm of rainwater runoff. Two of the decks are covered with southern pine treated to a mean retention of 0.24 kg/m³ Wolman AG™ preservative.

insecticides originally developed for use on terrestrial agricultural crops. These active ingredients include propiconazole, tebuconazole, imidacloprid and didecyldimethylammonium chloride (DDAC). The effects of the individual compounds were reviewed above. However, no information describing the combined effects of these biocides plus copper and the wood extractives present in rainwater runoff from pressure-treated wood was found. The combined effects on aquatic flora and fauna may be antagonistic, additive or synergistic. Brooks (2007) reported the results of a study designed to assess the combined effects of propiconazole, tebuconazole, imidacloprid and wood extractives released from a deck constructed with Wolman AG™ preserved southern pine decking.

5.12.1 Wolman AG™ study design

Southern pine decking (3.8 cm thick x 8.9 cm wide x 2.44 m long) was pressure-treated in two lots having measured retentions of 0.25 and 0.22 kg Wolman AG™/m³ (0.0159 *pcf* and 0.014 *pcf*). Wolman AG™ preservative contains 5% propiconazole, 5% tebuconazole, and 0.5% imidacloprid. The wood from the two lots was mixed and used to surface two of the three decks shown in Figure 5.5. Each deck measured 2.44 m x 2.44 m. The large size was necessary to collect the 66 L of rainwater required for a suite of five bioassays during the first 1.5 cm of rainfall. Rain was collected on PVC covered plywood located under the decks and slanted to direct the runoff into 22 L Cubitainers™ (Figure 5.5). The third deck was covered with untreated southern pine decking.

5.12.1.1 Climatological data

A variety of climatological endpoints including barometric pressure, rainfall, wind speed and direction, humidity, temperature and dew point were measured continuously at Aquatic Environmental Sciences using a Davis Vangage Pro™ weather station with a remotely located recorder and WeatherLink 5.1 software. Temperature during sample collection varied between 35.7 and 45.1°F.

5.12.1.2 Collection, storage and shipment of samples

Cubitainers™ were covered with opaque (black) plastic sheeting during collection of the first 66 L of runoff. Samples were stored in the dark in refrigerators and shipped via an overnight delivery service to the Pacific Environmental Science Center (PESC) and LanXess Germany for completion of the bioassays and analytical chemistry. Samples were maintained at 4 ± 2°C to minimize microbial catabolism of the biocides.

5.12.1.3 Sample analysis

Concentrations of tebuconazole, propiconazole, and imidacloprid in the rainwater runoff and all dilutions created during the bioassays were determined using HPTLC (AMD) with UV densitometric scanning according to Analytical Method 2201-0323401-99D by Dr. Thomas Jaetsch at the MPP Technical Center of LanXess Deutschland in Uedingen, Germany. Organic matter in the runoff was accumulated using RP18 cartridges at pH 2 (SPE technique) and subse-

quently separated on silica gel (AMD, solvent gradient elution). Pesticides were detected by UV densitometric scanning and determined by external standards.

5.12.1.4 Quality control

The analytical system was calibrated using 100 ng of each pesticide. No other quality assurance report was provided by LanXess Deutschland.

5.12.1.5 Bioassays at PESC

Leachate toxicity was initially characterized in a 48-h LC_{50} screening test on *Daphnia magna* followed by a 21-d chronic reproductive test using the same species to determine IC_{50} and IC_{25} concentrations. Other definitive tests included determination of the 96-h LC_{50} for juvenile salmonids (*Oncorhynchus mykiss*) and an amphipod (*Hyalella azteca*). The 72-h IC_{25} and IC_{50} concentrations were determined for the growth of an alga (*Selenastrum* sp.), and 5 and 15-min LC_{50} concentrations were determined for reductions in light emissions from the biolumninescent bacterium *Vibrio fischeri* using the Microtox™ system.

5.12.1.6 Analytical chemistry in support of the bioassays

The following samples were collected in EPA clean 250 ml amber Nalgene™ bottles by the Pacific Environmental Science Center upon receipt of the nine cubitainers of rainwater runoff.

- Samples from each of the six Cubitainers™
- Composites of the three replicates from each deck were created to obtain three stocks containing 66 liters each. A sufficient subsample from T2 and T3 was collected to analyze (at PESC) the parameters listed below. These same parameters were analyzed in the rainwater sample provided with the shipment.

Dissolved oxygen	Arsenic
Temperature	Chromium
pH	Copper
Dissolved Organic Carbon	Zinc
Total Organic Carbon	Cadmium
Hardness	Lead
Alkalinity	Magnesium
Potassium	Calcium
Sulfate	Sodium
Sulfide	Chlorine

- A *Daphnia* range-finding test using a composite of the water from T2 and T3 (the two treated decks) was initially undertaken to determine an appropriate dilution series.
- Subsamples of each dilution used in the bioassays (for all three treatments) were collected in amber 250 ml Nalgene™ bottles for analysis TEB, PROP, and IMID by LanXess Corporation.
- At the end of each bioassay using T2 and T3 (the treated deck samples), each dilution was sampled for determination of the amount of residual TEB, PROP, and IMID.

5.12.1.7 Data analysis

Analytical chemistry results were returned to PESC and Aquatic Environmental Sciences by LanXess Corporation. PESC determined the concentration based endpoints for each bioassay.

5.12.1.8 Phase II replicate chronic freshwater algae (Papdidocelis subcapitata syn. Selenastrum capricornutum) bioassay

This study was initiated on March 12, 2008 using 5/4 southern pine decking treated to a nominal retention of 0.21 kg/m^3 (0.013 *pcf*) with Wolman AG™ preservative. Figure 5.6 describes the 60 cm x 60 cm collecting surface, which was constructed using four different treated boards. Water control dams were constructed of untreated southern pine and sealed with silicon seal. Rainwater runoff from the deck flowed directly into a two liter Cubitainer™ supplied by CH2M Hill laboratory. The sample collection was

Figure 5.6 Wolman AG™ preserved southern pine deck used to collect the first flush of rainwater for a replicate chronic freshwater algae bioassay in March 2008.

initiated on March 13, 2008, and completed on March 14, 2008, following 0.81 cm of rainfall. The cubitainer™ was stored in a refrigerator at 4°C and shipped to CH2M Hill on March 18, 2008. It arrived at the analyzing laboratory the next day at a temperature of 6.9°C.

5.12.1.9 Bioassay protocol

A chronic bioassay (EPA-821-R-02-013) using the freshwater green algae (*Raphidocelis subsapitata* syn. *Selenastrum capricornutum*) was conducted at CH2M Hill. Tested concentrations were 6.25, 12.5, 25, 50 and 100% effluent using moderately hard (80 to 100 mg/L as $CaCO_3$) dilution water that had been filtered to 0.45 µm and amended with nutrients and EDTA.

5.12.1.10 Analytical chemistry

A subsample of the whole rainwater runoff from the treated wood deck was forwarded on phase change gel packs via an overnight delivery service by CH2M Hill to Environmental Micro Analysis Inc. in Woodland, California for determination of the concentrations of imidacloprid, propiconazole and tebuconazole in the whole effluent. The samples were extracted 6 d after collection and analyzed the next day.

5.12.2 Results: Concentrations of TEB, PROP and IMID in the first flush of rainwater runoff

The three biocides were not detected in runoff from the untreated control deck (T1). Concentrations in the whole effluent from treated decks T2 and T3 are provided in Table 5.48. The difference in concentrations between the two treated decks was not statistically significant. Concentrations of the three active ingredients determined in the 2008 freshwater algae supplemental bioassay are also provided. The concentration of imidacloprid in the supplemental work was only 32% of that observed in the original T2 and T3 samples. However, because of the variability between the three T2 and three T3 sample, the difference was not significant. Concentrations of tebuconazole and propiconazole were similar in the two studies.

The ratio of tebuconazole:propiconazole:imidacloprid in Wolman AG™ preservative prior to treatment is 10:10:1. The ratio of the three active ingredients in the runoff from the treated decks is summarized as a function of time in Table 5.49. Recall that tebuconazole and propiconazole are photolytically and hydrolytically stable in water, with half-lives of 31 and 85 d, respectively. Imidacloprid is hydrolytically stable but appears to be phytolytically broken down by UV light absorption. Therefore, one would expect more imidacloprid loss during collection and handling of samples, leading to lower ratios of tebuconazole:imidacloprid and propiconazole:imidacloprid. That expectation is not supported by the data (Table 5.49), which shows marginally higher proportional concentrations of imidacloprid in all but the initial SYP tebuconazole:imidacloprid proportion. The literature suggests that the higher imidacloprid concentrations are not due to selective degradation of tebuconazole and propiconazole leaving higher proportional concentrations of imidacloprid—rather the data suggests higher initial and long-term proportional losses of imidacloprid from both hem-fir and southern yellow pine.

5.12.2.1 Bioassay results

Endpoints were determined using measured concentrations of the three biocides, except in those few cases when

Table 5.48 Concentrations (µg/L) of TEB, PROP, and IMID in 100% rainwater runoff from southern pine decks pressure treated to a retention of 0.015 pcf with Wolman AG™ preservative. Ninety-five percent confidence limits are provided.

Treatment	Imidacloprid	Tebuconazole	Propiconizole
T2	150 ± 124	1,847 ± 1,237	930 ± 596
T3	140 ± 660	1,813 ± 1,026	960 ± 455
All groups	145 ± 38	1,830 ± 0,430	945 ± 201
2008 supplemental algae	47	1,270	989

Table 5.49 Ratios of tebuconazole:propiconazole:imidacloprid in Wolman™ AG preservative in runoff from pressure-treated decks.

	Preservative	Initial hem-fir	Late hem-fir	Initial southern yellow pine (bioassay)
Tebuconazole	10	15.28 (5942)	5.86 (80.9)	12.62 (1830)
Propiconazole	10	14.13 (5495)	4.68 (64.6)	6.52 (945)
Imidacloprid	1	1.00 (389)	1.00 (13.8)	1.00 (145)

Table 5.50 Known toxicity characteristics of tebuconazole, propiconazole, and imidacloprid as individual compounds (from Brooks 2007 and the U.S. EPA ECOTOX Database).

Biocide (µg/L)	Rainbow trout 96-h LC_{50}	*Daphnia magna* 48-h EC_{50}	*Hyalella azteca* 48-h EC_{50}	*Scenedesmus subspicatus* 96-h LC_{50}	*Daphnia magna* 21-d reproduction LOEC
Tebuconazole	5,700	4,000	Not found	1,095[c]	97.5[b]
Propiconazole	253,415[d]	7,250	Not found	93[a]	310 (NOEC)
Imidacloprid	156,050	37,656	55	>10,000	1,250

a. Data for Navicula seminulum from Table 5.43
b. Average of 2 NOECs and 2 LOECs from Table 5.43
c. Average for two 96-h tests on algae from Table 5.43
d. Average of two values from Table 5.43. The lower of the two was 830 µg/L

Table 5.51 *Daphnia* acute lethality (48-h LC_{50}) range finding test.

	Endpoint			
	Mean value	Lower 95% CI	Upper 95% CI	Ecotox value
Nominal concentration (%)	15.81	11.83	21.13	
Tebuconazole (µg/L)	259.8	206.0	327.7	4,000
Propiconazole (µg/L)	123.3	93.8	162.1	7,250
Imidacloprid (µg/L)	28.3	22.7	35.2	37,656

the biocides were below the detection limit. Table 5.50 describes the toxicity of tebuconazole, propiconazole, and imidacloprid listed in Brooks (2007).

Acute *Daphnia* spp. range-finding test on a composite of the treated southern pine decks

This 48-h test was conducted to determine the most suitable dilution series for subsequent bioassays. All three biocides were detected in all dilutions >1%. The 48-h LC_{50} for *Daphnia* sp. was determined to be 15.8% of the whole runoff. Biocide concentrations were measured at the start and end of the bioassay and the similarity of the concentrations indicated that they were stable over the 48-h period. The mean concentrations of the active ingredients are provided in Table 5.51 (±95% CI) together with U.S. EPA ECOTOX Database values. Note that the concentrations of all of the active ingredients at the concentration of runoff at the 48-h LC_{50} are small fractions of the values for the neat compounds given in the ECOTOX Database.

21-d chronic *Daphnia* reproduction test

Table 5.52 provides summary results for the Chronic *Daphnia* Reproduction Test that determined LC_{50} and LC_{25} endpoints. Results are summarized for the control (T1) and treated (T2 and T3) decks. U.S. EPA ECOTOX records for this test are included in the last column of the table. The results for the control deck demonstrate measurable toxicity associated with the wood extractives alone, which caused a 50% reduction in reproductive output from *Daphnia* at concentrations ≥ 55.7% and a 25% reduction in 38.2% of the runoff from the untreated deck. The treated decks were more toxic with 25% and 50% reductions at nominal concentrations of 8.95% and 18.7%, respectively. The concentration of tebuconazole at the endpoints (163.8 and 342.2 µg/L) were significantly higher (less toxic) than the ECOTOX value of 97.5 µg tebuconazole/L. The concentrations of imidacloprid and propiconazole associated with both the IC_{25} and the IC_{50} were less (more toxic) than

Table 5.52 Results of the 21-d *Daphnia* reproductive bioassay for the untreated deck (C1) and decks T2 and T3 surfaced with Wolman AG™ pressure-treated southern pine.

Treatment and endpoint	Percent runoff	Tebuconazole (µg/L)	Propiconazole (µg/L)	Imidacloprid (µg/L)
Control deck (C1)				
IC_{50}	55.7	0	0	0
IC_{25}	38.2	0	0	0
Treated decks T2 & T3				
IC_{50}	18.7	342.2	176.7	27.1
IC_{25}	8.95	163.8	84.6	13.0
IC_{50} U.S. EPA Ecotox value		97.5	310	1,250

the ECOTOX record. These results suggest that the toxicities of tebuconazole in combination with the other contaminants were reduced in the runoff—perhaps associated with binding with the wood extractives. At the bioassay endpoints, imidacloprid was present at a concentration (13.0 and 27.1 µg/L) that was only 1% to 2% of the lowest EPA ECOTOX LOEC of 1,250 µg/L. Propiconazole concentrations were lower, but similar to the ECOTOX records. There are several ways to evaluate these results. The concentration of imidacloprid was so much lower than published data that it does not appear to have contributed to the overall effect. The toxicity of tebuconazole appears to have been reduced in the mixture and in general, its combined toxicity appears to have been antagonistic (the combined effect was less toxic than the neat compound).

96-h rainbow trout acute lethality test (96-h LC_{50})

The 66 L of runoff from the untreated deck provided sufficient water for only a 100% effluent test of rainbow trout. One hundred percent rainbow trout mortality was experienced in the initial control deck (CT1) bioassay due to low dissolved oxygen (DO) concentrations (2.25 mg O_2/L). This was likely caused by the BOD associated with the wood extractives. The runoff was aerated for 24 h increasing the DO to 6.5 mg/L and the test rerun. The final DO in the second test was 7.9 mg O_2/L, which is within the range acceptable to salmonids (i.e., >6.0 mg/L). Forty percent (40%) of the rainbow trout exposed to the 100% untreated deck runoff died during the 96-h test. There were no control mortalities. These results indicate that the effluent from the untreated deck was toxic to rainbow trout, but the results do not allow for determination of an $LC_{50.}$ Tests of the treated deck runoff (T2 and T3) were run at concentrations of 1.56, 3.13, 6.25, 12.5 and 25%. Dissolved oxygen concentrations varied between 8.6 mg O_2/L in the 25% dilution to 9.7 mg O_2/L in the 1.56% dilution with no anticipated adverse effects associated with these oxygen concentrations. Results for the treated decks are provided in Table 5.53. The concentrations of the active ingredients are all several orders of magnitude less than the values for the neat compounds given in the U.S. EPA ECOTOX Database suggesting significant synergy of the biocides and natural wood extractives in the mixture. In part, that may be because nearly half (40%) of the trout died in runoff from the untreated deck suggesting that much of the toxicity was associated with the wood extractives.

96-h Hyalella azteca acute lethality test (96-h LT_{50})

Runoff from the untreated control deck (CT1) caused 50% mortality in this species at concentrations ≥ 76% indicating moderate toxicity associated with the wood extractives. The results for Wolman AG™ pressured treated southern pine decks are summarized in Table 5.54. No data describing a 96-h LC_{50} for any of the 3 biocides was found for *Hyalella*. However, the U.S. EPA ECOTOX Database contains data for other arthropods and the lowest of these are provided in the last column of the table. The results suggest that the observed concentration of propiconazole was too low to be a factor. However, the WET concentratons of imidacloprid and tebuconazole are within the range at

Table 5.53 Results of the rainbow trout acute lethality test (96-h LC_{50}) of rainwater runoff from treated decks T2 and T3 surfaced with Wolman AG™ pressure-treated southern pine.

	Value	Lower 95% CI	Upper 95% CI	U.S. EPA Ecotox records
Nominal Concentration	8.25%	7.23%	9.41%	
Tebuconazole (µg/L)	156.4	137.1	178.4	5,700
Propiconazole (µg/L)	80.8	70.8	92.2	253,415
Imidacloprid (µg/L)	12.4	10.9	14.1	156,415

Table 5.54 Results of the *Hyalella azteca* A96-h LC_{50} bioassay using rainwater runoff from treated decks T2 and T3 surfaced with Wolman AG™ pressure-treated southern pine.

	Value	Lower 95% CI	Upper 95% CI	U.S. EPA Ecotox (lowest arthropods)
Nominal concentration	27.8%	24.1%	32.1%	
Tebuconazole (µg/L)	508.7	441.3	587.4	≤490
Propiconazole (µg/L)	262.7	227.7	303.3	≤1,300
Imidacloprid (µg/L)	39.2	34.9	46.5	≤45

which these compounds affect other arthropods. There is no evidence of additive or synergistic effects in the *Hyalella azteca* acute lethality test.

72-h algae growth inhibition test

Concentrations of 100%, 50%, 25%, 12.5% 6.25%, 3.13%, 1.56%, 0.78%, 0.39%, 0.20% and a control were used for all treatments in the initial algae bioassay. A 4.6% concentration of runoff from the untreated deck (CT1) inhibited the growth of *Selenastrum capricornutum* by 25% and a concentration of 37% reduced growth by 50%, suggesting significant toxicity to *Selenastrum capricornutum* associated with the wood extractives from untreated southern pine. Growth inhibition associated with runoff from the two treated decks (T2 and T3) is summarized in Table 5.55. Because runoff from the untreated deck was highly toxic, it is difficult to determine the additional contribution to toxicity associated with tebuconazole, propiconazole and imidacloprid leached from the treated decks. The concentration of tebuconazole at the IC_{25} and IC_{50} were less than 2% of the U.S. EPA ECOTOX record for similar freshwater algae suggesting that tebuconazole did not contribute to the toxicity. The same is true for imidacloprid whose concentrations were three orders of magnitude less than previously recorded in the ECOTOX Database. The results of the supplemental algal bioassay were similar with an IC_{25} of 6.7% and a LOEC of 6.25%. The propiconazole concentrations of 18.0 µg/L in the original tests and 63.3 µg/L in the supplemental bioassay are both lower (more toxic) than the ECOTOX record of 93 µg/L for *Navicula seminulum* in an 11-d EC_{50} test. Because all of these values are lower than bioassays for the neat compounds, either the toxicity observed is associated primarily with the wood extractives or there is additive or synergistic toxicity associated with the mixture of biocides plus the wood extractives. However, the nominal concentration in the supplemental bioassay (6.7%) suggests that the treated wood runoff was less toxic than the runoff from the untreated control deck (4.6%) suggesting that the three biocides acted antagonistically with the wood extractives.

Acute liquid phase Microtox test

This test was conducted at concentrations of 12.5%, 6.25%, 3.13%, 1.56%, 0.l78%, 0.39%, 0.20% and 0.00% (control) to determine the 5- and 15-min IC_{50} for light output from bioluminescent bacteria *Vibrio fischeri*. The results of this test are summarized in Table 5.56 for the control and treated decks. Similar to the other bioassays, runoff from

Table 5.55 Results of the 72-h *Selenastrum capricornutum* growth inhibition test in rainwater runoff from the untreated deck (CT1) and decks T2 and T3 constructed with Wolman AG™ pressure-treated southern pine.

Treatment and Endpoint	Nominal (%)	Tebuconazole (µg/L)	Propiconazole (µg/L)	Imidacloprid (µg/L)
Control deck (T1)				
IC50	37.0	ND	ND	ND
IC25	4.6	ND	ND	ND
Treated decks T2 & T3				
IC50	1.9	34.8	18.0	2.8
IC25	0.10	1.8	1.0	0.14
Supplemental bioassay				
IC25	6.7	85.1	66.3	3.15
U.S. EPA Ecotox LOEC		1095	93.0	10,000

Table 5.56 Results of the 5 and 15 minute bioluminescent bacterial light output inhibition test (Microtox®) using rainwater runoff from an untreated deck (T1) and decks T2 and T3 constructed with Wolman AG™ pressure-treated southern pine.

Treatment and endpoint	Nominal (%)	Tebuconazole (µg/L)	Propiconazole (µg/L)	Imidacloprid (µg/L)
Control Deck (T1)				
5 minute IC_{50}	41.0	ND	ND	ND
15 minute IC_{50}	41.6	ND	ND	ND
Treated decks T2 & T3)				
5 minute IC_{50}	11.0	201.3	104.0	16.0
15 minute IC_{50}	7.9	144.6	74.7	11.5

the untreated wood deck inhibited light output at concentrations of 41.0 and 41.6%. Light output was further reduced in runoff from the treated decks, which exhibited 5- and 15-min IC_{50}s at 11.0% and 7.9%. respectively. No Microtox data was found for these biocides in the ECOTOX Database.

5.12.2.2 Summary and conclusions

Averages of 300 µg imidacloprid/L, 3450 µg tebuconazole/L, and 3725 µg propiconazole/L were observed in the initial runoff from pre-stained hem-fir treated to a retention of 0.020 *pcf* with Wolman AG™ preservative. These initial runoff concentrations were higher by factors of two to four than the concentrations in the southern pine decking treated to 0.015 *pcf* used in the Phase 2 bioassay study. Losses of the three biocides are roughly proportional to their concentration in the preservative solution. However, note that compared with the preservative solution, proportionally more imidacloprid was lost from hem-fir. Table 5.57 describes the initial losses observed in these two studies. As seen, the losses from the southern pine at the lower retention were less than half the losses from the hem-fir emphasizing the importance of tree species and/or retention to estimating concentrations in rainwater runoff.

Toxicity of rainwater runoff from untreated southern pine

It should be noted that runoff from the untreated southern pine decking was toxic to all of the test organisms. Forty percent of the rainbow trout died in the whole effluent from the untreated deck. The *Daphnia* 21-d IC_{25} for untreated pine was 38.2% of the whole effluent. Runoff from the untreated deck caused 50% loss of *Hyalella azteca* at concentrations ≥76% of the whole effluent. A concentration of only 4.6% of the untreated deck runoff was sufficient to cause a 25% decrease in the growth rate of *Selenastrum capricornutum*. Lastly a 41% dilution of the runoff from the untreated deck resulted in a 50% reduction in light output by *Vibrio fischeri* in the Microtox™ test.

Potential synergistic, antagonistic or additive effects of the combination of biocides used in Wolman AG™

The measured concentrations of the three biocides have been useful in assessing whether their combined toxicity is synergist, additive or antagonistic. This issue is examined in Table 5.58, which compares published values for each biocide in the U.S. EPA ECOTOX Database with the results of the deck studies. It is acknowledged that there are differences in the study protocols between the ECOTOX Database data and the bioassays used in Phase 2 of this

Table 5.57 Comparison of concentrations of IMID, TEB and PROP in rainwater runoff from hem-fir decking treated to a retention of 0.020 pcf and southern pine decking treated to a retention of 0.015 pcf and exposed to natural rainfall in the Pacific Northwest. Concentration values for the Phase I study were predicted based on losses at the average (0.275 in.) accumulated rainfall collected during the Phase II studies.

	Biocide concentrations in the first flush of rainwater runoff		
Species and retention	µg imidacloprid/L	µg tebuconazole/L	µg propiconazole /L
Hem-fir (0.020)	389	5,942	5,495
Southern pine (0.015)	145	1,830	945

Table 5.58 Comparison of the toxicity of individual biocides with the mixtures reported in these Phase 2 bioassays. All values are µg biocide/L. Values for *Selenastrum* are the average for T2 and T3 supplemental algal bioassay.

	48-h LC_{50} *Daphnia*	21-d chronic *Daphnia* (IC_{50})	96-h LC_{50} rainbow trout	96-h LC_{50} *Hyalella azteca*	72-h growth inhibition (IC_{50}) *Selenastrum*
Neat tebuconazole from Exotox data	4,000	97.5	5,700	No data	1,095[a]
Tebuconazole in deck runoff at endpoint	289.3	342.2	156.4	508.7	34.8/122.6
Neat propiconazole from Ecotox data	7,250	310	253,415	No data	93.0
Propiconazole in deck runoff at endpoint	149.4	176.7	55.7	262.7	18.0/63.3
Neat imidacloprid from Ecotox data	37,656	1,250	156,415	55	10,000[a]
Imidacloprid in deck runoff at endpoint	22.9	13.0	13.9	39.2	2.8/9.7

a. The values presented in Table 5.29 were developed using *Scenedesmus subspicatus*. The algal bioassay presented in this study used *Selenastrum capricornutum*. The observed differences may be associated with the different species.

study. However, the weight of evidence in Table 5.58 suggests that there are synergistic effects when the three biocides are combined with wood extractives in runoff from treated decks. These results also suggest that wood extractives in runoff from untreated wood are a major contributor to the toxicity and there is no evidence of synergy between the three biocides used in the wood preservative. In most cases, the concentrations of contaminant at the endpoint were several orders of magnitude less than (more toxic) the ECOTOX Database record. The only instance in which a biocide concentration exceeded the ECOTOX data was for tebuconazole in the 21-d *Daphnia* LC_{50} bioassay. In that bioassay, the untreated wood runoff caused a 50% reduction in offspring at >55.7% runoff. Reproductive output was reduced 50% when *Daphnia* was raised in water containing >18.7% treated deck runoff suggesting additional toxicity. That response appears to be associated with tebuconazole.

5.13 Summary of Bioassay Results

The toxicity associated with natural wood extractives in runoff from untreated wood together with the interaction of the three bioassays suggests that the biological benchmark developed for the model could rely on the percent of runoff that is toxic regardless the cause of the effect. Mean values for the two treated decks are summarized in Table 5.59. The most sensitive species was the algae (*Selenastrum* sp.) requiring a dilution factor between 29.4 and 52.6. The mean of these values is 41.0. A 100-y storm might produce 30 cm of rain in a 72-h period. The amount of runoff from a one cm length of a 5 m wide structure would be 500 cm width*1.0 cm*30 cm rain/3 d = 45 L. The dilution water in a 1.0-cm-wide section of the receiving water flowing at a very slow current speed of 0.5 cm/s would be 20 cm mixing depth*1.0 cm*0.5 cm/s*86,400 s/d*3 d/1000 cm^3/L = 2,592 L giving dilution factor of 57.6, which exceeds all of the required dilution factors determine using these bioassays.

A second management approach is available using the literature developed WQC described earlier in this chapter and summarized in Table 5.60. This approach has several advantages in that it allows consideration of more species and environments (saltwater and freshwater). The bioassays described above demonstrate significant toxicity associated with wood extractives lost from untreated wood. The most sensitive species in the bioassays was *Selenastrum,* which suffered a 25% reduction in growth at 1.9% effluent (2.8 µg imidacloprid)/L—and a 50% growth reduction.

5.13.1 Micronized copper (Wolmanized Residential Outdoor Wood, Dispersed CA-C)

This study was conducted in 2008 to assess the initial concentrations of copper, tebuconazole and propiconazole in southern pine treated to 0.08 ± 0.0003 *pcf* with WolmanDispersed CA-C preservative. The deck design, sample collection, sample handling, analytical chemistry and bioassay protocols were identical to those described above for the Wolman AG™.

5.13.1.1 Concentration of contaminants in the first 0.5 cm of rainwater runoff from Wolman Dispersed CA-C™ preserved southern pine decks

Biocide concentrations in the runoff are summarized in Table 5.61. Statistically significant differences between the two replicate decks were not observed.

Table 5.59 Summary of nominal concentrations of first flush rainwater runoff creating toxic effects in a suite of bioassays. The values presented are the means of two Wolman AG™ preserved southern pine decks exposed to natural rainfall in the Pacific Northwest.

Bioassay	Endpoint	Percent @endpoint	Dilution required
48-h LC50 *Daphnia*	48-h LC_{50}	15.8	6.3
21-d chronic *Daphnia*	IC_{50}	18.7	5.3
21-d chronic *Daphnia*	IC_{25}	8.9	11.2
96-h LC_{50} rainbow trout	96-h LC50	8.25	12.1
96-h LC_{50} *Hyalella azteca*	96-h LC50	27.8	3.6
72-h growth inhibition with *Selenastrum*	IC_{50}	1.9	52.6
72-h growth inhibition with *Selenastrum*	IC_{25}	3.4	29.4
5-min Microtox®	IC_{50}	11.0	9.1
15-min Microtox®	IC_{50}	7.9	12.7

Table 5.60 WQC developed from previous studies.

Compound	Literature developed freshwater WQC	
	Acute	Chronic
Tebuconazole	32.0 µg/L	18.5 µg/L
Propiconazole	51.0 µg/L	9.3 µg/L
Imidacloprid	27.3[a]/0.58[b] µg/L	12.0 µg/L

a. Benchmark recommended in slow moving waters (<10 cm/sec)

b. Benchmark recommended in fast moving water (>10 cm/sec) to protect sensitive species in the Order Emphemeroptera.

Table 5.61 Copper, tebuconazole and propiconazole concentrations in rainwater runoff from two decks covered with Wolman Dispersed CA-C preservative. Each value is the mean of triplicate measurements.

Active ingredient	Deck 1	Deck 2	Composite
Copper (µg dissolved Cu/L)	1,930	1,540	1,735
Tebuconazole (µg/L)	63.6	72.9	68.2
Propiconazole (µg/L)	42.2	82.8	62.5

Table 5.62 Percent of the runoff from the control deck constructed of untreated southern pine and the two decks constructed of Wolman Dispersed CA-C preserved southern pine at each of the bioassay endpoints evaluated at the Pacific Environmental Science Center.

Bioassay	Untreated deck	Dispersed CA-C preserved decks
Daphnia 48-h LC_{50}	Not evaluated	0.84
Oncorhunchus mykiss 96-h LC_{50}	>50	3.13
Daphnia 21-d IC_{25}	27.7	0.44 and 0.48
Daphnia 21-d IC50	37.1	0.56 and 0.66
Hyalella azteca 96-h LC50	18.5	5.25 and 7.37
Selenastrum 72-h IC25	12.0	0.39 and 0.49
Selenastrum 72-h IC_{50}	48.0	0.66 and 0.85
Microtox™ 5-min IC_{50}	18.1	6.8 and 7.6
Microtox™ 15-min IC_{50}	21.2	3.0 and 2.6

5.13.1.2 Bioassay test results for Wolman Dispersed CA-C™ preserved southern pine

Bioassays were conducted at percent whole effluent concentrations of 1.563, 0.781, 0.391, 0.195, 0.098 and 0.00 (control). The percentages of runoff from the untreated and treated decks at each bioassay endpoint are summarized in Table 5.62. Similar to the Wolman AG™ bioassays, runoff from the untreated control decks, containing only natural wood extractives, was toxic. For instance, the *Selenastrum* 72-h IC_{25} occurred at 12% of the untreated deck runoff and the 96-h LC_{50} for *Hyalella azteca* occurred in water containing 18.5% of the untreated deck runoff.

Runoff from the Dispersed CA-C preserved decks was more toxic with bioassay endpoints occurring at <1.0% runoff in many cases (Table 5.63). Two values, separated by a hyphen, are provided for each of the two decks. The data in Table 5.61 and 5.63 indicate low concentrations of tebuconazole and propiconazole in the runoff. The observed concentrations are one to three orders of magnitude lower than the effects concentrations for these compounds reported earlier in this chapter. In addition, concentrations of dissolved copper were on average 25 and 28 times higher than the concentrations of tebuconazole and propiconazole respectively. In general, the two organic fungicides are less toxic to the tested species than copper and therefore, the observed toxicity is most likely attributable to the dissolved copper. Also note that the reported copper concentrations in Table 5.63 are within the range of values recorded in the U.S. EPA ECOTOX Database. These results do not suggest antagonistic, additive, or synergistic toxicity associated with the combination of wood extractives and the three biocides. Therefore, it appears that Wolman Dispersed CA-C™ preserved wood can be safely

Table 5.63 Bioassay results (percent of the whole runoff) from two decks covered with southern pine treated to a retention of 0.08 pcf with Wolman Dispersed CA-C™ preservative.

	Composite from two treated decks (µg/L)				
Bioassay	% runoff (µg/L)	Tebuconazole	Propiconazole	Dissolved Cu	Ecotox database
Daphnia 48-h LC50	0.84	0.57	1.2	16.0	26–40
Oncorhunchus mykiss 96-h LC_{50}	1.93–2.07	2.13–7.39	1.66–7.39	36.7–35.5	5.7–470
Daphnia 21-d IC_{25}	0.44–0.48	7.32–0.33	0.05–0.27	7.06–7.10	
Daphnia 21-d IC50	0.56–0.66	9.27–0.88	0.16–0.69	8.79–10.45	5.0–160
Hyalella azteca 96-h LC_{50}	5.25–7.92	4.7–14.05	5.41–7.03	88.5–128.9	
Selenastrum 72-h IC_{25}	0.39–0.49	0.25–0.36	0.42–0.40	7.60–7.23	
Selenastrum 72-h IC_{50}	0.66–0.85	0.42–0.62	0.28–0.70	12.53–12.87	64.2–280
Microtox™ 5-min IC_{50}	7.2–6.8	0.34–4.35	1.16–2.89	1.25–132.0	
Microtox™ 15-min IC_{50}	2.8–2.97	0.40–1.89	1.36–1.25	1.47–132.0	

Table 5.64 Summary of recommended benchmarks for assessing the environmental risks associated with the use of pressure-treated wood immersed in or overlying freshwater and marine environments. Metal WQC are calculated at a hardness of 60 mg $CaCO_3$/L and the pentachlorophenol criteria at pH = 6.5

Contaminant	Freshwater (µg/L)		Marine (µg/L)		Sediment (mg/kg)	
	Acute	Chronic	Acute	Chronic	Freshwater	Marine
Sum priority PAH	3.0	3.0	3.0	3.0	37.6	13.3 @ 1% TOC
Copper	10.5[a]	7.3[b]	3.1	1.9	30 – 100 See Table 5.22	390
Arsenic	360	190	69	36	20	57
Chromium(III)	361.1[c]	130.8[d]	-	-	95	260
Chromium(VI)	16.0	11.0	1,100	50.0		
Zinc	74.2[e]	67.8[f]	90	81	410	140
Pentachlorophenol	5.49[g]	3.46[h]	13	7.9	0.4 @ 1%TOC	0.360
DDAC		49		49	10	10
Tebuconazole	32	18.5	-	-	-	-
Propiconazole	51.0	9.3	6.8	-	25	-
Imidacloprid	27.3 for V_{ss} >10 cm/sed 0.58 for V_{ss} <10 cm/s	12.0	30.8	15.4	-	-

a. Copper Acute = $0.960*\exp^{0.9422[\ln(\text{hardness})] - 1.464}$
b. Copper Chronic = $0.960*\exp^{0.8545[\ln(\text{hardness})] - 1.465}$
c. Chromium(III) Acute = $0.316*\exp^{(0.8190[\ln(\text{hardness})] + 3.688)}$
d. Chromium(III) Chronic = $0.860*\exp^{(0.8190[\ln(\text{hardness})] + 1.561)}$
e. Zinc Acute = $0.978*\exp^{(0.8473[\ln(\text{hardness})] + 0.8604)}$
f. Zinc Chronic = $0.986*e^{(0.8473[\ln(\text{hardness})] + 0.7614)}$
g. Pentachlorophenol Acute = $\exp^{[1.005(\text{pH}) - 4.830]}$
h. Pentachlorophenol Chronic = $\exp^{[1.005(\text{pH}) - 5.290]}$

managed by assessing predicted copper concentrations in runoff from overhead structures treated with this preservative.

5.14 Summary of benchmarks recommended for the risk assessment process described in Chapter 9

In general, the model presented in Chapter 9 relies on WQC recommended by the U.S. EPA. Where available, recommended sediment quality criteria are those published by State governments. Regulatory WQC and SQC for DDAC, tebuconazole, propiconazole and imidacloprid were not found. U.S. EPA ECOTOX, EXTOXNET, and LanXess (2005) data were used to develop benchmarks for these compounds. No data were found describing the biological response to sedimented tebuconazole or imidacloprid and no benchmarks are provided. As an interim measure, the benchmark for DDAC of 25 mg/kg dw is recommended. However, this recommendation is not based on data. Recommended benchmarks are summarized in Table 5.64.

5.15 Additional Benchmark Recommendations

The following considerations should be made when assessing the biological response to proposed treated wood structures.

- Biological effects depend on total contaminant concentrations in water and sediments. The default background values provided in Chapter 8 can be used to estimate pre-existing contaminant concentrations outside urban and/or industrial areas. It is recommended that dissolved and sediment contaminant concentrations be determined at sites where large projects are proposed or when the contaminant concentrations are predicted to exceed 75% of the benchmarks summarized in Table 5.64.

- In waters inhabited by threatened or endangered salmonids, use of the Biotic Ligand Model is recommended when background plus project projected concentrations of dissolved copper exceed 75% of the copper WQC.
- Sediment SQC were not developed for Tebuconazole or imidacloprid. Based on their physicochemical properties, imidacloprid is expected to photodegrade in the water prior to reaching sediments. A provisional benchmark of 25 mg/kg dw has been recommended. However, additional consideration of the biological community that might be put at risk should be undertaken if sedimented tebuconazole or imidacloprid are predicted to reach 10 mg/kg (the DDAC SQC).
- The bioassays conducted to assess the overall toxicity of runoff from Wolman AG™ and Dispersed CA-C™ preserved decks did not show evidence of additive or synergistic effects between the biocides used in the preservatives. The results for Dispersed CA-C indicate that biological effects associated with this preservative can be managed by modeling the copper losses. If copper losses are maintained below protective benchmarks, then it does not appear that tebuconazole and propiconazole will contribute in an additive or synergistic way to the toxicity.
- Both studies described in Section 5.12 reported significant toxicity associated with the natural wood extractives present in rainwater runoff from untreated southern pine. The toxicity of western red cedar and other decay resistant species is well known but not reviewed herein. That toxicity is one of the reasons that State governments have historically required removal of all logging debris from surface waters.
- Several studies describing carcinogenic effects of PAH in sole and the effect of copper on olfactory responses of salmonids were reviewed in detail. The studies are interesting and provide insight into the biological response to these contaminants. However, the review suggests that care must be taken in interpreting the results of these types of studies to discriminate between effects per se and effects that jeopardize either individual organisms or populations. Too many of the published studies reviewed in this effort did not include sufficient information regarding the environment. The environment is not in homeostasis in the absence of anthropogenic inputs. The evaluation of hundreds of reference locations in pristine environments indicates that populations of benthic organisms are constantly changing in response to a multitude of biophysical parameters that affect those communities. Looking at macrobenthic communities is like looking into a kaleidoscope. It is not possible to document all of the factors influencing populations of plants and animals, in part because we haven't identified all of them. However, it would be very helpful to at least document those that we know have an influence. The Biotic Ligand Model is a step in the right direction in this regard. Secondly, bioassays conducted in tap or deionized water are useful for understanding the response of organisms under those conditions. However, these types of bioassays do not well inform us with respect to the response of organisms and populations in natural aquatic environments. Individuals and populations appear less susceptible to many contaminants than laboratory bioassays indicate and appropriate WQC and SQC should reflect responses in the real world—not in beakers or aquaria—or at least the beakers and aquaria should better mimic the real world.

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