Appendix C

Contaminants Toxic to Wildlife

Appendix C

Contaminants Toxic to Wildlife

This appendix provides additional information on the sources of critical contaminants and associated data supporting the link between contaminants and adverse biological effects.

Arsenic

Arsenic occurs in nature in various forms, including inorganic and organic compounds and trivalent and pentavalent states (Eisler 1988). Trivalent arsenic is considerably more toxic than pentavalent arsenic (Clark 2001). Most living organisms normally contain measurable concentrations of arsenic. In some marine organisms normal arsenic concentrations, except for marine species may exceed 100 milligrams per kilogram (mg/kg) dry weight, usually as arsenobetaine. Arsenobetaine, a water-soluble pentavalent organoarsenical, is very stable, metabolically inert, and nontoxic and thus poses little toxicological risk to the organism or its consumers. A variety of marine organisms, including algae, crustaceans, and fish, bioaccumulate arsenic, but arsenic does not biomagnify through food chains (Clark 2001).

Adverse effects on estuarine and marine organisms have been reported at water concentration levels of 100 micrograms per liter (µg/l) and above. The 96-hour concentration lethal to 50% of an exposed test population (LC_{50}) of arsenic trioxide for larvae of the Dungeness crab (*Cancer magister*) is 232 µg/l; the 48-hour LC_{50} of sodium arsenite for abnormal development in the Pacific oyster (*Crassostrea gigas*) is 326 µg/l. Studies of benthic fauna in Puget Sound where sediments are contaminated with more than 57 mg/kg arsenic (dry weight) have shown significant reductions in the abundance of polychaetes, mollusks, and crustaceans. Toxic effects have also been reported in marine benthos in heavily contaminated areas of San Francisco Bay where sediment concentrations were measured at 50–60 mg/kg (dry weight) (Clark 2001).

Chromium

Chromium is widespread in the environment, occurring naturally in air, rocks, soil, and water. Anthropogenic sources include industrial production of stainless

steel; electroplating of chrome; use of dyes; leather tanning; and use of wood preservatives.

Chromium occurs in several forms or oxidation states, primarily hexavalent (chromium^{VI}) and trivalent (chromium^{III}). It is a micronutrient but is toxic at high concentrations. Adverse effects are substantially influenced by a variety of biotic and abiotic factors, including the species, age, and developmental stage of the organism; the temperature, pH, salinity, and alkalinity of the medium; the effects of interactions between chromium and other contaminants; and the duration of exposure and the chemical form of chromium involved. For chromium^{VI}, the 96-hour LC_{50} values for marine species range from 445 to 2,000 parts per billion (ppb) (Note: 1 ppb = 1 microgram per kilogram [µg/kg]). For chromium^{III}, the 96-hour LC_{50} values for marine species are 3,300–7,500 ppb. Studies of chickens (*Gallus domesticus*) fed 100 ppm chromium^{VI} for 32 days showed no adverse effects on survival, growth, or feeding capacity (Rosomer et al. 1961).

Copper

Copper is an essential element for many animals, especially marine decapod crustaceans, gastropods, and cephalopods, all of which use haemocyanin, a respiratory pigment that contains copper (Clark 2001). In these species, copper is usually stored in the liver, often at very high concentrations. It has been recorded in the liver of *Octopus vulgaris* at 4,800 parts per million (ppm) and 2,000 ppm in the hepatopancreas of the lobster (*Homarus gammarus*) (Note: 1 ppb = 1 microgram per kilogram [µ/gl]). The blood cells of oysters may contain up to 20,000 ppm copper. Despite these high concentrations in the tissues of some species, copper is highly toxic to many forms of marine invertebrates; as a result, it has historically been used as an antifouling agent for ships. The 96-hour LC_{50} for mollusks and crustaceans is 28–39 µg/l, with sublethal effects occurring at 1–10 µg/l. In fish, behavior, growth, migration, and metabolism can be adversely impaired at levels of 4–10 µg/l for sensitive species (Eisler 1998). The early life stages of fish are especially vulnerable (Luoma et al. 1998).

The assessment of effects of copper contamination on organisms in nature is difficult, in part because it is difficult to isolate the effects of copper from those of other contaminants, and also because it is not well understood how toxic effects at one trophic level affect processes at other trophic levels. Moreover, direct evidence of bioaccumulation is not proof of toxicity (Hornberger et al. 1999).

Long-term studies of marine organisms exposed to chronic copper contamination have shown that natural detoxifying mechanisms may allow them to adapt, survive, and actually thrive under conditions lethal to other unexposed populations. For example, in Restronguet Creek in southwest England, copper contamination of sediments reaches 2,148 ppm, but the flora and fauna of the area are unexpectedly normal (Clark 2001). However, not all systems exposed to copper contamination show the same resilience. More locally, a long-term study

of contamination in a mudflat in Palo Alto showed extractable copper levels of 30–60 mg/kg (i.e., 30–60 ppm) and tissue concentrations in the resident clam *Macoma baltica* ranging from 100 to 300 µg/kg (0.01–0.03 ppm) (Luoma 1995). Adverse effects on the clam were indicated by reproductive anomalies, including a reduction in the percentage of reproductive clams and an inordinately high percentage of males in the population. Nonetheless, the clam population in this area was shown to be 6 times more resistant to the effects of copper contamination than populations at uncontaminated sites.

Mercury

Mercury contamination is widespread in sediments and waters of the San Francisco Bay area. Elevated mercury levels are in large part a legacy of the California gold-mining era, when mercury was used in the gold-refining process. Mines such as the south bay's New Almaden Mine, which operated for many years in the upper Guadalupe River watershed extracting the mercury ore cinnabar, are known to be a source of mercury in the bay and its tributaries. Over time, leaching of mine tailings and overland transport of mercury-bearing sediments have resulted in the downstream accumulation of mercury in the watershed (Santa Clara Valley Water District and U.S. Army Corps of Engineers 2001). Mercury is also delivered to the bay via the Sacramento–San Joaquin Delta (Delta).

In aquatic environments, most mercury is chemically bound to suspended particles of soil or sediment; a smaller fraction is bound to dissolved organic carbon. Sediment-bound mercury may be available to aquatic organisms, and is thus a pollutant of concern; the potential for adverse environmental effects from sediment-bound mercury depends primarily on transport and depositional characteristics (e.g., particle size), and on the physical and chemical properties of the sediment. Additionally, sediment-bound mercury may be converted through both biotic and abiotic processes to its more bioavailable methylated form. Factors conducive to methylation of mercury include low-flow or stagnant waters, hypoxic or anoxic conditions in the water or sediment column, low pH ($pH<6$), and high concentrations of dissolved carbon. Most of these factors are in turn affected by biological processes such as metabolism, growth, and decay; for example, mercury methylation has been linked to the activity of sulfate-reducing bacteria in the shallow anoxic sediment column (Santa Clara Valley Water District and U.S. Army Corps of Engineers 2001).

Methyl mercury is readily adsorbed by aquatic plants, fish, and wildlife. It has been demonstrated to accumulate in their tissues and to transfer through the food web as contaminated food sources (plant or animal tissues) are consumed (Santa Clara Valley Water District and U.S. Army Corps of Engineers 2001). It has a variety of toxic effects, including birth defects, embryotoxicity, carcinogenicity, and neurotoxicity. In aquatic organisms, concentrations of 0.1–200 µg/l have been shown to produce adverse effects; toxicity increases with age of the organism, exposure time, temperature, lowered salinities, and the presence of other metals.

Mercury levels and uptake rates in exposed plants and wildlife depend on the source and type of mercury to which the organisms are exposed and on the structure of the local food web (i.e., the uptake pathway). Both terrestrial and aquatic plants take up inorganic and methyl mercury from water, sediment, and the atmosphere. Uptake rate varies by species, but mercury uptake is typically higher in aquatic plants than in terrestrial plants because methyl mercury in particular is more readily taken up from water than from sediment. Aquatic organisms at lower trophic levels (e.g., algae, phytoplankton, filter-feeding invertebrates) take up mercury predominantly from dissolved concentrations in the water column. Species at higher trophic levels (such as fish) accumulate mercury from two sources: bioaccumulation from the tissues of organisms they consume and direct adsorption from the water column (Hill et al. 1996).

Fish may bioaccumulate methyl mercury by a factor of approximately 100,000 (Gilmour and Henry 1991). Mercury concentrations in fish vary by species because of differences in diet, metabolic rate, and growth rate. Fish in oceanic waters may contain as much as 150 µg/kg (0.015 ppm) mercury in muscle tissue (Eisler 1987). In general, larger, older fish have higher mercury concentrations, partly because their prey consists of larger species from higher trophic levels. Piscivorous fishes also tend to have higher mercury concentrations than species that feed primarily on algae or invertebrates (Santa Clara Valley Water District and U.S. Army Corps of Engineers 2001). Mercury is known to interfere with fish reproduction, and mercury tissue levels of 5–7 mg/kg (5–7 ppm) have been shown to be lethal (Eisler 1987).

Mercury concentrations in other species at higher trophic levels, including birds and other wildlife, are also directly correlated with diet. Factors influencing mercury levels include species-specific sensitivity, foraging area, size of fish or other prey consumed, and the percentages of fish and other food sources in the diet. Because mercury is concentrated in the muscle tissue of exposed fish, birds such as the black-crowned night heron (*Nycticorax nycticorax*), common merganser (*Mergus merganser*), cormorant (*Phalacrocorax* sp.), and belted kingfisher (*Ceryle alcyon*) that prey on fish tend to bioaccumulate more mercury than species that feed on a greater variety of foods. Species such as the clapper rail (*Rallus longirostris obsoletus*) that consume primarily invertebrates are also likely to be vulnerable to bioaccumulation of methyl mercury in the food web.

Most of the body concentration of mercury in piscivorous birds is stored in the plumage (Clark 2001). Virtually all of it is in the methylated form and is shed when the birds molt. As much as 50% of the remaining body burden of mercury is transferred to the growing feathers following molt; the plumage thus provides an important elimination pathway for methyl mercury in many birds.

The eggs of seabirds, including species of gulls, are also tolerant to mercury contamination. Whereas reproductive dysfunction has been observed in mallards (*Anas platyrhynchos*) with eggs containing 6–9 µg/g (6–9 ppm) mercury, no effects on hatching or fledging occur in eggs of herring gulls (*Larus argentatus*) contaminated with up to 16 µg/g (16 ppm) mercury.

Mercury has been shown to produce a variety of toxic and teratogenic effects in humans; the forms of mercury that pose the greatest risk for human toxicity are elemental mercury and methyl mercury. Consumption of contaminated food sources, particularly fish, is a common exposure pathway for human toxicity; consumption of some fish caught in San Francisco Bay and the Delta has been specifically identified as a human health risk (Office of Environmental Health Hazard Assessment 2001). In addition, based on concerns regarding human and environmental toxicity, the U.S. Environmental Protection Agency (EPA) classifies soils and sediments that contain more than more than 20 mg/kg total mercury as hazardous waste; 23 mg/kg total mercury represents EPA's preliminary remediation goal for mercury (Santa Clara Valley Water District and U.S. Army Corps of Engineers 2001); bay and tributary sediments may locally meet or exceed these thresholds.

Nickel

Nickel is a heavy metal that can be a significant contaminant in estuarine sediments of industrialized areas. In relatively uncontaminated areas, typical concentrations of dissolved nickel are 0.2 µg/l (0.2 ppb) in the open ocean and 14–30 µg/g (14–30 ppm) in sediments.

Nickel is regarded as only moderately toxic by comparison with other metals such as mercury, and there is no evidence that nickel is bioaccumulated or biomagnified in marine food webs (Clark 2001). Nickel toxicity varies widely with organism and environmental condition and is particularly influenced by salinity and the presence of other ions. Toxicity ranges for the 96-hour LC_{50} include 17 ppm to more than 50 ppm for polychaetes, 1,150 ppb (0.115 ppm) to 47 ppm for crustaceans, 60–320 ppm for mollusks, 30–70 ppm for estuarine fish, and 8–350 ppm for marine fish (Clark 2001). The benthic species diversity of highly industrialized areas (e.g., Massachusetts Bay, Puget Sound, and Los Angeles Harbor) has been shown to be reduced in areas where nickel concentrations in sediments reach levels of 20–30 µg/g (20–30 ppm) (Clark 2001).

Polycyclic Aromatic Hydrocarbons

There are thousands of polycyclic aromatic hydrocarbon (PAH) compounds, each consisting of hydrogen and carbon arranged in the form of two or more fused benzene rings. PAH compounds differ in the number and position of aromatic rings and in the position of substituents on the basic ring system. Environmental concern has focused on 2-ring (naphthalene) to 7-ring (coronene) structures. Unsubstituted 2–3-ring PAHs exhibit significant toxicity, but are noncarcinogenic; 4–7-ring PAHs are significantly less toxic, but are demonstrably carcinogenic, mutagenic, or teratogenic to a large variety of organisms, including fish and birds.

In general, PAHs show little tendency to biomagnify in food chains, primarily because they are rapidly metabolized. Accumulation in aquatic organisms is largely related to the ability to metabolize the compounds. Species at lower trophic levels, such as plankton and mollusks, are unable to effectively metabolize and excrete the compounds, which therefore accumulate in their tissues. Organisms at higher trophic levels, including fish and crustaceans, only accumulate PAHs in highly contaminated areas.

Toxicity levels for larval and juvenile fish may be as low as 0.1–5 µg/l for sublethal effects (National Oceanic and Atmospheric Administration 1999). A wide variety of adverse biological effects linked with PAH contamination have been reported in numerous species of organisms under laboratory conditions. These include the formation of tumors, as well as effects on survival, growth, and metabolism. Responses to carcinogenic PAHs are quite variable between species, and can be significantly modified by many chemicals, including other PAHs. These complexities render attempts at extrapolating laboratory results to field situations extremely difficult and unreliable.

Polychlorinated Biphenyls

Polychlorinated biphenyls (PCBs) have been linked with a variety of adverse effects in aquatic organisms, including birth defects, reproductive failure, liver damage, mutation, carcinogenicity, wasting syndrome, and death. PCBs both bioaccumulate and biomagnify in natural food chains; bioconcentration from water has been documented at a factor of 10,000 to 100,000. The most significant levels of PCBs and the greatest potential for adverse effects on estuarine organisms are therefore likely to occur at higher trophic levels, primarily in invertebrate- and fish-eating birds (Zaranke et al. 1997). Fish-eating birds such as cormorants have been found to accumulate PCBs to high levels in their tissues with consequential adverse effects including reproductive failure, kidney/liver/heart damage, tremors, beak deterioration, loss of muscle coordination, behavior alterations, and eggshell thinning. Comparable effects are likely to occur in other birds that forage largely on marine invertebrates such as rails and various species of short-and long-legged waders. Because PCBs bind to particulate material and do not release directly into the water column, their bioavailability is linked to the level of organic matter and suspended sediment in the water column.

Chlorinated Hydrocarbons

Chlorinated hydrocarbons such as dichlorodiphenyltrichloroethane (DDT) are highly insoluble in water, with an aqueous saturation concentration of less than 1 ppb, but they are soluble in fats and may adsorb to particles. The distribution and bioavailability of these compounds in estuarine systems is largely associated with the presence of suspended sediments and microorganisms such as diatoms. Thus, filter-feeding organisms that directly or indirectly ingest sediment particles and

microorganisms, and organisms that prey on these first-order consumers, therefore form a primary route for accumulation and transfer of DDTs in both marine and terrestrial food chains. Because DDTs are difficult to excrete and are lipid-soluble, they tend to accumulate and concentrate in the fatty tissues of receptor organisms, leading to bioaccumulation and biomagnification through the food web.

Because organochlorines are stored in fatty tissues, they become biologically available and physiologically active only when fat tissues are metabolized. Receptors may therefore acquire considerable body burden of DDTs but show no ill effects except during conditions of starvation, when fat reserves are mobilized. Accordingly, it is difficult to be precise about the potential effect of these contaminants based on tissue concentrations only.

The highest concentration factors for DDT (~70,000) have been documented in bivalve mollusks such as oysters and clams. Accumulation factors for crustaceans and fish range from 100 to 10,000; those for seabirds are less than 10. Laboratory studies of phytoplankton show that DDT can reduce primary production as much as 50% at concentrations of only 1 µg/l (1 ppb). The 96-hour LC_{50} values for shrimp, including *Cragon* sp. and *Palaemonetes* sp., are 6–60 µg/l for DDT. Marine fish also appear to be very sensitive to a number of organochlorines, including DDT; the 96-hour LC_{50} for marine fish ranges from 0.4 µg/l to 89 µg/l.

Very large doses of DDT (on the order of several grams per kilogram of body weight) are required to cause death in birds and mammals. One critical sublethal effect of DDT and its residues on birds is interference with calcium metabolism, resulting in thinning of eggshells. During its use as a pesticide, bioaccumulation of DDT led to significant population declines of several top-level avian predator species, including peregrine falcon (*Falco peregrinus*), osprey (*Pandion haliaetus*), bald eagle (*Haliaeetus leucocephalus*), and brown pelican (*Pelecanus occidentalis*) (Anderson et al. 1975, Garcelon et al. 1989, Clark 2001). Contamination by DDTs has also been historically linked with reproductive problems in several predatory marine animals in California, including premature pupping in California sea lions (*Zalophus californianus californianus*) (DeLong et al. 1973, Gilmartin et al. 1976).

Tributylin

Tributylin (TBT) is an organotin biocide used in ship antifoulant paints. Like PCBs, pesticides, and a variety of other organic contaminants, TBTs are known to have adverse effects on marine life; as endocrine inhibitors, TBTs interfere with the normal hormonal processes of receptor organisms. They have been found to cause deformation in oysters and sex changes in whelks, and are also widely believed to cause immune suppression and increased susceptibility to lethal diseases in seals, sea lions, and sea otters (see Kannan et al. 1999, Nakata et al. 1998, Kajiwara et al. 2001).

References Cited

Printed References

Anderson, D. W., J. R. Jehl Jr., R. W. Risebrough, L. A. Woods Jr., L. R. DeWeese, W. G. Edgecomb. 1975. Brown pelicans: improved reproduction off the southern California coast. *Science* 1980:806–808.

Clark, R. B. 2001. *Marine pollution.* 5th Edition. Oxford, England: Oxford University Press.

DeLong, R. L., W. G. Gilmartin, and H. G. Simpson. 1973. Premature births in California sea lions: associated with high organochloring pollutant concentrations. *Science* 181:1168–1170.

Eisler, R. 1987. *Mercury hazards to fish, wildlife, and invertebrates: a synoptic review.* Biological Report 85(1.10) Contaminant Hazard Reviews, Report No. 10. Laurel, MD: U.S. Fish and Wildlife Service, Patuxtent Wildlife Research Center.

————. 1988. *Arsenic hazards to fish, wildlife, and invertebrates: a synoptic review.* Biological Report 85 (1.12) Contaminant Hazard Reviews, Report No. 12 (January). Laurel, MD: U.S. Fish and Wildlife Service, Patuxtent Wildlife Research Center.

Garcelon, D. K., R. W. Risebrough, W. M. Jarman, A. B. Chartrand, and E. E. Littrell. 1989. *Accumulation of DDE by bald eagles* (Haliaeetus leucocephalus) *reintroduced to Santa Catalina Island in southern California.* In B.U. Mayberg and R. D. Chancellor (eds.), Raptors in the modern world. Berlin: WWGBP.

Gilmartin, W. G., R. L. DeLong, A. W. Smith, J. C. Sweeney, B. W. DeLappe, R. W. Risebrough, L. A. Griner, M. D. Dailey, and D. B. Peakall. 1976. Premature parturition in the California sea lion. *J. Wildlife Dis*. 12:104–115.

Gilmour, C. C., and E. A. Henry. 1991. Mercury methylation in aquatic systems affected by acid deposition. *Environmental Pollution* 71:131.

Hill, W. R., A. J. Stewart, and G. E. Napolitano. 1996. Mercury speciation and bioaccumulation in lotic primary produces and primary consumers. *Canadian Journal of Fisheries and Aquatic Sciences* 53:812–819.

Hornberger, M. I., S. N. Luoma, D. J. Cain, F. Parschaso, C. Brown, R. Bouse, C. Wellise, and J. Thompson. 1999. *Bioaccumulation of metals by the bivalve* macoma baltica *at a site in South San Francisco Bay between 1997 and 1999: U.S. geological survey open-file report 99-55,* 42 p.

Kajiwara, N., K. Kannan, M. Muraoka, M. Watanabe, S. Takahashi, F. Gulland, H. Olsen, A. L. Blakenship, P. D. Jones, S. Tanabe, and J. P. Giesy. 2001. Organochlorine pesticides, polychlorinated biphenyls, and butylin compounds in blubber and livers of stranded California sea lions, elephant seals, and harbor seals from coastal California, USA. *Arch. Environmental Contamination and Toxicology* 41:90–99.

Kannan, K., K. S. Guruge, N. J. Thomas, S. Tanabe, and J. Giesy. 1999. Butylin residues in southern sea otters *(Enhydra lutris nereis)* found dead along California coastal waters. *Environmental Science Technology* 32:1169-1175.

Luoma, S. N. 1995. Prediction of metal toxicity in nature from bioassays: limitations and research needs. In: *Metal Speciation and Bioavailability in Aquatic Systems*. A. Tessier and D. R. Turner (eds.). Chichester, UK: John Wiley and Sons, pp. 609–659.

Luoma, S. N. Steinert, R. Streib-Montee, and P. Weiss. 1998. *The effects of copper on marine organisms.* In P. R. Seligman and A. Zirino (eds.), Chemistry, toxicity, and bioavailability of copper and its relationship to regulation in the marine environment. Technical document 3044. November. San Diego, CA: Office of Naval Research, Space and Naval Warfare Systems Center.

Nakata, H., K. Kannan, L. Jing, N. Thomas, S. Tanabe, and J. P. Geisy. 1998. Accumulation pattern of organochlorine pesticides and polychlorinated biphenyls in southern sea otters *(Enhydra lutris neris)* found stranded along coastal California, USA. *Environmental Pollution* 103:45–53.

Rosomer, G. L., W. A. Dudley, L. J. Machlin, and L. Loveless. 1961. Toxicity of vanadium and chromium for the growing chick. *Poultry Science* 40:1171–1173.

Santa Clara Valley Water District and U.S. Army Corps of Engineers. 2001. *Environmental impact report/environmental impact statement: Guadalupe Creek restoration project from Alamaden Expressway to Masson Dam, San Jose, California.* Prepared by Jones & Stokes. San Jose, CA.

Zaranke, D. T., R. W. Griffiths, and N. K. Kaushik. 1997. Biomagnification of polychlorinated biphenyls through a riverine food web. *Environ. Toxicol. Chem.*16:1463–1471.

Websites

Agency for Toxic Substances and Disease Registry. 2001. ToxFacts for mercury. Available: <http://www.atsdr.cdc.gov/tfacts46.html>. Accessed: December 2001.

National Oceanic and Atmospheric Administration. 1999. Report of the San Francisco Airport science panel. Available: <http://www.bcdc.ca.gov/airports/NOAAPANEL/noaapanel.htm>. Accessed: November 2001.

Office of Environmental Health Hazard Assessment Web site. 2001. Available: <http://www.oehha.ca.gov>. Accessed: November 2001.

Table of Contents

List of Acronyms

List of Citations

List of Figures
Error! No table of figures entries found.

List of Tables
Error! No table of figures entries found.

Miscellaneous
Error! No table of figures entries found.