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Toxicity Profiles: a brief discussion of the general fate and transport processes associated with selected groups of COPECs.

Inorganics					
Aluminum	Arsenic	Barium	Cadmium	Chromium	Copper
Cyanide	Lead	Manganese	Mercury	Nickel	Selenium
Silver	Thallium	Zinc			
Organics					
Pesticides	PAHs	PCBs	Dioxins		

INORGANICS

Heavy metals and other inorganic compounds are naturally-occurring in the environment, and in some cases are essential nutrients (i.e., calcium, magnesium, potassium, and sodium). Inorganics tend to adsorb strongly to clays, muds, humic, and organic materials. However, inorganics are very mobile in the environment. Depending upon the pH, hardness, salinity, oxidation state of the element, soil saturation, and other factors, inorganics are readily soluble.

Aluminum

Toxicity information about aluminum is generally lacking. It has been determined that fish tend to be more sensitive to aluminum toxicity than aquatic invertebrates (Sparling et al. 1997). Aluminum can cause pulmonary and developmental problems. Aluminum toxicity has been linked to soil pH--the amount of soluble aluminum, rather than the total aluminum concentration in the soil. Soils at a site with a pH greater than 5.5 can generally be considered non-toxic in terms of aluminum.

Arsenic

In plants, arsenic has been shown to cause wilting, chlorosis, browning, dehydration, mortality, and inhibition of light activation (Eisler 1988a). Arsenic is a carcinogen (cancer-causing), teratogen, and possible mutagen (causing mutations in genes/DNA) in mammals (ATSDR 1993). Chronic exposure can result in fatigue, gastrointestinal distress, anemia, neuropathy, and skin lesions that can develop into skin cancer in mammals. It can cause death in soil microbiota and earthworms. Cancer-causing and genetic mutation-causing effects occur in aquatic organisms, with those effects including behavioral impairments, growth reduction, appetite loss, and metabolic failure. Aquatic bottom feeders are more susceptible to arsenic. In birds tolerance to arsenic varies among species, but effects include destruction of gut blood vessels, blood-cell damage, muscular incoordination, debility, slowness, jerkiness, falling, hyperactivity, fluffed feathers, drooped eyelids, immobility, seizures, and systemic, growth, behavioral, and reproductive problems (Stanley et al. 1994; Whitworth et al. 1991; Camardese et al. 1990).

Barium

Elevated levels of barium can induce a wide range of effects in mammals including gastrointestinal distress, muscular paralysis, and cardiovascular effects. Barium is does not

bioaccumulate, and concentrations in higher species rarely exceed 10 mg/kg (Moore 1991).

Cadmium

Cadmium is highly toxic to wildlife; it is cancer-causing and teratogenic and potentially mutation-causing, with severe sublethal and lethal effects at low environmental concentrations (Eisler 1985a). It is associated with increased mortality, and it effects respiratory functions, enzyme levels, muscle contractions, growth reduction, and reproduction. It bioaccumulates at all trophic levels, accumulating in the livers and kidneys of fish (Sindayigaya, et al. 1994; Sadiq 1992). Crustaceans appear to be more sensitive to cadmium than fish and mollusks (Sadiq 1992). Cadmium can be toxic to plants at lower soil concentrations than other heavy metals and is more readily taken up than other metals (EPA 1981). On the other hand, some insects can accumulate high levels of cadmium without adverse effects (Jamil and Hussain 1992).

Chromium

There is no significant biomagnification of chromium in aquatic food webs (ATSDR, 1993). However, there are a wide range of adverse effects in aquatic organisms. In benthic invertebrates there has been observed reduced fecundity and survival, growth inhibition, and abnormal movement patterns (U.S. EPA 1980b). Fish experienced reduced growth, chromosomal aberrations, reduced disease resistance, and morphological changes.

The toxic effects of chromium are primarily found at the lower trophic levels. The main potential ecological impacts result from direct exposure of algae, benthic invertebrates, and embryos and fingerlings of freshwater fish and amphibians to chromium. Chromium may bioaccumulate in algae, other aquatic vegetation, and invertebrates, but it does not biomagnify. Chromium inhibits growth in duckweed and algae, reduces fecundity and survival of benthic invertebrates, and reduces growth of freshwater fingerlings. It is cancer-causing, mutation-causing, and teratogenic. Chromium exists in two oxidation states in the environment: trivalent (+3) and hexavalent (+6), the latter of which is more toxic. Chromium+6 is readily converted to chromium+3 in animals, which appears to protect higher organisms from the effects of low level exposures (Eisler 1986).

Copper

Copper is a micronutrient and toxin. It strongly adsorbs to organic matter, carbonates and clay, which reduces its bioavailability. Copper is highly toxic in aquatic environments and has effects in fish, invertebrates, and amphibians, with all three groups equally sensitive to chronic toxicity (U.S. EPA 1993; Horne and Dunson 1995). Copper is highly toxic to amphibians (including mortality and sodium loss), with adverse effects in tadpoles and embryos (Horne and Dunson 1995; Owen 1981). Copper will bioconcentrate in many different organs in fish and mollusks (Owen 1981). There is low potential for bioconcentration in fish, but high potential in mollusks. Copper sulfate and other copper compounds are effective algaecides (free copper ions are the lethal agent). Single-cell and filamentous algae and cyanobacteria are particularly susceptible to the acute effects, which include reductions in photosynthesis and growth, loss of photosynthetic pigments, disruption of potassium regulation, and mortality. Sensitive algae may be affected by free copper at low (parts per billion) ppb concentrations in freshwater.

There is a moderate potential for bioaccumulation in plants and no biomagnification.Toxic effects in birds include reduced growth rates, lowered egg production, and developmental abnormalities. While mammals are not as sensitive to copper toxicity as aquatic organisms, toxicity in mammals includes a wide range of animals and effects such as liver cirrhosis, necrosis in kidneys and the brain, gastrointestinal distress, lesions, low blood pressure, and fetal mortality. (ATSDR 1990c; Kabata-Pendias and Pendias 1992; Ware 1983; Vymazal 1995).

Cyanide

Cyanide toxicity is caused by the free cyanides (HCN and CN-) that inhibit cytochrome oxidase and thereby suppress aerobic respiration. Fish are the most susceptible organisms - sensitive species exhibit chronic and lethal effects at as low as 5 to 7 grams/liter (chronic)

and 20 to 76 grams/liter (lethal), respectively. The toxicity of complex cyanides is usually, but not always, low, but the degradation products often include free cyanides which are toxic. Free cyanides readily degrade in the open environment but persist in groundwater. They do not bioaccumulate. Sublethal effects in fish include reduced reproductive capacity (decreased egg number and viability, and reduced embryo and larval survival), impaired swimming ability, altered growth, and hepatic necrosis. Free cyanides are phytotoxic at higher concentrations than those associated with adverse effects in fish. Mammals are less sensitive than fish, and are relatively tolerant of intermittent sublethal exposures (Eisler 1991).

Lead

Lead is cancer-causing, and adversely effects reproduction, liver and thyroid function, and disease resistance (Eisler 1988b). The main potential ecological impacts of wetland contaminants result from direct exposure of algae, benthic invertebrates, and embryos and fingerlings of freshwater fish and amphibians to lead. It can be bioconcentrated from water, but does not bioaccumulate and tends to decrease with increasing trophic levels in freshwater habitats (Wong et al. 1978; Eisler 1988b). Lead adversely affects algae, invertebrates, and fish. There are also limited adverse effects in amphibians, including loss of sodium, reduced learning capability, and developmental problems (Horne and Dunson 1995; Freda 1991). Fish exposed to high levels of lead exhibit a wide-range of effects including muscular and neurological degeneration and destruction, growth inhibition, mortality, reproductive problems, and paralysis (Eisler 1988b; EPA 1976). Lead adversely affects invertebrate reproduction; algal growth is affected. Lead partitions primarily to sediments, but becomes more bioavailable under low pH, hardness and organic matter content (among other factors). Lead bioaccumulates in algae, macrophytes and benthic organisms, but the inorganic forms of lead do not biomagnify.

At elevated levels in plants, lead can cause reduced growth, photosynthesis, mitosis, and water absorption (Eisler 1988b). Birds and mammals suffer effects from lead poisoning such as damage to the nervous system, kidneys, liver, sterility, growth inhibition, developmental retardation, and detrimental effects in blood (Eisler 1988b; Amdur et al. 1991).
Lead poisoning in higher organisms has been associated with lead shot and organolead compounds, but not with food chain exposure to inorganic lead (other than lead shot, sinkers or paint) (Eisler 1988b). There are complex interactions with other contaminants and diet. Lead poisoning in higher organisms primarily affects hematologic and neurologic processes.

Manganese

Elevated levels of manganese in birds have been shown to cause the following effects: decreased hemoglobin, anemia, reduced growth; in mammals, effects include alterations of brain chemicals, gastric irritation, delayed testicular development, low birth weights, behavioral changes, and muscular weakness (ATSDR 1991).

Mercury

Mercury is a mutagen (mutation-causing), teratogen, and carcinogen (cancer-causing), with toxicity and environmental effects varying with the form of mercury, dose, and route of ingestion, and with the exposed organism's species, sex, age, and general condition (Eisler, 1987a, Fimreite 1979). There is a high potential for bioaccumulation and biomagnification with mercury, with biomagnified concentrations reported in fish up to 100,000 times the ambient water concentrations (Eisler 1987a, Callahan et al. 1979). Methylmercury is the most toxic form. Inorganic mercury is methylated primarily by bacteria in both anaerobic and aerobic environments. The organic mercury compounds are more readily absorbed and poorly excreted in comparison with inorganic forms. The primary targets of acute exposures are the central nervous system and kidneys in fish, birds and mammals.

In invertebrates, effects range from non-observable to chromosomal abnormalities in some flies and reduced segment regeneration in worms (Eisler 1987a). Mercury can inhibit frog metamorphosis and has many effects in fish. In water, at concentrations at or well below even 1 ppb (part-per-billion), mercury can cause effects including: loss of appetite, brain lesions, cataracts, abnormal motor coordination, and behavioral changes (MacDonald 1993). There are also effects on reproduction, growth, behavior, metabolism, blood chemistry,

osmoregulation, and oxygen exchange at relatively low concentrations of mercury (Eisler 1987a). Juveniles are commonly more susceptible than adults.

Upper trophic level fish, birds and mammals are particularly vulnerable because of the pronounced biomagnification of organomercury (Eisler 1987a). There are numerous effects in birds, including delayed testicular development, altered mating behavior, reduced fertility, reduced survivability and growth in young, and gonadal atresia. In mammals, it has been shown that mercury can cause ataxia, aphagia, tremors, and diminished movement coordination (ATSDR 1994). There are varied neurological and reproductive effects as well (Cagiano et al. 1990; Khera et al. 1973).

Nickel

Nickel is cancer-causing (carcinogen) and mutation-causing (mutagen). Some observed effects of nickel in aquatic environments include tissue damage, genotoxicity, and growth reduction (Environment Canada 1994a). Mollusks and crustaceans are more sensitive than other organisms.

Selenium

Selenium undergoes bioconcentration, bioaccumulation, and biomagnification as trophic levels increase (Taylor et al., 1992). It can enter the food web through both sediments and surface water. Elevated levels cause growth reduction in green algae (Eisler 1985b). In other aquatic organisms, the following adverse effects have been observed: loss of equilibrium and other neurological disorders, liver damage, reproductive failure, reduced growth, reduced movement rate, chromosomal aberrations, reduced hemoglobin and increased white blood cell count, and necrosis of the ovaries.

Silver

Silver may biomagnify in some aquatic invertebrates (Adriano 1986). However, it is highly toxic to aquatic organisms (EPA 1992). Elevated concentrations can cause larval mortality, developmental abnormalities, and reduced larval growth in fish (Klein-MacPhee et al. 1984); growth reduction in juvenile mussels (Calabrese et al. 1984); and adverse effects on reproduction in gastropods (Nelson et al. 1983). There are some indications of toxicity in plants. However, there are other reports suggesting that silver is not highly phytotoxic. Silver is toxic to soil microbes, thus inhibiting biotransformation (ATSDR 1990b). Effects on mammals include pulmonary edema, congestion, and mortality.

Thallium

Low rates of bioconcentration may occur in aquatic systems and thallium may be as toxic as copper on a weight basis (Zitko et al. 1975). Thallium can cause reductions in larval fish growth and percent embryo hatchability and mortality (LeBlanc and Dean 1984).

Zinc

In many types of aquatic plants and animals, growth, survival, and reproduction can all be adversely affected by elevated zinc levels (Eisler 1993). Zinc in aquatic systems tends to be partitioned into sediment and less frequently dissolved as hydrated zinc ions and organic and inorganic complexes (MacDonald 1993). Zinc is toxic to plants at elevated levels, causing adverse effects on growth, survival, and reproduction (Eisler 1993). Terrestrial invertebrates show sensitivity to elevated zinc levels, with reduced survival, growth, and reproduction. Elevated zinc levels can cause mortality, pancreatic degradation, reduced growth, and decreased weight gain in birds (Eisler 1993; NAS 1980); and elevated zinc can cause a wide range of problems in mammals including: cardiovascular, developmental, immunological, liver and kidney problems, neurological, hematological (blood problems), pancreatic, and reproductive (Eisler 1993; Domingo 1994).

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ORGANICS

POLYCHLORINATED BIPHENYLS (PCBs)

PCBs are mutation-causing, cancer-causing, and teratogenic. They are readily absorbed through the gut, respiratory system, and skin in mammals and will concentrate in the liver, blood, muscle, adipose (fatty) tissue, and skin (Eisler 1986). Mutation tends to decrease with increasing chlorination (U.S. EPA 1980).

In general, in aquatic systems, increased toxicity is associated with increasing exposure, younger developmental stages, crustaceans, and lower chlorinated biphenyls (Eisler 1986). An increase in somatic (non-reproductive cell) mutations have been observed in ostrich ferns (Matteuccia struthiopteris).

Toxic effects in avian species included the following: morbidity, tremors, upward pointing beaks, muscular incoordination, and hemorrhagic areas in the liver (Eisler 1986). Other sublethal effects include delayed reproduction and chromosomal aberrations in Ringed Turtle-doves; courtship and nestbuilding behavioral impairments in Mourning Doves; reduced hatchability in chicken eggs; and decline in sperm concentration in American Kestrels. However, birds tend to more resistant to acute exposure than other groups: no adverse reproductive effects were observed in Screech Owls fed 3 ppm PCB diets or in Japanese Quail, Northern Bobwhites, and Mallard Ducks.

Mink are very sensitive to PCBs, with concentrations as low as 0.1 mg/kg dietary fresh weight producing an LD-50 (lethal dose - 50: the level of contamination at which 50% of the sample population dies) in 3 months and complete reproductive inhibition among the survivors (Eisler 1986). Other effects include anorexia, weight loss, and lethargy. On the other hand, compared to mink, the European ferret is highly resistant to PCBs. Rhesus monkeys are extremely sensitive to PCBs, with an increase in stillborns and abortions, lowered birth rates, hyperpigmentation, skin eruptions, eye problems, and altered behavioral patterns.

There are a number of effects observed in aquatic organisms due to exposure to PCBs (Eisler 1986). Those effects include growth reduction in algae and brook trout; reduced egg survival and reduced fertilization success in flounder, minnows, sea urchins (prior to fertilization, eggs were more resistant to PCBs at insemination and afterwards); and complete reproductive failure in brook trout. Cancer-causing effects and biochemical perturbations were observed in trout liver cells and marine fishes; with anemia, hyperglycemia, and altered cholesterol metabolism in brown trout fed diets with 10 PPM PCBs (U.S. EPA 1980).

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PESTICIDES

DDE is bioavailable to soil invertebrates and plants and has been shown to bioaccumulate in some grains (Verma and Pillai 1991). It is also very persistent in aquatic systems, absorbing strongly to sediments, and bioconcentrating in aquatic organisms, including fish and other organisms (HSDB 1997). DDE tends to bioconcentrate in lower-trophic levels and will accumulate in food webs. DDT is toxic to many types of aquatic organisms, even at low concentrations.

Heptachlor and gamma-BHC are both highly toxic to aquatic invertebrates (EPA 1980). However, although fish are less susceptible to heptachlor than invertebrates, gamma-BHC is highly toxic to fishes as well. Birds show a wide range of susceptibility to pesticides: dieldrin (less toxic than in aquatic organisms); heptachlor (moderately to highly toxic); gamma-BHC (slightly to moderately toxic); DDT (slightly to non-toxic). However, DDT causes eggshell thinning and embryo mortality, especially in predatory birds. There is also courtship behavior changes and other reproductive impairments (EXOTOXNET 1996).

DDT and its metabolite DDE are highly persistent and lipophilic compounds subject to pronounced biomagnification. The extremely low water solubilities result in strong adsorption to soil particles and very low leaching losses. The main physical causes of attenuation of soil DDT are volatilization (estimated half-life of 100 days) and erosion. Microbes biodegrade DDT to DDE and DDD under aerobic (with oxygen) and anaerobic (without oxygen) conditions, respectively. Both metabolites are more persistent than DDT. Plants absorb DDT and its metabolites from soil, but they are poorly translocated and remain primarily in the roots. The eating of leaves is therefore not a significant route of exposure to soil DDT. The toxicity of DDT to earthworms is low (Edwards and Bohlen 1992), so bioaccumulation by earthworms is a significant route of exposure to vermivores and can result in lethal doses (Barker 1958).

DDT and DDE are moderately toxic to rodents with oral LD50s in the low to high 100s mg/kg bw. However, DDT is a cumulative poison, and the chronic lethal dose may be substantially less than the acute lethal dose, for example, the chronic minimum lethal dose in mallards is 50 mg/kg bw/day, only 2% of the acute LD50 (Tucker and Crabtree 1970). DDT is a neurotoxin that affects the central nervous system by increasing neurotransmitter release, disrupting neural ion regulation, and inhibiting neural ATPase activities. Symptoms include excitability, tremors, convulsions and death. Chronic effects are of greater significance for higher organisms than acute effects. DDT adversely affects avian and mammalian reproduction by eggshell thinning, infertility, and embryo- and fetotoxicity. The effects may be related to the estrogenic activity of o,p'-DDT and o,p'-DDE. DDT and DDE are also cancer-causing in rodents (ATSDR 1994b).

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DIOXINS

The most toxic of the chlorinated dioxin isomers is 2,3,7,8-TCDD (Eisler 1986c). It has been associated with lethal, cancer-causing, teratogenic, reproductive, mutation-causing, tissue damaging, and immunotoxic effects. In fish, the following effects were observed: reduced growth, fin necrosis, death, declining interest in feeding (5-8 days postexposure), skin discoloration, reduced resistance to fungal infestations, reduced swimming, teratogenesis, tissue damage, degeneration and necrosis of the liver in fry, and opercular defects in fry. In general, older and larger fish die last; and smaller or younger specimens succumb first. Bioaccumulation does occur in fish. Among fish, body burdens of 2,3,7,8-TCDD increased with increasing concentration in the water column and with increasing duration of exposure; on removal to uncontaminated water, less than 50% was lost in 109 days.

Invertebrates, plants, and amphibians were comparatively resistant to 2,3,7,8-TCDD.

Birds exhibited the following effects from dioxins (Eisler 1986c): death, enlarged livers, severe emaciation, high accumulations of uric acid salts in connective tissues, and fluid accumulations in the pericardial and abdominal cavities, excessive drinking, loss of appetite, hypoactivity, emaciation, weakness, debility, muscular incoordination, increased reaction to stimuli, fluffed feathers, huddled position, unkempt appearance, falling, tremors, spasms, convulsions, necrosis, fatty degeneration, and immobility. Birds may bioaccumulate from fish prey, but dioxins do not appear to biomagnify.

In mammals, poisoning by 2,3,7,8-TCDD is typically characterized by loss of body weight and delayed lethality; large interspecies differences exist in lethal dosages and toxic effects (Eisler 1986c). For example, 2,3,7,8-TCDD produces prominent chloracne-type skin lesions in humans and monkeys, and severe liver damage in rats, mice, and rabbits. Other effects include tissue damage, atrophy of the thymus, edema, hemorrhagic tracheitis, pleural hemorrhage, and dystrophic lesions of the liver, skin hyperkeratosis, gastric ulcers, and lung and kidney lesions, teratogenesis, carcinogenesis (in the liver, pharnyx, lungs, skin, and thyroid), and fetotoxicity. Suppression of thymus-dependent cellular immunity, particularly in young animals, may contribute to their death. Developing mammalian fetuses are especially sensitive to 2,3,7,8-TCDD, and maternal exposure results in increased frequencies of stillbirths. Among live births, exposure to 2,3,7,8-TCDD produces teratogenic effects such as cystic kidney, cleft palate, and spinal column deformities; dioxin poisoning also produces decreased litter size at birth, increased number of stillborns, and reduced survival and growth of young in both the Fl and F2 generations (first and second generations of offspring--e.g., children and grandchildren of animals exposed to a chemical). Higher dose level in monkeys for extended periods (i.e., 500 ppt in diets equivalent to about 0.011 ug/kg body weight daily for 9 months) caused death (63%) or, among survivors, abortion, chloracne, nail loss, scaly and dry skin, and progressive weakness. Most treated monkeys remained fairly alert to external stimuli until just prior to death. On removal from the 500 ppt 2,3,7,8-TCDD diet and transfer to an uncontaminated diet, a severely affected monkey became pregnant and gave birth to a well-developed infant after an uneventful gestation. This suggests that some 2,3,7,8-TCDD damaging effects are not permanent.

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POLYCYCLIC AROMATIC HYDROCARBONS (PAHs)

PAHs are highly potent carcinogens that can produce tumors in some organisms at even single

doses; but other non-cancer-causing effects are not well understood (Eisler 1987b). Their effects are wide-ranging within an organism and have been found in many types of organisms, including non-human mammals, birds, invertebrates, plants, amphibians, fish, and humans. However, their effects are varied and so generalizations cannot be readily made. It has been shown that the fungus Cunninghamella elegans can inhibit the mutation-causing properties of various PAHs, including: benzo(a)pyrene and benzo(a)anthracene. Effects on benthic invertebrates include inhibited reproduction, delayed emergence, sediment avoidance, and mortality. Fish exposed to PAH contamination have exhibited fin erosion, liver abnormalities, cataracts, and immune system impairments leading to increased susceptibility to disease (Fabacher et al. 1991; Weeks and Warinner 1984; 1986; O'Conner and Huggett 1988).

Mammals can absorb PAHs by inhalation, dermal contact, or (more poorly) ingestion (Eisler 1987b). Plants can absorb PAHs from soils through their roots, and translocated them to other plant parts such as developing shoots. Uptake rates are generally governed by PAH concentration, PAH water solubility, soil type, and PAH physicochemical state (vapor or particulate). Lower molecular weight PAHs are absorbed more readily than higher molecular weight PAHs. PAH-induced phytotoxic effects are rare, howerver the database on this is limited. Some higher plants can catabolize PAHs, but this metabolic pathway is not well defined. Certain plants contain substances that can protect against PAH effects, inactivating their cancer-causing and mutation-causing potential. Additionally, PAHs synthesized by plants may act as growth hormones.

In aquatic systems, PAHs tend towards increased toxicity with increased molecular weight (Eisler 1987b). In addition, although the rate of uptake from the environment is variable among species, bioaccumulation tends to be rapid.

Adsorption of PAHs in soil is directly proportional to soil organic matter (OM) content and the Kow of the PAH (greater in high molecular weight (HMW) PAHs than in low molecular weight (LMW) PAHs, and is inversely proportional to soil particle size (roughly 2 orders of magnitude greater on silts and clays as compared with sands). LMW PAHs have higher volatilization rates and are more readily leached as compared with HMW PAHs. Both LMW and HMW PAHs are microbially degraded, but the rates are higher for the former probably because of weaker adsorption and greater bioavailability. Examples of soil half-lives are approximately 100-200 and 300-500 days for LMW and HMW PAHs, respectively; however, they will be longer in hazardous waste sites toxic to bacteria. Plants absorb PAHs from soil, especially LMW PAHs, and readily translocate them to above-ground tissues. The concentrations in plants are substantially lower than in soil, and they are poorly correlated because of deposition and absorption of atmospheric PAHs. Eating of leaves (foliar herbivory) does not appear to be a significant route of exposure to soil PAHs. Bioaccumulation has been shown in terrestrial invertebrates and voles, earthworm levels were 30-60 times greater than soil concentrations (Gile et al. 1982), but PAH metabolism is sufficient to prevent biomagnification. The oral toxicity of PAHs ranges from very to moderately toxic (50 to 1000s mg/kg bw) in rats. Many PAHs are cancer-causing, producing tumors in epithelial tissues in "practically all animal species tested" (Eisler 1987b). Other effects in terrestrial organisms are not well known, but may include adverse effects on reproduction, development, and immunity (ATSDR 1993c).

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References