TOXICITY TO HETEROTROPHIC PROCESSES AND SOIL AND LITTER INVERTEBRATES

ORGANICS

Chromium

Chromium (Cr) occurs in the environment as either chromium (III) or chromium (VI). Trivalent chromium is an essential metal in animals, playing an important role in insulin metabolism (Larngard and Norseth, 1979). Hexavalent chromium is more toxic than chromium (III) because of its high oxidation potential and the ease with which it penetrates biological membranes (Steven et al., 1976; Taylor and Parr, 1978). Chromium (III), the predominant form in the environment, exhibits decreasing solubility with increasing pH, and is completely precipitated at a pH above 5.5. In most soils, chromium is primarily present as precipitated chromium (III), which is not bioavailable and has not been know to biomagnify through food chains in its inorganic form (Eisler, 1986). Chromium is released into the environment in the processing of chromate, electroplating, production at tanning and textile plants, pigment production, and cooling tower preservation. Cr is naturally released into the environment through the weathering of soils (Fishbein, 1976).

Toxicity to Heterotrophic Processes and Soil and Litter Invertebrates. Liang and Tabatabai (1977) investigated the effects of various metals on N mineralization by native soil microflora in four soils. Chromium(III) at 260 ppm reduced N mineralization in the soil containing the highest organic matter content. The effects of Cr(III) on dehydrogenase activity of the native soil microflora in soil from the Rocky Mountain Arsenal was assessed by Rogers and Li (1985). After 6 days, a concentration of 30 ppm Cr (the lowest concentration tested) reduced dehydrogenase activity by 54%. Juma and Tabatabai (1977) evaluated the effect of Cr on soil acid and alkaline phosphatase activities in microbes. Acid and alkaline phosphatase activities were affected at 1635 ppm in all three soils to about the same degree, but greater inhibition of alkaline phosphatase activity occurred in the soil with the greatest content of organic matter and clay. Ross et al. (1981) evaluated the relative toxicities of forms of Cr to respiration of native soil microflora in a loam and a sandy loam soil. Cr (III), tested at only 100 ppm, caused reductions in both soils of 41 and 48%. A concentration of 10 ppm (the lowest concentration tested) Cr (VI) caused reductions in both soils (27 and 33%). Cr(VI) was more toxic than Cr(III) to soil respiration. Bhuiya and Cornfield (1976) investigated the effects of several metals on N mineralization and nitrification by native soil microflora in a sandy soil at different pH levels. At 6 weeks, mineralization and nitrification were reduced by 1000 ppm Cr at pH 7, but not at pH 6. After 12 weeks, neither mineralization nor nitrification was affected by Cr at either pH.

Haanstra and Doelman (1991) investigated short- and long-term effects of Cr on arylsulfatase activity, urease activity (Doelman and Haanstra, 1986), and total phosphatase activity (Doelman and Haanstra, 1989) by native soil microflora in five soils. The highest EC_{50}s were 3203, 5512, and 4470 ppm, respectively, for arylsulfatase, phosphatase, and urease activities found in different soils. The lowest was 17 ppm in the sand for arylsulfatase and 1170 and 490 ppm in the clay for phosphatase and urease. In an 18-month study, the highest EC_{50}s were 1798, 20020, and 1110 ppm Cr, respectively, for arylsulfatase, phosphatase, and urease activities found in different soils. The lowest were 12 and <1 ppm in the clay for arylsulfatase and urease activities and 2692 ppm in the sandy loam for phosphatase activity. The benchmark for Cr for microbes was established at 10 ppm because the 10th percentile lies between the EC_{50} values of 12 and 15 ppm from the work of Haanstra and Doelman (1991). Confidence in this benchmark is high because of the relatively large amount of data available for a variety of functional measures.

Abbasi and Soni (1983) assessed the effect of Cr(VI), added as $K_2Cr_2O_7$, on survival and reproduction of the earthworm *Octochaetus pattoni*. Survival was the most sensitive measure with a 75% decrease resulting from 2 ppm Cr, the lowest concentration tested. The number of cocoons

produced was not diminished until the concentration reached 20 ppm Cr (highest concentration tested); the number of juveniles produced was not affected. Soni and Abbasi, (1981) found no survival of *Pheretima posthuma* after 61 days in a paddy soil to which 10 ppm Cr(VI) (lowest concentration tested) was added. van Gestel et al. (1992), also found growth of *E. andrei* to be more sensitive to Cr than reproduction. 32 ppm Cr (III) reduced growth by 30% while cocoons/worm/week, percent fertile cocoons, and juveniles/worm/week were reduced 28, 22, and 51%, respectively, by 100 ppm Cr. Molnar et al. (1989) examined the effects of Cr(III) and Cr(VI) on growth and reproduction of *Eisenia fetida*. Reproduction after 8 weeks was the measure most sensitive to Cr(III) with a 55% decrease in the number of cocoons and hatchlings at 625 ppm Cr(III).

It is difficult to set a benchmark concentration for toxicity of Cr to earthworms. Survival may be more sensitive than reproduction to the metal when it is added to the earthworm substrate as a soluble salt. The relative toxicity of Cr(III) and Cr(VI) is not clear from these studies. Cr(VI) ions can pass through cell membranes with much greater ease than Cr(III) ions. However, it is thought that Cr(VI) is reduced to Cr(III) inside the cell (Molnar et al., 1989); this latter may be the final active form. Without a better understanding of Cr transformations in the soil, transport across earthworm cell membranes, and reactions within the cell, it is difficult to separate the effects of the two different forms. The 0.4 ppm benchmark for Cr is based on the work of Abbasi and Soni (1983). A safety factor of 5 was applied to the 2 ppm LOEC because it caused a 75% reduction in earthworm survival. Confidence in this benchmark is low because it is based on only five reported concentrations causing toxicity to earthworms (Table 4).

Copper

Copper (Cu) occurs in natural waters primarily as the divalent cupric ion in free and complexed forms (EPA 1985-Cu). Copper is a minor nutrient for both plants and animals at low concentrations, but is toxic to aquatic life at concentrations only slightly higher. Concentrations of 0.001 to 0.010 mg/L are usually reported for unpolluted surface waters in the United States. Common copper salts, such as the sulfate, carbonate, cyanide, oxide, and sulfide are used as fungicides, as components of ceramics and pyrotechnics, for electroplating, and for numerous other industrial applications (ACGIH, 1986). The largest anthropogenic releases of copper to the environment result from mining operations, agriculture, solid waste, and sludge from sewage treatment plants.

Toxicity to Heterotrophic Processes and Soil and Litter Invertebrates. Liang and Tabatabai (1977) investigated the effects of various metals on N mineralization by native soil microflora in four soils. Copper at 320 ppm severely reduced N mineralization in one soil. The effects of Cu on dehydrogenase activity of the native soil microflora in soil from the Rocky Mountain Arsenal was assessed by Rogers and Li (1985). A concentration of 30 ppm Cu (the lowest concentration tested) Cu reduced dehydrogenase activity by 28%. Bollag and Barabasz (1979) evaluated the effects of Cu on denitrification by three species of soil-dwelling *Pseudomonas* species of bacteria in autoclaved soil and by native soil microflora. In the autoclaved soil LOECs ranged from 10 (lowest concentration tested) to 250 ppm (highest concentration tested). Denitrification by the native soil population was reduced 44% by 250 ppm Cu. The effects of adding Cu, as $CuSO_4$, to a sandy loam adjusted to three pH levels on N mineralization during a 21-day incubation was assessed by Quraishi and Cornfield (1973). Mineralization was decreased by 1000 ppm Cu at all three pH levels (5.1, 5.9, and 7.3) with inhibitory effect increasing with decreasing pH from 39% to 100%.

Bhuiya and Cornfield (1972) assessed the effects of several metals on C mineralization by native microflora in a sandy soil, with or without added organic matter. After a further 12 weeks, soil respiration was reduced in the Cu-treated soil with amendment but not in the unamended soil. The influence of soil characteristics on effects of Cu on arylsulfatase activity was evaluated by Al-Khafaji and Tabatabai (1979). In the soil with the highest pH (7.6), Cu had no effect. Arylsulfatase activity in the other three soils was reduced by Cu at a concentration of 1590 ppm. Reductions were the least severe in the soil having the highest organic carbon and clay content. The effects of Cu (as $CuCl_2$) on carbon and nitrogen mineralization and nitrogen transformations in alfalfa-amended sieved soil were determined by Suter and Sharples (1984). At 10 days, respiration was reduced by 24% at 100 ppm, but significant reductions occurred at 500 ppm on days 3 and 14 and at 1000 ppm on all dates. Ammonia concentration was decreased significantly by 58% on days 4 and 11 at 10 ppm, but there was no increase of effects with concentration on those dates, and ammonia concentrations were unchanged or increased at those concentrations on later dates. Ammonia concentrations were significantly increased at 500 and 1000 ppm from day 18 to 53, and after day 18 there were regular patterns of increasing ammonia at increasing Cu levels. Nitrate levels were reduced by 21% on day 4 at a concentration of 10 ppm, but not on later dates. On day 11, nitrate concentrations were significantly reduced to 50–1000 ppm (but increased at 10 ppm), and on days 18 to 53, they were significantly reduced only at 500 and 1000 ppm Cu. These results suggest that nitrification is highly sensitive to Cu, relative to C and N mineralization. The benchmark for Cu has been established at 100 ppm, and confidence in this benchmark is high.

Neuhauser et al. (1984) evaluated the effects of soluble forms of copper on growth and reproduction *E. fetida*. After 6 weeks, both growth (weight) and cocoon production were decreased (75 and 85%) by 2000 ppm Cu, while 1000 ppm had no effect. Neuhauser et al. (1985) used the OECD artificial soil to estimate LC_{50} of Cu (added as Cu nitrate) for adult *E. fetida*. After 14 days, the LC_{50} was 643 ppm Cu. Spurgeon et al. (1994) kept adult *E. fetida* in contaminated OECD artificial soil for 8 weeks to test the effects of Cu (as $Cu(NO_3)_2$) on survival and growth of the earthworms. After 56 days, the calculated LC_{50} was 555 ppm, and the EC_{50} for cocoon production was 53.3 ppm. Bengtsson et al. (1986) looked at the effects of copper on *Dendrobaena rubida* at different acidities. After 4 months at pH 4.5, the number of cocoons produced per worm, hatchlings/cocoon, and total number of hatchlings were reduced 70, 64, and 74%, respectively, by 100 ppm Cu, the lowest concentration tested. The percent hatched cocoons was not affected. At pH 5.5, the number of cocoons produced per worm, hatchlings/cocoon, and percent hatched cocoons were reduced 96, 100, and 100%, respectively, by 500 ppm Cu, while 100 ppm had no effect. The total number of hatchlings was not affected. At pH 6.5, the number of cocoons produced per worm, hatchlings/cocoon, and percent hatched cocoons were reduced 90, 100, and 100%, respectively, by 500 ppm Cu, while 100 ppm had no effect. The total number of hatchlings was not affected.

In experiments by van Gestel et al. (1991) using Cu ($CuCl_2$) mixed homogeneously with the OECD substrate, growth of *E. fetida* was reduced 32% by 100 ppm (32 ppm had no effect). The EC_{50} for clitella development (sexual development) was >100 ppm Cu. In a study examining the effects of soil factors on Cu toxicity and uptake, Ma (1982) used a sandy loam soil spiked with $CuCl_2$ to determine the effects of Cu on survival of adult *Lumbricus rubellus*. After 12 weeks, 1000 ppm Cu caused an 82% decrease in survival while 150 ppm had no effect. The effect of soil organic carbon on toxicity of Cu ($CuSO_4$) to the earthworm *Octolasium cyaneum* was evaluated by Streit and Jaggy (1983). They determined the 14-day LC_{50} in a Brown soil, a Rendzina soil, and a peat soil containing 3.2, 14, and 43% organic carbon, respectively. The LC_{50} concentrations were 180, 850, and 2500 ppm, respectively. The relative sensitivity of several lumbricid earthworms to Cu ($CuCl_2$) added to a sandy soil was investigated by Ma (1988). EC_{50}s for cocoon production of *L. rubellus*, *Aporrectodea caliginosa*, and *Allolobophora chlorotica* were 122, 68, and 51 ppm Cu. The work

of Streit and Jaggy (1983) and others shows that the organic carbon content of the soil is a strong determinant of the bioavailability and toxicity of copper. From the studies cited, it appears that low pH has a compounding effect, with an increase in Cu availability resulting from more acid conditions. Overall, reproduction is more sensitive than mortality, and there is no consistent evidence that one genus of earthworms is any less tolerant to Cu under a given set of conditions than another genus. The benchmark for Cu was established at 50 ppm. Confidence in this benchmark is moderate.

The acute toxicity of Cu to the nematode *C. elegans* in four soils and in solution was evaluated by Donkin and Dusenbery (1993). A concentration of 105 ppm Cu in solution caused 50% mortality while at least 400 ppm (sandy loam soil) was required in soil. The highest LC_{50} (1061 ppm) was associated with the highest percentage organic matter in the loam soil. Parmalee et al. (1993) used a soil microcosm to test the effects of Cu on survival of nematodes and microarthropods feeding on native soil organic matter. There was an average reduction of approximately 70% in number of individuals of most categories of nematodes (fungivores, bactivores, herbivores, hatchlings) at 400 ppm total Cu, while 185 ppm had no effect. The number of individuals of the omnivores/predators category was reduced 85% by the lowest concentration of Cu tested, 72 ppm. Total microarthropod numbers were reduced about 50% by 400 ppm. Hopkin and Hames (1994) investigated the effects of Cu (as $CuNO_3$) in food on survival and reproduction of the terrestrial isopod *Porcellio scaber*. After 360 days, the number of juveniles produced was decreased 53% by 50 ppm Cu while 100 ppm of Cu was required to reduce total survival. The slug, *Arion ater*, was used as the test organism by Marigomez et al. (1986) to determine the effects of several pollutants on terrestrial mollusks. After 27 days, the animals experienced a 55% decrease in growth at 1000 ppm Cu, while 300 ppm had no effect. The studies of Donkin and Dusenbery (1993) and Parmalee et al. (1993) taken together show a higher concentration in soil than in solution is required to affect the survival of nematodes. Differences among groups of nematodes in sensitivity to Cu is shown by Parmalee et al. (1993). The application of soluble form of Cu to food material by Hopkin and Hames (1994) and Marigomez et al. (1986) show very distinct sensitivities of woodlice and slugs to Cu. The lowest toxic concentration reported in these two studies (72 ppm Cu) is higher than the benchmarks for earthworms (50 ppm) and microbes (30 ppm) (Table 4).

Mercury

Mercury (Hg) has no known biological function and is toxic to fish and wildlife. It is a mutagen and carcinogen that adversely affects the central nervous, renal, and reproductive systems of wildlife. Hg occurs in the environment as elemental mercury, Hg_2(II) and Hg(II), the latter of which is naturally oxidized from elemental mercury (Eisler, 1987). Mercury in ambient waters commonly occurs as mercury (II) or methylmercury. Mercury (II) can be methylated by both aerobic and anaerobic bacteria in the slime coat, liver, and intestines of fish, but methylation apparently does not occur in other tissues or in plants (EPA, 1985-Hg). From a toxicological standpoint, methylmercury (MeHg) poses a greater threat to biota due to its high stability and the ease with which it penetrates membranes in living organisms. Hg(II), however, is more prevalent in aquatic systems, bound up and unavailable in sediment layers. Biota bioconcentrate mercury compounds which can be further biomagnified through food chains (Wren, 1986). High concentrations of mercury in water are often associated with low alkalinity lakes and newly created bodies of water (Weiner and Stokes, 1990). Alkalinity, ascorbic acid, chloride, dissolved oxygen, hardness, organic complexing agents, pH, sediment, and temperature probably affect the acute and chronic toxicity and bioaccumulation of the various forms of mercury.

Anthropogenic sources of Hg include the combustion of fossil fuels, metal mining and processing plants, chloralkali plants, and disposal of batteries and fluorescent lamps (NAS 1978, and Das et al., 1982; as cited in Eisler, 1987).

Toxicity to Heterotrophic Processes and Soil and Litter Invertebrates. van Fanssen (1973) investigated the effects of an inorganic and an organic mercury compound on N mineralization and nitrification by native soil microflora in two alkaline soils. In the clay soil, both $HgCl_2$ and phenylmercury acetate reduced nitrification at 100 mg/kg Hg (10 mg/kg had no effect), but the organic form was more inhibitory than the inorganic form. Mineralization was not affected by the inorganic form but was decreased by phenylmercury acetate. In the dune sand, $HgCl_2$ severely reduced nitrification at 100 mg/kg (10 mg/kg had no effect) and phenylmercury acetate reduced nitrification at 10 mg/kg Hg (lowest concentration tested). Mineralization was not affected by the organic form but was decreased by $HgCl_2$ at 100 mg/kg Hg. This work indicates that the relative toxicity of various forms of Hg can be influenced by soil characteristics. Landa and Fang (1978) investigated the effects of mercuric chloride (up to 100 mg/kg Hg) on carbon mineralization by native soil microflora in five agricultural topsoils varying in pH and organic matter content. The magnitude of effects varied greatly among the soils and were not related to those two soil characteristics. Effects ranged from an 87% reduction at 0.1 mg/kg (the lowest concentration tested) in one of the soils to no effect at 100 mg/kg in another soil.

Liang and Tabatabai (1977) investigated the effects of Hg on N mineralization by native soil microflora in four soils. Mercury reduced N mineralization in all soils at 1003 mg/kg The greatest magnitude of the toxic effect was seen in the soil having the lowest pH and organic matter and clay contents. The influence of soil characteristics on effects of Hg on arylsulfatase activity was evaluated by Al-Khafaji and Tabatabai (1979). In two soils tested with a lowest concentration of 502 mg/kg, arylsulfatase activity was greatly reduced. In the two soils tested with a lowest concentration of 5015 mg/kg, arylsulfatase activity was inhibited almost totally. No clear differences between the soils with regard to effects on toxicity of Hg could be discerned.

Frankenberger and Tabatabai (1981) investigated the effect of Hg on amidase activity in three soils in shaker flask assays. After 2 1/2 hours, amidase activity was reduced in all three soils at 5015 mg/kg. The greatest reduction occurred in the soil with the lowest pH and organic matter and clay contents. Juma and Tabatabai (1977) evaluated the effect of Hg on soil acid and alkaline phosphatase activities. Acid and alkaline phosphatase activities were affected at 5015 mg/kg in all three soils to about the same degree. The benchmark of 30 mg/kg Hg is the 10th percentile of the 27 reported effective values. Confidence in this benchmark is high because of the relatively large amount of data available for a variety of functional measures.

Abbasi and Soni (1983) assessed the effect of Hg(II), added as $HgCl_2$, on survival and reproduction of *Octochaetus pattoni*. Survival and cocoon production were reduced 65 and 40% at 0.5 mg/kg Hg, the lowest concentration tested. The number of juveniles produced was not affected. The effect of methyl mercury on survival and segment regeneration of *Eisenia fetida* was investigated by Beyer et al. (1985). A concentration of 12.5 mg/kg Hg reduced survival by 21%, and the ability to regenerate excised segments was reduced by 69%. Methyl mercury at 2.5 mg/kg had no effect. It is not possible to evaluate the relative toxicity of forms of Hg based on these two studies which used different systems and evaluated two different families of earthworms. A benchmark of 0.1 mg/kg was established for Hg based on the work of Abbasi and Soni (1983). A safety factor of 5 was applied to the 0.5 mg/kg LOEC because it caused a 65% reduction in earthworm survival. Confidence in this benchmark is low because of the limited amount of data.

The slug, *Arion ater*, was used as the test organism by Marigomez et al. (1986) to determine the effect of mercury (as $HgCl_2$) on terrestrial mollusks. After 27 days on this diet, the animals experienced a 26% decrease in growth at 1000 mg/kg Hg, while 300 mg/kg had no effect.

Nickel

Nickel is a naturally occuring element that may exist in various mineral forms. It forms 0.008% of the earth's crust (NAS, 1980). Soil and sediment are the primary receptacles for nickel, but mobilization may occur depending on physico-chemical characteristics of the soil (ATSDR, 1988; USAF, 1990). Nickel is used in a wide variety of applications including metallurgical processes and electrical components, such as batteries (ATSDR, 1988; USAF, 1990). There is some evidence that nickel may be an essential trace element for mammals. Nickel occurs in nature in the nonionic and divalent states; other valence states occur very infrequently (Mastromatteo, 1986). Although nickel can exist in several oxidation states, the divalent cation state predominates and is generally considered the most toxic form (EPA 1986-Ni). As with many metals the toxicity of nickel increases as hardness decreases. Fish and invertebrates have approximately the same range of sensitivity.

Toxicity to Heterotrophic Processes and Soil and Litter Invertebrates. The effects of Ni, as nickel sulfate, on dehydrogenase activity of the native soil microflora in soil from the Rocky Mountain Arsenal was assessed by Rogers and Li (1985). After 6 days, a concentration of 30 ppm (lowest concentration tested) Ni reduced dehydrogenase activity by 39%. Babich and Stotzky (1982) evaluated the effects of two forms of Ni on mycelial growth rate of a number of soil-dwelling fungi inoculated individually into autoclaved sandy loam soil. The concentrations at which growth was reduced ranged from 50 to 750 ppm for Ni (added in chloride or sulfate form). Giashuddin and Cornfield assessed the effect of Ni added in oxide (1979) and sulfate (1978) forms on N and C mineralization by native soil microflora in a sandy soil. After 42 days incubation, soil respiration (C mineralization) was reduced in soil containing 10 ppm Ni from Ni sulfate (lowest concentration tested), and N mineralization was affected at 100 ppm. Soil respiration was reduced in soil containing 50 ppm Ni from Ni oxide (lowest concentration tested), and N mineralization was affected at 1000 ppm. Because of the test concentrations used, it is difficult to assess the relative toxicity of these two forms of Ni to C mineralization. When the soil pH was raised to 6.9 (from its normal 5.9), soil respiration was reduced in soil containing 250 ppm Ni from Ni oxide (lowest concentration tested), and N mineralization was affected at 1000 ppm. The effect of Ni from Ni sulfate was not tested at the higher pH. Raising the pH appeared to affect soil respiration but not N mineralization. A benchmark of 90 ppm Ni was established based on the 56 reported effective values. Confidence in this benchmark is high because of the relatively large amount of data available for a variety of functional measures.

The effects of Ni (added to horse manure as Ni acetate) on *E. fetida* were investigated by Malecki et al. (1982). In the 8-week test, 300 ppm Ni caused a 41% decrease in cocoon production, while 200 ppm had no effect. In the 20-week test, 200 ppm Ni caused a 23% decrease in cocoon production, while 100 ppm had no effect. Neuhauser et al. (1985) used the OECD artificial soil to determine LC_{50} of Ni (added as Ni nitrate) for adult *E. fetida*. After 14 days, the LC_{50} was calculated to be 757 ppm Ni. Neuhauser et al. (1984) evaluated the effects of soluble forms of nickel on growth and reproduction *E. fetida*. After 6 weeks, cocoon production was decreased 33% by 250 ppm Ni, the lowest concentration tested. Growth was not affected until 500 ppm was added to the substrate. In a study examining the effects of soil factors on Ni toxicity and uptake, Ma (1982) used a sandy loam soil spiked with $NiCl_2$ to determine the effects of Ni on survival of adult *Lumbricus rubellus*. After 12 weeks, 1000 ppm Cd caused a 31% decrease in survival while 150 ppm had no effect. A

benchmark of 200 ppm has been established for Ni based on the work of Malecki et al. (1982) which showed inhibition of reproduction at this concentration. Confidence in this benchmark is low because of the limited amount of data.

Zinc

Zinc (Zn) is an essential trace element in all organisms; it assures the stability of biological molecules and structures such as DNA, membranes, and ribosomes (Eisler, 1993). It is used commercially primarily in galvanized metals and metal alloys, but zinc compounds also have wide applications as chemical intermediates, catalysts, pigments, vulcanization activators and accelerators in the rubber industry, UV stabilizers, and supplements in animal feeds and fertilizers. Zinc compounds are also used in rayon manufacture, smoke bombs, soldering fluxes, mordants for printing and dyeing, wood preservatives, mildew inhibitors, deodorants, antiseptics, and astringents (Lloyd, 1984; ATSDR, 1989). Zinc phosphide is used as a rodenticide. Zinc makes up about 0.002% of the earth's crust (NAS, 1980) and occurs in many forms in natural waters and aquatic sediments.

In freshwater with pH >4 and <7, the dominant forms of dissolved zinc are the free ion (aquo ion complex) (98%) and zinc sulfate (2%) (Campbell and Stokes, 1985), whereas at pH 9.0, the dominant forms are the monohydroxide ion (78%), zinc carbonate (16%), and the free ion (6%) (EPA, 1987-Zn).

Zinc occurs in nature as a sulfide, oxide, or carbonate (Eisler, 1993). It is divalent in solution. Zinc interacts with many chemicals, and it may diminish the toxic effects of cadmium and protect against lead toxicosis in terrestrial animals (Eisler, 1993). Background concentrations seldom exceed 0.040 mg/L in water or 200 mg/kg in soil or sediment (Eisler, 1993).

Although it is essential for normal growth and reproduction (Prasad, 1979; Stahl et al., 1989) and important to central nervous system function (Eisler, 1993), the primary toxic effect of zinc is on zinc-dependent enzymes that regulate RNA and DNA. It is most harmful to aquatic life in conditions of low pH, low alkalinity, low dissolved oxygen, and elevated temperature. Zinc is relatively nontoxic in mammals, but excessive intake can cause a variety of effects. It is not known to be carcinogenic by normal exposure routes (Eisler, 1993).

Toxicity to Heterotrophic Processes and Soil and Litter Invertebrates. Wilson (1977) evaluated the effect of Zn, as zinc sulfate solution, on nitrification by native soil microflora in three soils. After 49 days, nitrification was severely inhibited in all three soils (98 to 100%) by 1000 ppm Zn, while Zn at 100 ppm had no effect. Haanstra and Doelman (1991) investigated short- and long-term effects of Zn on arylsulfatase activity, urease activity (Doelman and Haanstra, 1986), and total phosphatase activity (Doelman and Haanstra, 1989) by native soil microflora in five soils. In the 6-week study the highest EC_{50}s were 5559, 3623, and 1780 ppm for arylsulfatase, phosphatase, and urease activities, all in the soil with the greatest content of clay. The lowest EC_{50}s of 909, 220, and 420 ppm Zn were all found in the sand. In the 18-month study, the highest EC_{50}s were 9679, 4872, and 290 ppm Zn in different soils. The lowest EC_{50}s were 375, 170, and 70 ppm in different soils. Bhuiya and Cornfield (1974) investigated the effects of 1000 ppm Zn added as Zn oxide to a sandy soil at three pH levels on nitrogen mineralization by native soil microflora. After 42 days, N mineralization was reduced in the pH 7.7 soil but not in soils at pH 6 and 7. The effects of Zn on dehydrogenase activity of the native soil microflora in soil from the Rocky Mountain Arsenal was assessed by Rogers and Li (1985). A concentration of 300 ppm Zn reduced dehydrogenase activity by 30%; Zn at 150 ppm had no effect. Laskowski et al. (1994) looked at the effects of several metals

at low to moderate levels of $CdCl_2$ (up to 250 ppm Cd), $Pb(NO_3)_2$ (up to 2500 ppm Pb), or $ZnCl_2$ on the respiration rate of acid-mixed forest litter. Only zinc had an effect at the concentrations tested. A concentration of 1000 ppm Zn reduced respiration by 26%; Zn at 200 ppm had no effect. The effect of Cd and Zn on respiration of soil microflora in field-collected black oak forest soil/litter microcosms was evaluated by Chaney et al. (1978). After 23 days, Cd at a concentration up to 6 ppm had no effect. Respiration was decreased 21% by Zn at 479 ppm; Zn at 47 ppm had no effect. The benchmark of 100 ppm Zn is the 10th percentile of the 46 reported effective values. Confidence in this benchmark is high because of the relatively large amount of data available for a variety of functional response factors.

van Gestel et al. (1993) evaluated the effect of zinc added as $ZnCl_2$ to OECD artificial soil (pH 6.2), on the growth and reproduction of *E. andrei*. The numbers of cocoons and juveniles produced were reduced 31 and 42% by 560 ppm, while 320 ppm had no effect. The percent fertile cocoons and number of juveniles per fertile cocoon were not affected until Zn was added to a concentration of 1000 ppm Zn, and percent growth of individuals increased with increasing Zn concentration. Spurgeon et al. (1994) test the effects of Zn (as $Zn(NO_3)_2$) on survival and growth of *E. fetida*. After 56 days, the calculated LC_{50} was 745 ppm, and the EC_{50} for cocoon production was 276 ppm. Neuhauser et al. (1985) used the OECD artificial soil (pH 6) to determine LC_{50} of Zn [added as Zn (NO_3)] for adult *E. fetida*. After 14 days, the LC_{50} was calculated to be 662 ppm Zn. van Rhee (1975) tested the effects of one concentration of Zn (1100 ppm) added to a polder soil on body weight, number of cocoons produced per week, mortality and sexual development of *Allolobophora caliginosa*. After 60 days, there was a 53% loss of body weight and a 22% increase in mortality; clitellum development and cocoon production were completely inhibited. Neuhauser et al. (1984) evaluated the effects of soluble forms of zinc on growth and reproduction *E. fetida*. After 6 weeks, cocoon production was decreased 50% by 2500 ppm Zn, while 1000 ppm had no effect. Growth was not affected until 5000 ppm was added to the substrate. The effects of Zn added to horse manure (as zinc acetate) on *E. fetida was* investigated by Malecki et al. (1982). In the 8-week test, 2000 ppm Zn caused a 36% decrease in cocoon production, while 1000 ppm had no effect. In the 20-week test, 5000 ppm Zn caused a 53% decrease in cocoon production, while 2500 ppm had no effect. The EC_{50} value of 276 (Spurgeon et al., 1994) was the lowest toxic concentration of the seven reported. Confidence in the benchmark of 200 ppm Zn is low because of the limited amount of data.

Hopkin and Hames (1994) investigated the effects of Zn (as $ZnNO_3$) in food on survival and reproduction of the terrestrial isopod *Porcellio scaber*. Survival and number of juveniles produced after 360 days were decreased 100% by 1000 ppm Zn while 500 ppm had no effect. Zinc (as ZnO) was added to litter from the O_2 layer under woodlands to evaluate the effects of that metal on *Porcellio scaber* (Beyer et al. 1984). The only concentration tested, 5000 ppm, caused a 26% decrease in survival. Beyer and Anderson (1985) also used *Porcellio scaber* in experiments to determine the effect of Zn, added ZnO to ground deciduous leaf litter, on several population parameters. The maximum number of individuals in generation 2 and the survival rate for generation 2 were decreased 22 and 27% by 1600 ppm Zn in the diet, while 800 ppm had no effect. The slug, *Arion ater*, was used as the test organism by Marigomez et al. (1986) to determine the effect of Zn (as $ZnCl_2$) on terrestrial mollusks. After 27 days on this diet the animals experienced a 38% decrease in growth at 10 ppm Zn, the lowest concentration tested. The experiments evaluating the toxicity of Zn to the woodlouse *Porcellio scaber* show a high tolerance to the metal when it is added to the food in soluble form. The work with *Arion ater* (Marigomez et al., 1986) indicates that this organism is more sensitive than *P. scaber* to Zn. On the other hand, growth of individual woodlice was not evaluated and may be more sensitive than survival and reproduction.

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TOXICITY TO PLANTS

INORGANICS

Antimony

Antimony (Sb) is a naturally occurring metalloid element (displaying both metallic and nonmetallic properties) existing in valence states of 3 and 5 (Budavari, 1989; ATSDR, 1990). Metallic antimony and a few trivalent antimony compounds are the most significant regarding exposure potential and toxicity (ATSDR, 1990). Antimony is used in metallurgical processes, paints and enamels, various textiles, rubber, and fire retardation (antimony trioxide). Some antimonials such as potassium antimony tartrate have been used medicinally as parasiticides (Beliles, 1979).

Toxicity to Plants in Soil. Primary reference data for the toxicity of antimony to plants grown in soil was unavailable, however, the secondary reference data on the phytotoxicity of plants in soil noted undefined, qualitative phytotoxic effects on plants grown in surface soil (Kloke, 1979). Antimony is considered a nonessential metal and is easily taken up by plants if available in the soil in soluble forms (Kabata-Pendias and Pendias, 1984).

Arsenic

Arsenic (As) is a naturally-occuring metaloid found in air and all living organisms. It is present in the earth's crust at approximately 2 mg/kg and is sparingly soluble in water and body fluids. It occurs as two forms in ambient media, arsenic (III), usually the most toxic, and arsenic (V) (EPA, 1985-As) with its magnitude of bioavailability and toxicity dependent upon the oxidation state and temperature (McGeachy and Dixon, 1992). The state is dependent on environmental conditions, including Eh, pH, organic content, suspended solids and sediment. The relative toxicities of the various forms of arsenic apparently vary from species to species. Arsenic may be released into aquatic ecosystems by anthropogenic sources including the manufacture and use of arsenical defoliants and pesticides, electric generating stations, manufacturing companies, mineral or strip mines, steel production, fossil fuel combustion and smelting operations (Sorensen, 1991; McGeachy and Dixon, 1989; Ferguson and Gavis, 1972; NRCC, 1978) and natural leaching of the soils. Arsenic levels in a river ecosystem were found to be dependent upon the availability of arsenic, rain water dillution, extent of complexation with dissolved organic matter and possibly the metabolic activity of aquatic plants (Koranda et al. 1981). As soil clay concentration increases, arsenic adsorption into the soil increases as a function of soil pH, texture, iron, aluminum, organic matter and time (Woolson, 1977). Arsenic is known as one of the most toxic elements to fish with acute exposures resulting in immediate death (Sorensen, 1991).

Toxicity to Plants in Soil. The tolerance of spruce seedlings to As in soil was tested in field plots by Rosehart and Lee (1973). Three-yr old seedlings grown 335 d in soil to which 1000 mg/kg As was added as As(III) (lowest concentration tested) experienced a 50% reduction in height.

Soil type affected the toxicity of As(III) added to two soils on the shoot weight of cotton and soybeans grown from seed for 6 weeks (Deuel and Swoboda, 1972). In a sandy loam soil, shoot weights of both crops were reduced (cotton 22%; soybeans 45%) by 11 mg/kg As (the lowest concentration tested). Soybean growth in a clay soil was reduced 28% by 22.4 mg/kg As (lowest concentration tested). Cotton growth in this soil was reduced 29% by 89.6 mg/kg As.

The source of As(V) has been shown to influence the effect on corn grown from seed for 4 weeks in a loamy sand (pH 7.1). Plant weight reductions were almost 100% for NaH_2AsO_4, over

75% for $Al(H_2AsO_4)_3$, and about 65% for $Ca(H_2AsO_4)_2$ with the addition of 100 mg/kg As (Woolson et al., 1971).

Will and Suter (1995) reported soil NOEC and soil LOEC values for the effects of arsenic derived from experiments conducted in soil. The soil NOEC values ranged from 10 to 62.7 mg/kg and the soil LOEC values ranged from 2 mg/kg (barley) to 1000 mg/kg (spruce) for the phytotoxicity of arsenic.

Soil-plant uptake factors (concentration in plant/concentration in soil) for grass and herbs were calculated at 0.0166 and 0.0005, respectively (Efroymson et al., 1996).

Toxicity to Plants in Solution. Mhatre and Chaphekar (1982) found no effect of As(III) (As_2O_3), up to 1 mg/kg As, on germination of seeds of sorghum, alfalfa, mung bean, cluster bean, and radish. After 5d, reductions in root length occurred between 0.001 mg/kg As (29% reduction in cluster bean) and 1 mg/kg As (55 and 87% reductions in alfalfa and mung bean). The concentrations of As (V), from Na_2HAsO_4, required for a 50% reduction in seed germination and root length of mustard after 3d exposure in solution (pH 7.3), was reported by Fargasova (1994) to be 30 ppm. The EC50 for root length was 5.5 mg/kg As.

Will and Suter (1995) reported NOEC and LOEC values for the effects of arsenic in solution The NOEC's ranged from 0.001 to 0.1 mg/kg and the LOEC's ranged from 0.001 mg/kg (LCT for cluster bean) to 30 mg/kg (LC50 for mustard).

Phytotoxic Mode of Action. Arsenic is not essential for plant growth. It is taken up actively by roots, with arsenate being more easily absorbed than arsenite. Arsenic and phosphate ions are likely taken up by the same carrier (Asher and Reay, 1979). The phytotoxicity is strongly affected by the form in which it occurs in soils. Arsenite (III) is more toxic than arsenate (V), and both are considerably more toxic than organic forms (Peterson et al., 1981). In experiments with toxic levels of As, rice and legumes appear to be more sensitive than other plants. Symptoms include wilting of new-cycle leaves, retardation of root and top growth, violet coloration, root discoloration, cell plasmolysis, leaf necrosis and death (Aller et al., 1990). Arsenic is chemically similar to phosphorous, it is translocated in the plant in a similar manner and is able to replace P in many cell reactions. Arsenic (III) probably reacts with sulphydryl enzymes leading to membrane degradation and cell death. Arsenic (V) is known to uncouple phosphorylation and affect enzyme systems (Peterson et al., 1981).

Cadmium

Cadmium (Cd) occurs predominately in the form of free divalent cations in most well oxygenated, low organic matter, fresh waters (EPA 1985-Cd). However, both particulate matter and dissolved organic matter can bind cadmium in biologically unavailable forms. There is no evidence that cadmium is a biologically essential or beneficial element (Eisler, 1985). Cd toxicity is related to water hardness, with a reduction in toxicity associated with increased water hardness (EPA, 1985-Cd). Therefore, the cadmium toxicity values presented in Table 2 that are not from tests conducted in waters of moderate hardness are normalized to 100 mg/L using the slopes calculated by the EPA (1985-Cd).

Toxicity to Plants in Soil. A number of researchers have measured reductions in growth of a variety of plants in different soils with 10 mg/kg or less of Cd added to soil as soluble salts. Plants tested include sycamore and spruce trees, wild flowering plants, and crops and horticultural

plants (corn, lettuce, radish, wheat). Soils range from light sands to heavy silty clay loams in the acid to neutral pH range. There are no clear trends in responses indicating that any particular type of plant is more sensitive to Cd (reductions in growth range from 23 to 45%), or that growth conditions (pH and soil texture) consistently affected toxicity. Grasses appear to be generally less sensitive than several other plant groups with growth adversely affected at concentrations greater than 10 mg/kg (up to 160 mg/kg for oats) under a variety of growth conditions.

Will and Suter (1995) reported a large range of soil NOEC and LOEC values for the toxicity of cadmium in soil. The NOEC values ranged from 1 to 56.3 mg/kg and the LOEC values ranged from1 to 300 mg/kg.

Soil-plant uptake factors (concentration in plant/ concentration in the soil) for grass, herbs, and tree/shrubs were calculated at 0.2303, 0.0042, and 0.7837, respectively (Efroymson et al., 1996).

Toxicity to Plants in Solution. Will and Suter (1995) reported a large range of soil NOEC and LOEC values for the toxicity of cadmium in solution. The NOEC values ranged from 0.01 to 11.2 mg/L and the LOEC values ranged from 0.01 to 692 mg/L.

Phytotoxic Mode of Action. Cadmium is not essential for plant growth. If present in available form, it is readily taken up by the roots and translocated through the plant, and accumulated. Cadmium is chemically similar to Zn, an essential element. Competition between the two for organic ligands may explain some of the toxic effects of Cd, and the ameliorative effects of Zn on Cd toxicity. Cadmium depresses uptake of Fe, Mn, and probably Ca, Mg, and N (Wallace et al., 1977d; Iwai, et al. 1975). Cadmium is toxic at low concentrations. Symptoms resemble Fe chlorosis, and include necrosis, wilting, reduced Zn levels, and reduction in growth. The mechanisms of toxicity include reduced photosynthetic rate, poor root system development, reduced conductivity of stems, and ion interactions in the plant. Agronomic crops are more sensitive to Cd toxicity than trees (Adriano, 1986).

Chromium

Chromium (Cr) occurs in the environment as either chromium (III) or chromium (VI). Trivalent chromium is an essential metal in animals, playing an important role in insulin metabolism (Larngard and Norseth, 1979). Hexavalent chromium is more toxic than chromium (III) because of its high oxidation potential and the ease with which it penetrates biological membranes (Steven et al., 1976; Taylor and Parr, 1978). Chromium (III), the predominant form in the environment, exhibits decreasing solubility with increasing pH, and is completely precipitated at a pH above 5.5. In most soils, chromium is primarily present as precipitated chromium (III), which is not bioavailable and has not been know to biomagnify through food chains in its inorganic form (Eisler, 1986). Chromium is released into the environment in the processing of chromate, electroplating, production at tanning and textile plants, pigment production, and cooling tower preservation. Cr is naturally released into the environment through the weathering of soils (Fishbein, 1976).

Toxicity to Plants in Soil. It is believed that Cr(VI) is more toxic to plants and more mobile inside the plant than Cr(III). It has also been shown that valence of the Cr ion is more important in determining the distribution of the element than the specific species. There are, however, conflicting views on the uptake and metabolism of Cr(III) and Cr(VI). One argument is that Cr(VI) may be absorbed into roots while the other states that Cr(VI) is reduced to Cr(III) just before absorption into the roots (Smith et al., 1989).

Turner and Rust (1971) investigated the effect of Cr added as Cr(VI) on soybean seedlings grown 3 days in a loam soil. Fresh shoot weight was reduced 30% by 30 mg/L Cr, while 10 mg/kg had no effect. Adema and Henzen (1989) calculated EC50 concentrations for effects of Cr added as Cr(VI) on lettuce, tomato and oats grown in a growth chamber from seed for 14d. The EC50 for lettuce in a humic sand soil (pH 5.1, % organic matter 3.7) was greater than 11 mg/kg while in a loam soil (pH 7.4, % organic matter 1.4) it was 1.8 mg/kg Cr. The EC50 for tomato in the humic sand soil was 21 mg/kg, while in the loam soil it was 6.8 mg/kg Cr. The EC50 for oats in the humic sand soil was 31 mg/kg, while in the loam soil it was 7.4 mg/kg Cr. Results of these experiments show the ameliorating effects of organic matter on Cr (VI) toxicity.

Farasgova (1994) studied the effects of metals on the germination and root growth of *Sinapsis alba* seeds at various pHs. 72h LC50s for germination were 123.03 mg/kg at pH 2.46 and 100.0 mg/kg at pH 7.25. 72h LC50s for root growth inhibition were 5.01 mg/kg at pH 2.46 and 45.71 mg/kg at pH 7.25.

Will and Suter (1995) reported soil NOEC and soil LOEC values for the toxicity of chromium to plants in soil. The NOEC values ranged from 0.35 to 11 mg/kg and the LOEC values ranged from 1.8 to 31 mg/kg. A bioconcentration factor of 1000 was calculated for barley seedlings (Smith et al., 1989).

Soil-plant uptake factors (concentration in plant/ concentration in the soil) for grass, herbs, and tree/shrubs were calculated at 0.0663, 0.0007, and 0.0249, respectively (Efroymson et al., 1996).

Toxicity to Plants in Solution. Calculated EC50 concentrations for effects of Cr(VI) added as $K_2Cr_2O_7$ on lettuce, tomato and oats grown from seed for 14d ranged from 0.16 (lettuce) to 1.4 mg/kg Cr (oats) (Adema and Henzen, 1989). The concentration of Cr(VI), from $(NH_4)_2CrO_4$, required for a 50% reduction in seed germination and root length of mustard after 3d exposure in solution (pH 7.3), was reported by Farasgova (1994) to be 100 mg/kg. EC50 for root length was 46 mg/kg Cr. Using a 1:1 combination of Cr(III) ($CrCl_3$) and Cr(VI) (K_2CrO_7) in nutrient solution (pH 5), Hara et al. (1976) measured a 68% reduction in weight of cabbage with 10 mg/kg Cr (2 mg/kg had no effect).

Top weight of soybean seedlings grown for 5d in nutrient solution containing Cr(VI) was reduced 21% by 1 mg/kg Cr, while 0.5 mg/kg had no effect (Turner and Rust, 1971). Wallace et al. (1977a) measured a 30% reduction in leaf weight of bush beans grown 11d in nutrient solution containing 0.54 mg/kg Cr as (Cr VI).

Length of the longest root of rye grass was reduced 69% by exposure to 2.5 mg/kg Cr(VI) (lowest concentration tested) in nutrient solution (pH 7) for 14d (Wong and Bradshaw, 1982). Length of the longest shoot was not affected at this concentration. Breeze (1973) found little difference in the toxicity of Cr(III) [$Cr_2(SO_4)_3$] and Cr(VI) ($K_2Cr_2O_7$) to rye grass seed germination. Seed exposed to solutions containing 50 mg/kg Cr (III) or (VI) reduced germination 37 and 38% after 2.5d.

Nutrient solution containing 0.05 mg/kg Cr(III) [$Cr_2(SO_4)_3$] reduced leaf and stem weights of chrysanthemum seedlings exposed for 2 d by 31 and 36% (Patel et al., 1976). This was the lowest concentration tested and root weight was not affected.

Based on these experiments, there is an indication that the source of the Cr affects plant response and seed germination is not as sensitive as growth.

Will and Suter (1995) reported NOEC and LOEC values for the toxicity of chromium to plants in solution. The NOEC values ranged from 0.004 to 50 mg/L and the LOEC values ranged from 0.052 to 100 mg/L.

Phytotoxic Mode of Action. Chromium is not an essential element in plants. The (VI) form is more soluble and available to plants than the (III) form and is considered the more toxic form (Smith, et al., 1989). In soils within a normal Eh and pH range, Cr(VI), a strong oxidant, is likely to be reduced to the less available Cr(III) form, although the (III) form may be oxidized to the (VI) form in the presence of oxidized Mn (Bartlett and James, 1979). In nutrient solution, however, both forms are about equally taken up by plants and toxic to plants (McGrath, 1982). Cr(VI), as CrO_4^{2-}, may share a root membrane carrier with SO_4^{2-}. Cr(VI) is more mobile in plants than Cr(III) but translocation varies with plant type. After plant uptake, Cr generally remains in the roots because of the many binding sites in the cell wall capable of binding especially the Cr(III) ions (Smith et al., 1989). Within the plant Cr(VI) may be reduced to the +3 form and complexed as an anion with organic molecules. Symptoms of toxicity include stunted growth, poorly developed roots, and leaf curling. Chromium may interfere with C, N, P, Fe, and Mo metabolism, and enzyme reactions (Kabata-Pendias and Pendias, 1984).

Cobalt

Cobalt (Co), a hard, silvery white metal, is strategically important in the production of high-temperature alloys and permanent magnets (Brobst and Pratt, 1973). Cobalt salts are utilized in the production of pigments and in paint dryers as catalysts (Lustigman et al., 1995). It is found in the earth's crust at an average concentration of 20 mg/kg. Cobalt is an essential element making up approximately one-half of the contents of vitamin B12, which is necessary in the prevention of pernicious anemia (Oehme, 1979). Comparatively little is known about the toxicity of cobalt.

Toxicity to Plants in Soil. Linzon (1978) reported unspecified toxic effects on plants grown in a surface soil with the addition of 20 mg/kg Co.

Soil-plant uptake factors (concentration in plant/ concentration in the soil) for grass, herbs, and tree/shrubs were calculated at 0.0096, 0.0056, and 0.0026, respectively (Efroymson et al., 1996).

Toxicity to Plants in Solution. Wallace et al. (1977a) evaluated the effect of Co as $CoSO_4$ on bush beans grown for 21d in nutrient solution. Leaf dry weight was reduced 22% by the addition of 0.06 mg/kg Co, the lowest concentration tested. Root and stem weight were not affected at this concentration. Chrysanthemum seedling root weight was reduced 55% after 21d growth in nutrient solution containing the same concentration of Co as $CoSO_4$ (Patel et al., 1976). Leaf and stem weight were not affected at this concentration.

Will and Suter (1995) reported NOEC and LOEC values for the toxicity of cobalt to plants in solution. The NOEC values ranged from 1 to 50 mg/kg and the LOEC values ranged from 0.06 to 50 mg/kg.

Phytotoxic Mode of Action. Cobalt is not known to be essential to plants except legumes in symbiosis with N_2-fixing microorganisms. When translocated from roots it travels in the xylem as the Co(II) ion (Tiffin, 1967). Toxicity symptoms due to excess Co are typical of Fe deficiency

induced chlorosis and necrosis, and root tip damage (Wallace et al., 1977a). There appears to be inhibition of mitosis and chromosome damage (Aller et al., 1990).

Copper

Copper (Cu) occurs in natural waters primarily as the divalent cupric ion in free and complexed forms (EPA 1985-Cu). Copper is a minor nutrient for both plants and animals at low concentrations, but is toxic to aquatic life at concentrations only slightly higher. Concentrations of 0.001 to 0.010 mg/L are usually reported for unpolluted surface waters in the United States. Common copper salts, such as the sulfate, carbonate, cyanide, oxide, and sulfide are used as fungicides, as components of ceramics and pyrotechnics, for electroplating, and for numerous other industrial applications (ACGIH, 1986). The largest anthropogenic releases of copper to the environment result from mining operations, agriculture, solid waste, and sludge from sewage treatment plants.

Toxicity to Plants in Soil. Miles and Parker (1979) found approximately 68% reductions in root and shoot weights of little bluestem grown from seed for 12 weeks in a sandy soil (pH 7.8, % organic matter 2.5), with 100 mg/kg Cu as $CuSO_4$ added (only concentration tested). Growth was reduced in a second sandy soil (pH 4.8, % organic matter 1.9) by 86% with the addition of 100 mg/kg Cu (only concentration tested). Wallace et al. (1977c) evaluated the effects of Cu, added as $CuSO_4$ to a loam soil, on leaf and stem weights of bush beans grown from seed for 17 days. Leaf weight was reduced 26% by 200 mg/kg Cu, while 100 mg/kg had no effect.

Soil-plant uptake factors (concentration in plant/ concentration in the soil) for grass, herbs, and tree/shrubs were calculated at 0.1409, 0.0012, and 0.2439, respectively (Efroymson et al., 1996).

Toxicity to Plants in Solution. The effect of Cu on stem diameter increase and plant weight of red pine, maple, dogwood, and honeysuckle was examined by Heale and Ormrod (1982). All seedlings (90-d old) grown for 110 d in nutrient solution containing 4 mg/L Cu from $CuSO_4$ (lowest concentration tested) were affected. Reductions in rate of stem diameter increase and in plant weight were 41 and 50%, 79 and 67%, and 97 and 74% for maple, dogwood, and honeysuckle, respectively. Red pine experienced a 28% decrease in plant weight at 4 mg/L Cu but the stem diameter increase was unaffected up to 20 mg/L Cu (highest concentration tested).

Wong and Bradshaw (1982) measured a 71% reduction in length of longest root in rye grass grown for 14 d in nutrient solution (pH 7) to which 0.031 mg/L Cu as $CuSO_4$ was added (lowest concentration tested). After 4 d, root length of rice seedlings was reduced 64% by 64 ppm Cu (6.4 mg/L had no effect) added as $CuSO_4$ to nutrient solution (Gupta and Mukherji, 1977).

Maize seedlings germinated and grown for 10 d in solution containing $CuSO_4$ had a 40% reduction in total fresh weight in the 0.06 mg/L Cu treatment (lowest concentration tested) (Stiborova et al., 1986). This same concentration caused a 45% reduction in root weight of chrysanthemums grown for 21d in nutrient solution with $CuSO_4$ added (Patel et al., 1976).

Will and Suter (1995) reported NOEC and LOEC values for the toxicity of copper to plants in solution. The NOEC values ranged from 0.5 to 50 mg/L and the LOEC values ranged from 0.031 to 100 mg/L.

Phytotoxic Mode of Action. Copper is a micronutrient essential for plant nutrition. It is required as a co-factor for many enzymes and is an essential part of a copper protein involved in

photosynthesis. Copper occurs as part of enzymes and enzyme systems. Root absorption appears to be passive, perhaps in organo-copper complexes (Jarvis and Whitehead, 1983), and active through a specific carrier (Fernandes and Henriques, 1991). Copper may be deficient in low-copper soils because the metal is adsorbed to cells in the root system. The form in which it is taken into the root affects its binding there (Wallace and Romney, 1977c). Copper can be transported in the xylem and phloem of plants complexed with amino acids.

The most common toxicity symptoms include reduced growth, poorly developed root system, and leaf chlorosis (Wong and Bradshaw, 1982). The basic deleterious effect of Cu is related to the root system where it interferes with enzyme functioning (Mukherji and Das Gupta, 1972). It also strongly interferes with photosynthesis and fatty acid synthesis (Smith et al., 1985).

Mercury

Mercury (Hg) has no known biological function and is toxic to fish and wildlife. It is a mutagen and carcinogen that adversely affects the central nervous, renal, and reproductive systems of wildlife. Hg occurs in the environment as elemental mercury, Hg_2(II) and Hg(II), the latter of which is naturally oxidized from elemental mercury (Eisler, 1987). Mercury in ambient waters commonly occurs as mercury (II) or methylmercury. Mercury (II) can be methylated by both aerobic and anaerobic bacteria in the slime coat, liver, and intestines of fish, but methylation apparently does not occur in other tissues or in plants (EPA, 1985-Hg). From a toxicological standpoint, methylmercury (MeHg) poses a greater threat to biota due to its high stability and the ease with which it penetrates membranes in living organisms. Hg(II), however, is more prevalent in aquatic systems, bound up and unavailable in sediment layers. Biota bioconcentrate mercury compounds which can be further biomagnified through food chains (Wren, 1986). High concentrations of mercury in water are often associated with low alkalinity lakes and newly created bodies of water (Weiner and Stokes, 1990). Alkalinity, ascorbic acid, chloride, dissolved oxygen, hardness, organic complexing agents, pH, sediment, and temperature probably affect the acute and chronic toxicity and bioaccumulation of the various forms of mercury.

Anthropogenic sources of Hg include the combustion of fossil fuels, metal mining and processing plants, chloralkali plants, and disposal of batteries and fluorescent lamps (NAS 1978, and Das et al., 1982; as cited in Eisler, 1987).

Toxicity to Plants in Soil. There was no primary reference data describing toxicity of Hg to plants grown in soil. Kloke (1979) report unspecified toxic effects on plants grown in a surface soil with the addition of 0.3 mg/kg Hg.

Toxicity to Plants in Solution. Several authors have provided information on the relative phytotoxicity of forms of Hg. Results indicate that organic forms are more toxic than inorganic forms. The effect of Hg, as $HgCl_2$, on root elongation of 3-week old Norway spruce seedlings grown for 7d in nutrient solution (pH 4) was examined by Lamersdorf et al. (1991). The only concentration tested, 0.002 mg/L Hg, reduced root elongation by 31%. Methyl mercury (CH_3HgCl) completely stopped root elongation at a concentration of 0.0002 mg/L, the only concentration tested. Schlegel et al. (1987) investigated the effects of inorganic ($HgCl_2$) and organic (CH_3HgCl) Hg on needle chlorophyll content, transpiration rate, and CO_2 uptake of 2-week old spruce seedlings in nutrient solution (pH 4.3) for 35d. Methyl Hg at 0.002 mg/L Hg (lowest concentration tested) reduced transpiration rate, and CO_2 uptake by 49, and 73%. At 0.02 mg/L Hg (lowest concentration tested), both forms reduced needle chlorophyll content approximately 28%.

Mukhiya et al. (1983) compared the toxicity of different Hg compounds to barley growth in solution at concentrations of 1 to 50 mg/L, and found organic forms to be more toxic than inorganic forms. After 7d, mercury as $C_8H_8HgO_2$ (phenyl mercuric acetate) at 5 mg/L reduced growth approximately 25%. Mercuric acetate ($C_4H_6HgO_4$) at 10 mg/L Hg reduced root length and plant weight 23%. Mercurous chloride (Hg_2Cl_2) and mercuric chloride ($HgCl_2$) at 50 mg/L reduced growth approximately 25%.

Seeds of sorghum, alfalfa, mung bean, cluster bean , and radish were allowed to germinate and grow for 5d in solutions containing 0.001 to 1 mg/L Hg as $HgCl_2$ (Mhatre and Chaphekar, 1982). At 0.01 mg/L Hg, root length reductions ranged from 22 for radish to 52% for alfalfa, with Pennisetum, mustard, sorghum, and cluster bean having intermediate reductions. Shoot length of *Pennisetum*, alfalfa, and cluster bean were also reduced at this concentration 25, 37, and 26%, respectively. Root length of pea was reduced 40% by the addition of 0.1 mg/L. Root and shoot lengths of mung bean were reduced 28 and 50% at this concentration. The concentration of Hg, from $HgCl_2$, required for a 50% reduction in seed germination and root length of mustard after 3d exposure in solution (pH 7.4), was reported by Fargasova (1994) to be 129 mg/L. EC50 for root length was 9.3 mg/L Hg. After 14d, lengths of longest root and shoot of germinating rye grass seedlings were reduced 40 and 23% by 5 mg/L Hg (lowest concentration tested) added to nutrient solution (pH 7) as $HgCl_2$ (Wong and Bradshaw, 1982).

Plant Uptake. Organic forms of Hg may be translocated from roots to shoots to a greater degree than inorganic forms in some plants (Huckabee and Blaylock, 1973). Gay (1975) reports that pea plants form methylmercury as an intermediate product from Hg added to the soil in organic and inorganic forms.

Soil-plant uptake factors (concentration in plant/ concentration in the soil) for grass, and tree/shrubs on the Oak Ridge Reservation were calculated at 0.0339, and 0.0468, respectively (Efroymson et al., 1996).

Nickel

Nickel is a naturally occuring element that may exist in various mineral forms. It forms 0.008% of the earth's crust (NAS, 1980). Soil and sediment are the primary receptacles for nickel, but mobilization may occur depending on physico-chemical characteristics of the soil (ATSDR, 1988; USAF, 1990). Nickel is used in a wide variety of applications including metallurgical processes and electrical components, such as batteries (ATSDR, 1988; USAF, 1990). There is some evidence that nickel may be an essential trace element for mammals. Nickel occurs in nature in the nonionic and divalent states; other valence states occur very infrequently (Mastromatteo, 1986). Although nickel can exist in several oxidation states, the divalent cation state predominates and is generally considered the most toxic form (EPA 1986-Ni). As with many metals the toxicity of nickel increases as hardness decreases. Fish and invertebrates have approximately the same range of sensitivity.

Toxicity to Plants in Soil. A limited number of studies have evaluated the effects of Ni on oak, rye grass, corn, cotton, and beans. Dixon (1988) measured a 30% weight reduction of red oak seedlings grown for 16 weeks in a sandy loam soil (pH 6, % organic matter 1.5) with addition of 50 mg/kg Ni ($NiCl_2$) (no effect at 20 mg/kg Ni).

Rye grass shoot weight was reduced 66% with the addition of 180 mg/kg Ni (as $NiSO_4$) to a loam soil (pH 4.7) in which the plants had been grown for 4 weeks from seed (90 mg/kg had no

effect) (Khalid and Tinsley, 1980). Oats grown from seed for 110 d in the presence of 50 mg/kg Ni (as $NiCl_2$) in soil (pH 6.1, % organic matter 1.4) had reductions of 38 and 63% in grain and straw weight (Halstead et al., 1969). In a second soil (pH 5.7, % organic matter 4.1) only straw weight was reduced (45%) by addition of 100 mg/kg Ni (50 mg/kg had no effect). Traynor and Knezek (1973) measured a 21% reduction in corn plant weight with the addition of 294 mg/kg Ni (as $NiCl_2$) to a sandy soil (pH 5, % organic matter 2) in which the plants had been grown for 5 weeks from seed. Addition of 220 mg/kg had no effect.

Wallace et al. (1977b) report the results of experiments on the effects of Ni (as $NiSO_4$) on seedlings of a variety of plants grown in a loam soil at several pH's. Corn grown in soil at pH 4.2, 5.6, and 7.5 experienced 74, 80, and 50% reductions in shoot weight after 14 days growth with the addition of 250 mg/kg Ni. Ni at 100 mg/kg had no effect. At pH 5.8, bush beans grown for 16 days had a 64% reduction in shoot weight with the addition of 100 mg/kg (lowest concentration tested). At pH 7.5, a 36% reduction in plant weight occurred with 250 mg/kg Ni, while 100 mg/kg had no effect. After 28 days growth in a loam soil at pH 5.8, bush bean leaf weight was reduced 45% by the addition of 100 mg/kg Ni, while 25 mg/kg had no effect. For barley under these same growth conditions, 25 mg/kg Ni (lowest concentration tested) reduced shoot weight 88%.

Two-week old cotton seedlings grown for 35 d in soil (pH 6.8) to which 100 mg/kg Ni was added (lowest concentration tested) experienced reductions in leaf and stem weights of approximately 45 to 60% (Rehab and Wallace, 1978).

Toxicity to Plants in Solution. The effect of Ni on stem diameter increase and plant weight of red pine, maple, dogwood, and honeysuckle was examined by Heale and Ormrod (1982). Seedlings (90-d from cutting) of red pine and honeysuckle grown for 110 d in nutrient solution containing 2 mg/L Cu from $NiSO_4$ (lowest concentration tested) had reductions in stem diameter increase and plant weight of 100, and 25%, and 84 and 65%, respectively.. Reductions in stem diameter increase in plant weight were 70% dogwood grown in solution containing 10 mg/L Ni, while 2 mg/L had no effect. Maple experienced a 48% decease in plant weight only at 10 mg/L Cu with the stem diameter increase remaining unaffected up to 20 mg/L Cu (highest concentration tested).

The effects of Ni on several horticultural and field crops have been evaluated. Wong and Bradshaw (1982) measured a 29% decrease in length of longest root of rye grass when germinated and grown for 14 d in nutrient solution (pH 7) with 0.13 mg/L Ni [$Ni(NH_4)_2(SO_4)_2$], the lowest concentration tested. Wallace (1979) measured 92 and 68% decreases in root and leaf weights of bush bean seedlings when grown for 21 d in nutrient solution (pH 5) with 1.2 mg/L Ni, the only concentration tested. The effects of 0.25 to 20 mg/L Ni, from $NiSO_4$, on germination and radicle length after 3 d growth in solution of radish, cabbage, turnip, lettuce, wheat, and millet were determined by Carlson et al. (1991b). There was no effect on seed germination up to 20 mg/L Ni. Effective concentrations ranged from 1 mg/L (25% reduction in radicle length of lettuce and turnip) to 12 mg/L (40% reduction in radicle length of millet). The effect of Ni, as $NiCl_2$, on plant weight of cotton grown in nutrient solution (pH 6) was evaluated by Rehab and Wallace (1978). Plant weight was reduced 92% by 5.9 mg/L Cd, while 0.59 mg/L had no effect.

Patel et al. (1976) found 26 and 27% decreases in leaf and stem weights of chrysanthemum seedlings when grown for 14 d in nutrient solution with 0.59 mg/L Ni ($NiSO_4$), while 0.006 mg/L had no effect. Root weight was not affected at 0.59 mg/L Ni.

Phytotoxic Mode of Action. Nickel is not generally considered to be an essential element for plants. However, it may be required by nodulated legumes for internal N transport as part of the urease enzyme (Aller et al., 1990). It is generally adsorbed as the Ni(II) ion and translocated in xylem and phloem with an organic chelate (Hutchinson, 1981). Nickel is fairly uniformly distributed between roots and shoots (Wallace and Romney, 1977b). Symptoms of Ni toxicity are generally Fe-deficiency induced chlorosis and foliar necrosis (Khalid and Tinsley, 1980). Excess nickel affects nutrient absorption by roots, root development, and metabolism, and it inhibits photosynthesis and transpiration. Nickel can replace Co and other heavy metals located at active sites in metallo-enzymes and disrupt their functioning.

Selenium

Selenium (Se), is an essential nutrient for some plants and animals when present in trace amounts. However, at levels currently present in the environment, Se poses a significant toxic risk. Anthropogenic sources include the combustion of fossil fuels, municipal wastes, irrigation run-off and industrial effluents. Lechate from seleniferous soils also contributes to the presence of Se in the environment (Eisler, 1985b). Selenium has a combination of attributes which make it an unusual pollutant (EPA, 1987-Se). These attributes include, but are not limited to, the following: it is an essential trace nutrient; it can occur in three oxidation states and be reduced or oxidized by various organisms; it is a metalloid with physicochemical properties similar to sulfur, which may reduce the toxicity of selenium or be replaced by selenium in biologically important compounds; it can reduce the toxicity of several heavy metals and have varying effects on cadmium and mercury toxicity; both water and food are important exposure pathways for aquatic biota; and there are substantial natural and anthropogenic releases to water. The two oxidation states (Se(IV) and Se(VI)) for which the most toxicity data exist are discussed separately. Plants have the ability to convert inorganic selenium to organic selenium compounds, thereby increasing their biological availability (Lo and Sandi, 1980).

Toxicity to Plants in Soil. Wan et al. (1988) investigated the effects of Se(VI), as Na_2SeO_4, on alfalfa grown from seed to 0.25 bloom stage in three soils. In the sandy loam soil (pH 6.7, % organic matter 13) and in the two clay loam soils (pH 5.6, % organic matter 15; pH 6.9, % organic matter 13), shoot weight was reduced 83, 33, and 56% by the addition of 1.5 mg/kg Se(VI), while 0.5 mg/kg had no effect. The effect of Se(VI) (Na_2SeO_4) on alfalfa grown from seed to bloom was examined in five silty clay loam soils, ranging in pH from 6.9 to 7.8, by Soltanpour and Workman (1980). Shoot weight was reduced by 2 mg/kg in 4 of the 5 soils (91, 74, 23, and 27% reductions), with the greatest reductions in soils with the lowest organic matter content (%'s organic matter 3.1, 3.7, 5, and 6.5, respectively).

Carlson et al. (1991a) investigated the effects of Se(VI) (as Na_2SeO_4) and Se (IV) (as Na_2SeO_3) on sorgrass (*Sorghum*) grown from seed for 42 days in several soils. In a loamy sand soil (% organic matter 19) at pH 5.5 and 6.0, there were 59 and 53% reductions in shoot weight with the addition of 1 mg/kg Se(VI), (lowest concentration tested). No reductions were observed with additions of up to 4 mg/kg Se(IV). In a sandy soil at pH 4.9, 1 mg/kg Se(VI) and 2 mg/kg Se(IV) caused 64 and 61% reductions in shoot weight. In this same sandy soil limed to pH 6.5, Se(IV) had no effect up to 4 mg/kg and Se(VI) reduced shoot weight 66% at 1 mg/kg.

Soil-plant uptake factors (concentration in plant/ concentration in soil) for herbs and tree/shrubs were calculated at 0.004 and 0.072, respectively (Efroymson et al. 1996). However, some plants are Se hyperaccumulators.

Toxicity to Plants in Solution. Martin (1937) evaluated the effect of Se (IV) from Na_2SeO_3 on root and shoot weight, and plant height of wheat and buckwheat seedlings growing in nutrient solution for 42d. Selenium at 1 mg/L (lowest concentration tested) reduced wheat root and shoot weight, and plant height 41, 40, and 23%. This concentration also reduced buckwheat root and shoot weight, and plant height 59, 75, and 44%. Selenium VI (Na_2SeO_4) at 0.79 mg/L (lowest concentration tested) caused a 21% reduction in root weight of bush bean seedlings grown in nutrient solution (pH 4.4) (Wallace et al., 1980).

Phytotoxic Mode of Action. Selenium is absorbed by plants as selenite (IV), selenate (VI) or in organic form and the selenate may be the more toxic. It is believed that selenate is taken up actively while selenite uptake is largely passive (Peterson et al., 1981). Selenium is translocated to all parts of the plant, including the seed, in low molecular weight compounds (Broyer et al., 1972). Toxicity symptoms include chlorosis, stunting and yellowing of the leaves. The mechanism of toxicity is thought to be indiscriminate replacement of S by Se in proteins and nucleic acids with disruptions in metabolism (Trelease et al., 1960).

Silver

Silver (Ag), a basic element, occurs naturally in the environment as a soft, silver colored metal (ATSDR, 1989b). It also occurs in a powdery white or dark gray to black compound. Silver is found at an average of 0.1 mg/kg in the earth's crust and about 0.3 mg/kg in soils. Silver metals and silver compounds are used in the production of surgical protheses, fungicides, coinage, jewelry, and dental fillings (Fisher et al., 1984). The accumulation of silver in marine algae appears to result from adsorption rather than uptake; bioconcentration fators of 13,000 - 66,000 have been reported (Fisher et al., 1984; ATSDR, 1989b).

Toxicity to Plants in Soil. There was no primary reference data showing toxicity of Ag to plants grown in soil. Linzon (1978) reported unspecified toxic effects on plants grown in a surface soil with the addition of 2 mg/kg Ag.

Toxicity to Plants in Solution. Wallace (1979) examined the effect of Ag from $AgNO_3$ on shoot weight of bush bean seedlings grown in nutrient solution (pH 5) for 13 d. Silver at 0.16 mg/L reduced shoot weight 58% while 0.016 mg/L had no effect.

Phytotoxic Mode of Action. Silver taken up by plants remains in the root system precipitated with phosphate or chloride (Ward et al., 1979). The toxicity of Ag is related to the binding potential of Ag^+ ions to enzymes and other active molecules at cell surfaces (Cooper and Jolly, 1970).

Thallium

Thallium (Tl) is a widely distributed metal, occurring at concentrations of approximately 1 mg/kg in the earth's crust (Kazantzis, 1979). Tl exists as Tl(II) or the more stable, and soluble, Tl(I) and is soluble over a wide range of pH (Kwan and Smith, 1991). Industrial uses of thallium include alloys, electronic devices, special glass and explosives (Zitko, 1975). Coal-fired power plants are major sources of Tl air pollution due to its presence in flyash (Wallwork-Barber et al., 1985). The international market for thallium is limited, therefore its removal from mining effluent is of low priority (Zitko et al., 1975). Thallium has been used since the 1920s as a rodenticide and is a major primary and secondary source of poisoning for raptors and other predatory mammals

(Crabtree, 1962; Robinson, 1948: as cited in Bean and Hudson, 1976). Due to their high toxicity to larger mammals, their use against larger predatory animals was cancelled in 1972 (Zitko, 1975).

Toxicity to Plants in Soil. No primary data were found showing toxicity of Tl to plants grown in soil. Kloke (1979) reported unspecified toxic effects on plants grown in a surface soil with the addition of 1 mg/kg Tl.

Soil-plant uptake factors (concentration in plant/ concentration in soil) for grass and trees/shrubs were calculated at 0.0143 and 0.0038 (Efroymson et al., 1996).

Toxicity to Plants in Solution. The effect of Tl, as $TlCl_3$, on root elongation of 3-week old Norway spruce seedlings grown for 7d in nutrient solution (pH 4) was examined by Lamersdorf et al. (1991). The only concentration tested, 0.02 mg/kg Tl, reduced root elongation by 27%.

The effects of 0.5 to 40 mg/kg Tl, from Tl_2SO_4, on germination and radicle length after 3d growth in solution of radish, cabbage, turnip, lettuce, wheat, and millet were determined by Carlson et al. (1991b). There was no effect on seed germination up to 40 mg/kg Tl. Effective concentrations ranged from 0.5 mg/kg (65% decrease in lettuce radicle length) to 7.5 mg/kg Tl (23% decrease in cabbage radicle length). Carlson et al. (1975) measured 40 and 55% reductions in photosynthesis when corn and sunflower seedlings were grown in nutrient solution containing 1 mg/kg Tl ($TlCl_2$) (lowest concentration tested).

Phytotoxic Mode of Action. Thallium is not essential for plant growth. Soluble Tl is readily taken up by plants and translocated to aerial parts, probably because of its chemical similarity to K. Toxic effects on plants include impairment of chlorophyll synthesis and seed germination, reduced transpiration due to interference in stomatal processes, growth reduction, stunting of roots, and leaf chlorosis (Adriano, 1986).

Zinc

Zinc (Zn) is an essential trace element in all organisms; it assures the stability of biological molecules and structures such as DNA, membranes, and ribosomes (Eisler, 1993). It is used commercially primarily in galvanized metals and metal alloys, but zinc compounds also have wide applications as chemical intermediates, catalysts, pigments, vulcanization activators and accelerators in the rubber industry, UV stabilizers, and supplements in animal feeds and fertilizers. Zinc compounds are also used in rayon manufacture, smoke bombs, soldering fluxes, mordants for printing and dyeing, wood preservatives, mildew inhibitors, deodorants, antiseptics, and astringents (Lloyd, 1984; ATSDR, 1989a). Zinc phosphide is used as a rodenticide. Zinc makes up about 0.002% of the earth's crust (NAS, 1980) and occurs in many forms in natural waters and aquatic sediments.

In freshwater with pH >4 and <7, the dominant forms of dissolved zinc are the free ion (aquo ion complex) (98%) and zinc sulfate (2%) (Campbell and Stokes, 1985), whereas at pH 9.0, the dominant forms are the monohydroxide ion (78%), zinc carbonate (16%), and the free ion (6%) (EPA, 1987-Zn).

Zinc occurs in nature as a sulfide, oxide, or carbonate (Eisler, 1993). It is divalent in solution. Zinc interacts with many chemicals, and it may diminish the toxic effects of cadmium and protect against lead toxicosis in terrestrial animals (Eisler, 1993). Background concentrations seldom exceed 0.040 mg/L in water or 200 mg/kg in soil or sediment (Eisler, 1993).

Although it is essential for normal growth and reproduction (Prasad, 1979; Stahl et al., 1989) and important to central nervous system function (Eisler, 1993), the primary toxic effect of zinc is on zinc-dependent enzymes that regulate RNA and DNA. It is most harmful to aquatic life in conditions of low pH, low alkalinity, low dissolved oxygen, and elevated temperature. Zinc is relatively nontoxic in mammals, but excessive intake can cause a variety of effects. It is not known to be carcinogenic by normal exposure routes (Eisler, 1993).

Toxicity to Plants in Soil. Muramoto et al. (1990) measured the effects of addition of Zn as ZnO to an alluvial soil (pH 6) on root and stem weights, stem length, and grain yield of wheat and rice grown from seed to maturity. Root weight of rice was reduced about 29% by 1000 mg/kg (lowest concentration tested). Wheat grain yield and plant weight were reduced 66 and 28% by 1000 mg/kg (lowest concentration tested).

The effect of Zn on soybean growth has been evaluated. Number of seeds produced per plant was decreased by 28% when plants were grown from seed to maturity in an average garden soil to which 25 mg/kg Zn was added as $ZnSO_4$ (Aery and Sakar, 1991). Zn at 10 mg/kg had no effect. The work of White et al. (1979) shows the ameliorating effect on Zn toxicity of increased pH in a sandy loam soil. Soybean leaf weight was reduced 30% by 131 mg/kg Zn at pH 5.5, while 115 mg/kg had no effect. At pH 6.5, leaf weight was reduced 33% by 393 mg/kg Zn, while 327 mg/kg had no effect.

Lata and Veer (1990) measured 45 and 22% reductions in plant weights of spinach and coriander after 60 days in soil with 87 mg/kg Zn.

Toxicity to Plants in Solution. Carroll and Loneragan (1968) measured effects of Zn on weight of 1-week old seedlings of barrel medic (*Medicago*), subterranean clover, and lucerne (*Medicago*) grown for 46 d in nutrient solution (pH 6). Zinc at 0.41 mg/L reduced weights 80, 40, and 37%, respectively, while 0.08 mg/L had no effect. Rye grass root growth was reduced 63% after 14 d growth in solution (pH 7) containing 1.85 mg/L Zn ($ZnSO_4$) (Wong and Bradshaw, 1982). After 16 d, weights bush bean plant weight was reduced approximately 40% by 6.6 mg/L Zn (as $ZnSO_4$), while 0.66 mg/L had no effect (Wallace et al., 1977c).

Patel et al. (1976) found a 30% decrease in root and stem weights of chrysanthemum seedlings when grown for 21 d in nutrient solution with 6.5 mg/L Zn (as $ZnSO_4$), while 0.65 mg/L had no effect.

Phytotoxic Mode of Action. Zinc is an essential element for plant growth. It has a part in many enzymes, and is involved in disease protection and metabolism of carbohydrates and proteins. Zinc is actively taken up by roots in ionic form, and less so in organically chelated form (Collins, 1981).It is fairly uniformly distributed between roots and shoots being transported in the xylem in ionic form (Wallace and Romney, 1977c). Transport in the phloem appears to be as an anionic complex (van Goor and Wiersma, 1976). Toxicity symptoms include chlorosis and depressed plant growth (Chapman, 1966). It acts to inhibit CO_2 fixation, phloem transport of carbohydrates, and alter membrane permeability (Collins, 1981).

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TOXICITY TO MAMMALS AND BIRDS

INORGANICS

Arsenic

Arsenic (As) is a naturally-occuring metaloid found in air and all living organisms. It is present in the earth's crust at approximately 2 mg/kg and is sparingly soluble in water and body fluids. It occurs as two forms in ambient media, arsenic (III), usually the most toxic, and arsenic (V) (EPA, 1985-As) with its magnitude of bioavailability and toxicity dependent upon the oxidation state and temperature (McGeachy and Dixon, 1992). The state is dependent on environmental conditions, including Eh, pH, organic content, suspended solids and sediment. The relative toxicities of the various forms of arsenic apparently vary from species to species. Arsenic may be released into aquatic ecosystems by anthropogenic sources including the manufacture and use of arsenical defoliants and pesticides, electric generating stations, manufacturing companies, mineral or strip mines, steel production, fossil fuel combustion and smelting operations (Sorensen, 1991; McGeachy and Dixon, 1989; Ferguson and Gavis, 1972; NRCC, 1978) and natural leaching of the soils. Arsenic levels in a river ecosystem were found to be dependent upon the availability of arsenic, rain water dillution, extent of complexation with dissolved organic matter and possibly the metabolic activity of aquatic plants (Koranda et al. 1981). As soil clay concentration increases, arsenic adsorption into the soil increases as a function of soil pH, texture, iron, aluminum, organic matter and time (Woolson, 1977). Arsenic is known as one of the most toxic elements to fish with acute exposures resulting in immediate death (Sorensen, 1991).

Toxicity to Mammals. Tissues of animals generally contain an average of <0.5 mg/kg (Venugopal and Luckey, 1978). Arsenic is a carcinogen and teratogen. Effects include reduced growth, hearing/sight loss, liver/kidney damage, and death (Eisler, 1988). Inorganic arsenic is usually more toxic than organic arsenic compounds. Arsenic may be a required micronutrient; growth, survival, and reproduction of goats is poor if the diet contains <0.05 mg/kg As (NAS, 1977). Wildlife mortality and malformations have been observed for chronic doses of 1-10 mg As/kg bw and dietary concentrations of 5-50 mg/kg (Eisler, 1988). Acute LD50s for mammals of 35–100 mg calcium arsenate/kg body weight and 10-50 mg lead arsenate/kg body weight have been reported (NRCC, 1978).

After a 14d exposure to arsine gas, mice had a significant decrease in red blood cells, haemoglobin, and hematocrit numbers. The spleen to body ration increased from 38 to 236% at 0.5 to 5.0 mg/L As (Hong et al., 1989). The solubility in organic solvents and relative nonpolarity of arsine gas allow it to transverse biologic membranes of stem cells and/or react with sulfhydryl groups of proteins necessary for osmotic balance within erythrocytes (Graham et al., 1946; Levinsky et al. 1970).

Schroeder and Mitchner (1971) exposed mice to 5 mg/kg sodium arsenite in drinking water for three generations. While mice exposed to arsenic survived well, litter size decreased in subsequent generations. A dose of 0.38 mg arsenic/kg over a lifetime was sufficient to cause a slight decrease in the median lifespan of laboratory mice (Schroeder and Balassa, 1967), but it had no effect on growth. As little as 3 mg arsenic trioxide/kg body weight or 1 mg sodium arsenite/kg body weight can be lethal (NAS, 1977).

Because metabolism of arsenic in rats is unlike that in other animals, results of toxicity studies using rats generally should not be extrapolated to other species (Eisler, 1988).

Soil-small mammal uptake factors for *Sigmodon hispidus, Peromyscus leucopus*, and *Oryzomys palustais* were calculated at 0.001, 0.004, and 0.010, respectively (Sample et al., 1996).

Toxicity to Birds. Among birds, LD50s for arsenic compounds range from 17.4 to 3300 mg/kg bw (Eisler, 1988). While no mortality was observed among mallard ducks fed a diet containing 100 mg/kg sodium arsenite for 128 days, 12% to 92% mortality was observed for ducks fed diets containing 250 to 1000 mg/kg arsenite (USFWS, 1964). Camardese et al. (1990) and Whitworth et al. (1991) fed mallards diets containing 30, 100, or 300 mg/kg sodium arsenate. While no effects were observed on behavior, growth was reduced for male ducks consuming 300 mg/kg arsenic and for female ducks at all exposure levels. Arsenic levels in the Dunlin (*Calidris alpina*) paralleled each other in the preegland and kidney which suggests that excretion of the toxicant is via the preegland (Goede and de Bruin, 1985). Swelling of the granular endoplasmic reticulum (GER) of the Coturnix quail suggest that ions accumulate in the interior of the GER with a subsequent influx of water due to a disruption of the normal osmotic balance across the GER membrane. ATP production is reduced through the binding of the citric acid enzymes, and in turn upsets the sodium pump which maintains normal osmotic balance (Bartik and Pisac, 1981; Buchanan, 1962;Clarke and Clarke, 1967; Nystrom, 1984).

Barium

Barium (Ba) is found in the more common mineral forms barite ($BaSO_4$) and witherite ($BaCO_3$) (Oehme, 1979). Barium fluorosilicate and carbonate are forms often used as pesticides. Approximately 400 mg/kg of barium is found in the earth's crust. Some plants accumulate barium from the soil. Barium has industrial applications in areas such as paper manufacturing, fabric printing and dyeing, synthetic rubber production, and drilling fluids. Barium in groundwater has been found to range from 0 to over 20 mg/L (Gilkeson et al., 1983)

Toxicity to Mammals. At low doses, barium acts as a muscle stimulant and at higher doses affects the nervous system of mammals eventually leading to paralysis. The LD50 for rats is listed as 630 mg/kg for barium carbonate, 118 mg/kg for barium chloride, and 921 mg/kg for barium acetate (Lewis and Sweet, 1984).

Schroeder and Mitchener (1975a, b) exposed rats and mice to 5 mg barium/L in drinking water for their lifetime. There was a slight but significant reduction in longevity of treated male mice when measured as the mean age at death of the last surviving 10% of animals. The overall average life span of the group, however, was about the same as the control group. In another study, Perry et al. (1983) exposed rats to 0, 1, 10, or 100 mg/kg barium for up to 16 months. A significant increase in average blood pressure was observed in the highest dose group; a slight but statistically significant increase was seen in the 10 mg/kg dose group. Information on developmental and reproductive toxicity of barium to mammals is not available.

Uptake factors for the small mammals *Sigmodon hispidus* and *Oryzomys palustais* were calculated at 0.003 and 0.023, respectively (Sample et al., 1996).

Toxicity to Birds. The LD50 of barium to chickens is 623 mg/kg (Johnson et al., 1960). While chickens will tolerate 1000 mg/kg barium in their diet without adverse effects, 2000 mg/kg reduces growth, 8000 mg/kg produces 50% mortality in 4 weeks, and diets containing 16,000 or 32,000 mg/kg barium are 100% lethal.

Cadmium

Cadmium (Cd) occurs predominately in the form of free divalent cations in most well oxygenated, low organic matter, fresh waters (EPA 1985-Cd). However, both particulate matter and

dissolved organic matter can bind cadmium in biologically unavailable forms. There is no evidence that cadmium is a biologically essential or beneficial element (Eisler, 1985a). Cd toxicity is related to water hardness, with a reduction in toxicity associated with increased water hardness (EPA, 1985-Cd). Therefore, the cadmium toxicity values presented in Table 2 that are not from tests conducted in waters of moderate hardness are normalized to 100 mg/L using the slopes calculated by the EPA (1985-Cd).

Toxicity to Mammals. While there is little information to indicate that this relatively rare metal is biologically essential or beneficial, Cd has been suggested as the cause of various deleterious effects to wildlife (Eisler 1985a). Mammals and birds are comparatively resistant to the biocidal properties of Cd, which include growth retardation, anemia, and testicular damage. Cd tends to bioaccumulation the liver and kidney, eventually acting as a cumulative toxin. Cd residues of 2 mg/kg whole body fresh weight are evidence of Cd contamination, and residues >5 mg/kg whole animal fresh weight may be life-threatening (Eisler 1985a).

The lowest oral dose resulting in death for rats was 250 mg Cd/kg body weight (EPA, 1980). Weigel et al., (1987) fed rats 0.24, 0.85, or 2.25 mg/kg Cd in diet for 8 weeks. Concentrations $\geq$0.85 mg/kg resulted in reduced food intake, reduced body weights, and reduced enzyme activity, but no hematological effects were noted. Ma et al. (1991) determined that an average cadmium intake of 15 mg/kg/day corresponded with critical renal metal loads of 120 mg/kg, a level indicative of adverse health effects. Rats on a diet with 5 mg/kg Cd suffered shortened lifespans (Schroeder et al. 1965). Cd at 50 mg/kg in the diet depleted iron from rat livers (Whanger, 1973). In a 3 generation reproductive study, the population of mice exposed to 1 mg/kg $CdCl_2$ in their drinking water died out after the second generation (Schroeder and Mitchner, 1971). Rats receiving >6 mg Cd/kg body weight daily during pregnancy gave birth to malformed fetuses (Ferm and Layton, 1981).

Soil-small mammal uptake factors for *Sigmodon hispidus* and *Peromyscus leucopus* were calculated at 0.002 and 0.032, respectively (Sample et al., 1996).

Toxicity to Birds. No mortality was observed among adult mallard ducks fed diets containing 0, 2, 20, and 200 mg/kg Cd, however egg production was significantly reduced in the group consuming 200 ppm Cd (White and Finley, 1978). In addition, the testes of males in the 200 mg/kg Cd group atrophied and the spermatogenic process was disrupted (White et al., 1978). Among mallard ducklings, 20 mg/kg Cd in the diet produces mild to severe kidney lesions, reduces packed cell volume and hemoglobin concentrations in the blood (Cain et al., 1983). Avoidance behavior of black ducklings is impaired by diets containing 40 mg/kg Cd (Heinz and Haseltine, 1983).

Chromium

Chromium (Cr) occurs in the environment as either chromium (III) or chromium (VI). Trivalent chromium is an essential metal in animals, playing an important role in insulin metabolism (Larngard and Norseth, 1979). Hexavalent chromium is more toxic than chromium (III) because of its high oxidation potential and the ease with which it penetrates biological membranes (Steven et al., 1976; Taylor and Parr, 1978). Chromium (III), the predominant form in the environment, exhibits decreasing solubility with increasing pH, and is completely precipitated at a pH above 5.5. In most soils, chromium is primarily present as precipitated chromium (III), which is not bioavailable and has not been know to biomagnify through food chains in its inorganic form (Eisler, 1986b). Chromium is released into the environment in the processing of chromate, electroplating, production at tanning and textile plants, pigment production, and cooling tower preservation. Cr is naturally released into the environment through the weathering of soils (Fishbein, 1976).

Toxicity to Mammals. At high concentrations, chromium is a mutagen, teratogen and carcinogen (Eisler, 1986b). The LD50 for chromium (III) in mice is 260 mg/kg bw and 5 mg/kg BW for chromium (VI). Rats fed Cr(VI) reached a toxic threshold at 1000 mg/kg (Steven et al., 1976). Similar results were observed among rats consuming water containing 25 mg/L Cr(VI) for 1 year (Mackenzie et al., 1958). Tissue accumulation of hexavalent Cr was nine times higher than trivalent chromium.

Soil-small mammal uptake factors for *Sigmodon hispidus, Peromyscus leucopus*, and *Oryzomys palustais* were calculated at 0.001, 0.030, and 0.121, respectively (Sample et al., 1996).

Toxicity to Birds. Chromium is of concern to aquatic birds due to its tendency to accumulate in the roots which are a highly selected food. Although weight gain, egg laying, fertility and embryonic mortality were unaffected at both concentrations tested (10 and 50 mg/kg Cr(III) for 10 months), black duck hens experienced higher mortality as reproductive season approached and those in the higher concentration group were unable to raise as large a proportion of their broods to 10 weeks of age (Haseltine et al., unpublished manuscript). While no malformations were observed among ducklings from treated birds, growth and survivorship was reduced. Physiological effects in the adult ducks included moderate to severe kidney damage in 33% of the ducks tested and mild nephrosis in 33%. Blood examination revealed alteration in packed cell volume and haemoglobin count, both proving to increase with increased doseage.

Heinz and Haseltine (1981) observed no effects on avoidance behavior from fright stimulus of black ducklings fed a diet containing 20 or 200 mg/kg Cr(III).

Copper

Copper (Cu) occurs in natural waters primarily as the divalent cupric ion in free and complexed forms (EPA 1985-Cu). Copper is a minor nutrient for both plants and animals at low concentrations, but is toxic to aquatic life at concentrations only slightly higher. Concentrations of 0.001 to 0.010 mg/L are usually reported for unpolluted surface waters in the United States. Common copper salts, such as the sulfate, carbonate, cyanide, oxide, and sulfide are used as fungicides, as components of ceramics and pyrotechnics, for electroplating, and for numerous other industrial applications (ACGIH, 1986). The largest anthropogenic releases of copper to the environment result from mining operations, agriculture, solid waste, and sludge from sewage treatment plants.

Toxicity to Mammals. Copper is a component of a number of metalloenzymes such as catalase, peroxidases, and cytochrome oxidase and is essential for the utilization of iron (Goyer, 1991; Stokinger, 1981). Although most copper salts occur in two valence states, as cuprous (Cu^{+}) or cupric (Cu^{2+}) ions, the biological availability and toxicity of copper is most likely associated with the divalent state (ATSDR, 1990). Copper sulfate is the most common copper salt. Copper is soluble in nitric acid and hot sulfuric acid, slightly soluble in hydrochloric acid and ammonia, and insoluble in water (Stokinger, 1981).

The metabolism of copper involves mainly its transfer to and from various organic ligands, most notably sulfhydryl and imidazole groups on amino acids and proteins (ATSDR, 1990). The liver is one of the main organs involved in the storage and metabolism of copper. Absorption of ingested copper occurs primarily in the upper gastrointestinal tract (EPA, 1987-Cu). Soluble copper compounds (oxides, hydroxides, citrates) are readily absorbed but water-insoluble compounds

(sulfides) are poorly absorbed (Venugopal and Luckey, 1978). Zinc, molybdenum, and other metals may decrease dietary copper absorption (USAF, 1990a).

In animal studies, oral exposure to copper caused hepatic and renal accumulation of copper, liver and kidney necrosis at doses of ≥100 mg/kg/day, and hematological effects at doses of 40 mg/kg/day (EPA, 1986-Cu; Haywood, 1985; Rana and Kumar, 1978; Gopinath et al., 1974; Kline et al., 1971). Oral or intravenous administration of copper sulfate can increase fetal mortality and developmental abnormalities in experimental animals (Lecyk, 1980; Ferm and Hanlon, 1974). Rat oral LD50 values for various copper compounds are 140 mg/kg for copper chloride ($CuCl_2$); 470 mg/kg for copper oxide (Cu_2O); 940 mg/kg for copper nitrate ($Cu(NO_3)_2 \cdot 3H_2O$); and 960 mg/kg for copper sulfate ($CuSO_4 \cdot 5\ H_2O$) (Stokinger, 1981). Deaths in animals given lethal doses of copper have been attributed to extensive hepatic centrilobular necrosis (USAF, 1990a).

In a 90-day subchronic study with copper cyanide (CuCN), high mortality, attributed to hemolytic anemia, was seen in both male and female rats receiving 50 mg/kg/day by gavage, but not in those receiving ≤5 mg/kg/day (EPA 1986-Cu). In general, male rats appeared to be more sensitive to the effects of CuCN than female rats. Rats receiving 500 mg/kg copper in their diet (about 5 mg/day) appeared normal, while rats receiving 1000 mg/kg exhibited depressed growth, those at 2000 mg/kg hardly grew at all, and those on a 4000 mg/kg diet lost weight rapidly and died (Boyden et al., 1938). Salt licks containing 5–9% copper sulfate caused anorexia, hemolytic anemia, icterus, and hemoglobinuria, followed by death within 2 days in sheep using the licks (Gopinath et al., 1974). The estimated ingested dose was 40–49 g over a 25- to 86-day period. Lecyk (1980) observed reduced litter size, decreased fetal weights, and skeletal abnormalities in the offspring of mice fed diets supplemented with 3000 or 4000 mg/kg copper sulfate (155 or 207 mg copper/kg/day, respectively) for one month prior to gestation and on days 0–19 of gestation.

Aulerich et al., (1982) reported an increased mortality rate in the offspring of minks fed a diet supplemented with >3 mg copper/kg/day as copper sulfate for 50 weeks. Although kit mortality was greater and litter mass was reduced relative to controls, reproductive performance of mink fed diets supplemented with up to 200 mg/kg copper for 357 days was within the normal range for the species (Aulerich et al., 1982). Lifetime exposure to 42.4 mg copper/kg/day (as copper gluconate) in drinking water caused a 12.8% decrease in the maximal lifespan in mice (Massie and Aiello, 1984).

Soil-small mammal uptake factors for *Sigmodon hispidus, Peromyscus leucopus*, and *Oryzomys palustais* were calculated at 0.001, 0.388, and 26.498, respectively (Sample et al., 1996).

Toxicity to Birds. Domestic chicks on diets ≥324 mg/kg copper grew slowly; mortality increased with dietary copper concentrations of 1270 mg/kg (Mayo et al., 1956). Arthur et al., (1958) observed no ill effects in chicks fed ≤500 mg/kg copper in diet up to 8 weeks of age. Dietary copper levels from 588–1176 mg/kg for 10 weeks exerted a toxic effect on chick growth; the minimum toxic level of copper appeared to be about 500 mg/kg (Mehring et al., 1960). Turkey poults tolerated 676 mg/kg copper in starter diets for 21 days with no deleterious effects, but copper was definitely toxic at levels >1620 mg/kg (Vohra and Kratzer, 1968). Chickens given a daily dose of >70 mg/kg of $CuCO_3$ died while those receiving <60 mg/kg exhibited slight symptoms of copper poisoning but survived (Pullar, 1940). No symptoms of copper poisoning were observed in domestic mallards ingesting ≤29 mg/kg/day of $CuCO_3$, but daily intakes ≥55 mg/kg/day were toxic (Pullar, 1940).

Mercury

Mercury (Hg) has no known biological function and is toxic to fish and wildlife. It is a mutagen and carcinogen that adversely affects the central nervous, renal, and reproductive systems of wildlife. Hg occurs in the environment as elemental mercury, Hg_2(II) and Hg(II), the latter of which is naturally oxidized from elemental mercury (Eisler, 1987). Mercury in ambient waters commonly occurs as mercury (II) or methylmercury. Mercury (II) can be methylated by both aerobic and anaerobic bacteria in the slime coat, liver, and intestines of fish, but methylation apparently does not occur in other tissues or in plants (EPA, 1985-Hg). From a toxicological standpoint, methylmercury (MeHg) poses a greater threat to biota due to its high stability and the ease with which it penetrates membranes in living organisms. Hg(II), however, is more prevalent in aquatic systems, bound up and unavailable in sediment layers. Biota bioconcentrate mercury compounds which can be further biomagnified through food chains (Wren, 1986). High concentrations of mercury in water are often associated with low alkalinity lakes and newly created bodies of water (Weiner and Stokes, 1990). Alkalinity, ascorbic acid, chloride, dissolved oxygen, hardness, organic complexing agents, pH, sediment, and temperature probably affect the acute and chronic toxicity and bioaccumulation of the various forms of mercury.

Anthropogenic sources of Hg include the combustion of fossil fuels, metal mining and processing plants, chloralkali plants, and disposal of batteries and fluorescent lamps (NAS 1978, and Das et al., 1982; as cited in Eisler, 1987).

Toxicity to Mammals. Daily doses of 0.1–0.5 mg/kg bw/day and dietary concentrations of 1.0–5.0 mg/kg are lethal to sensitive mammals (Eisler, 1987). Central nervous system toxicity, weight loss, and mortality were observed among rats fed a diet containing 250 mg/kg methyl mercury (MeHg) for 2 weeks (Verschuuren et al., 1976a). Rats consuming 2.5 mg/kg MeHg in the diet for 2 years displayed reduced growth, increased kidney weight, and altered kidney histochemistry (Verschuuren et al., 1976b). To study effects on reproduction, Verschuuren et al. (1976c) fed rats a diet containing 0, 0.1, 0.5, and 2.5 mg/kg MeHg for three generations. While no effects were observed among rats fed 0.1 or 0.5 mg/kg MeHg, offspring viability was reduced among rats in the 2.5 mg/kg treatment. Among mink, 93-day consumption of diets containing 1.8 to 15.0 mg/kg MeHg produced mortality, ataxia, anorexia, and paralysis (Wobeser et al., 1976), with the highest exposures showing the greatest effects.

Soil-small mammal uptake factors for *Peromyscus leucopus*, and *Oryzomys palustais* on the Oak Ridge Reservation were calculated at 0.035, and 1.13, respectively (Sample et al., 1996).

Toxicity to Birds. The acute LD50 for MeHg for *Coturnix* quail ranges from 14.4 to 33.7 mg/kg bw (Eisler, 1987). Growth was decreased and mortality increased among leghorn cockerels fed diets containing 6 to 18 mg/kg MeHg (Fimreite, 1970). Ring-necked pheasants fed diets of MeHg-treated grains displayed reduced egg production and hatchability and laid more shell-less eggs than controls (Fimreite, 1971). Heinz (1979) fed mallard ducks a diet containing 0.5 mg/kg MeHg for three generations. While MeHg consumption did not affect adult weights or weight change during the reproductive season, MeHg-exposed females laid fewer eggs (with more eggs outside the nest box), produced fewer young, and produced slightly thinner eggshells. Young of MeHg-treated adults were less responsive to maternal calls and hyper-responsive to fright stimuli.

Nickel

Nickel is a naturally occuring element that may exist in various mineral forms. It forms 0.008% of the earth's crust (NAS, 1980). Soil and sediment are the primary receptacles for nickel,

but mobilization may occur depending on physico-chemical characteristics of the soil (ATSDR, 1988; USAF, 1990). Nickel is used in a wide variety of applications including metallurgical processes and electrical components, such as batteries (ATSDR, 1988; USAF, 1990b). There is some evidence that nickel may be an essential trace element for mammals. Nickel occurs in nature in the nonionic and divalent states; other valence states occur very infrequently (Mastromatteo, 1986). Although nickel can exist in several oxidation states, the divalent cation state predominates and is generally considered the most toxic form (EPA 1986-Ni). As with many metals the toxicity of nickel increases as hardness decreases. Fish and invertebrates have approximately the same range of sensitivity.

Toxicity to Mammals. The absorption of nickel is dependent on its physico-chemical form, with water soluble forms being more readily absorbed. Soluble nickel compounds tend to be more toxic than insoluble compounds (Goyer, 1991). The metabolism of nickel involves conversion to various chemical forms and binding to various ligands (ATSDR, 1988). Nickel is excreted in the urine and feces with relative amounts for each route being dependent on the route of exposure and chemical form. Most nickel enters the body via food and water consumption.

Oral LD50 values for rats range from 67 mg nickel/kg (nickel sulfate hexahydrate) to >9000 mg nickel/kg (nickel powder) (ATSDR, 1988). Toxic effects of oral exposure to nickel usually involve the kidneys with some evidence from animal studies showing a possible developmental/reproductive toxicity effect (ATSDR, 1988; Goyer, 1991).

Inorganic nickel compounds are well-tolerated when taken orally by rodents in doses up to 500 mg/kg (Mastromatteo, 1986). Rats continually fed a 250 ppm nickel diet for 16 months suffered no deleterious effects and were considered in excellent condition (Phatak and Patwardhan, 1952). Progressive accumulation of nickel was not observed in the tissues assayed. In a three-generation study of rats, Ambrose et al. (1976) reported a no-observed-adverse-effects level (NOAEL) and lowest-observed-adverse-effects level (LOAEL) of 5 mg/kg/day and 50 mg/kg/day, respectively. Doses of 24.15 mg/kg-day administered as nickel sulfate in the diet had no adverse effects on reproduction of the rats. Growth in dogs was depressed by dietary concentrations of 2500 ppm nickel sulfate hexahydrate; in the rats, growth was depressed at dietary concentrations >1000 ppm (Ambrose et al., 1976).

Toxicity to Birds. Weber and Reid (1968) fed a basal diet of up to 1300 ppm nickel sulfate or nickel acetate to domestic chicks for 4 weeks. Growth of chicks was significantly depressed at 700 ppm nickel and above. Doses of 21.4 mg/kg-day administered as nickel sulfate in the diet had no adverse effects on weight gain after 4 weeks. Mallard ducklings on diets with ≥800 ppm nickel would be adversely affected (Cain and Pafford, 1981).

Selenium

Selenium (Se), is an essential nutrient for some plants and animals when present in trace amounts. However, at levels currently present in the environment, Se poses a significant toxic risk. Anthropogenic sources include the combustion of fossil fuels, municipal wastes, irrigation run-off and industrial effluents. Lechate from seleniferous soils also contributes to the presence of Se in the environment (Eisler, 1985b). Selenium has a combination of attributes which make it an unusual pollutant (EPA, 1987-Se). These attributes include, but are not limited to, the following: it is an essential trace nutrient; it can occur in three oxidation states and be reduced or oxidized by various organisms; it is a metalloid with physicochemical properties similar to sulfur, which may reduce the toxicity of selenium or be replaced by selenium in biologically important compounds; it can reduce the toxicity of several heavy metals and have varying effects on cadmium and mercury toxicity; both

water and food are important exposure pathways for aquatic biota; and there are substantial natural and anthropogenic releases to water. The two oxidation states (Se(IV) and Se(VI)) for which the most toxicity data exist are discussed separately. Plants have the ability to convert inorganic selenium to organic selenium compounds, thereby increasing their biological availability (Lo and Sandi, 1980).

Toxicity to Mammals. While selenium is an essential nutrient that interacts with Vitamin E and maintains muscle integrity, it has a very narrow tolerance range (Eisler, 1985b). In mammals, chronic selenium poisoning is induced by diets containing 1–44 mg/kg selenium (Harr, 1978). Symptoms include liver cirrhosis, lameness, loss of hair, emaciation, reduced conception, and increased fetal resorption. Plants convert inorganic selenium to organic selenium compounds, thereby increasing their biological availability (Lo and Sandi, 1980).

To evaluate the effects of selenium on reproduction, Schroeder and Mitchner (1971) exposed mice to 3 mg/L selenate in drinking water for three generations. This dosage level increased juvenile mortality, number of runts, and resulted in reproductive failure by the third generation. In another study, exposure to 3 mg/L selenate or selenite in water for a lifetime had no effect on mouse longevity and no tumorigenicity was observed (Schroeder and Mitchner, 1972).

Raccoons on the Kesterson National Wildlife Refuge in California were found to bioaccumulate selenium (Clark et al., 1989). While peak births at the refuge was 2 months later than reported at other locations, no adverse effects on raccoon reproduction were observed.

Soil-small mammal uptake factors for *Peromyscus leucopus*, and *Oryzomys palustais* were calculated at 0.143 and 0.109, respectively (Sample et al., 1996). These factors are unitless quotients of concentration in the whole animal/ concnetration in soil, all on a dry weight basis.

Toxicity to Birds. Selenium is both embryotoxic and teratogenic to birds, with organic selenium (selenomethionine) being more toxic than inorganic selenium (Hoffman and Heinz, 1988). Mallard ducks were fed diets containing 1, 5, 10, 25, or 100 mg/kg selenite (Heinz et al., 1987) or 1, 2, 4, 8, or 16 mg/kg selenomethionine (Heinz et al., 1989) for about 10 weeks. Exposure to 1, 5, or 10 mg/kg selenite or 1, 2, or 4 mg/kg selenomethionine in the diet had no effect on survival, growth, or reproductive success of adults. The diet containing 100 mg/kg selenite killed 11 of 12 adults. While only one adult receiving the 25 mg/kg diet died, time to laying and the interval between eggs was increased, and duckling survivorship was reduced in this treatment (Heinz et al., 1987). Diets containing 8 and 16 mg/kg selenomethionine resulted in 6.8% and 67.9% malformed embryos, respectively. In addition, duckling survival was significantly reduced (Heinz et al., 1989).

The most visible incident of environmental selenium toxicity occurred at the Kesterson National Wildlife Refuge. Agricultural wastewater containing approximately 0.3 mg/L selenium was used for marsh management at the refuge (Ohlendorf et al., 1986). Mean selenium concentrations in plants, invertebrates, and fish at the site were 22–175 mg/L (dry weight). As a result, reproductive success among water birds was poor, and the incidence of embryo mortality and developmental abnormalities was dramatically increased.

Metabolism of selenium may be significantly modified through interactions with heavy metals, and selenium may provide some protection from adverse effects associated with various metals, including cadmium and mercury (Eisler, 1985b). Arsenite inhibits methylation of selenium but increases fecal excretion of selenite (Venugopal and Luckey, 1978).

Thallium

Thallium (Tl) is a widely distributed metal, occurring at concentrations of approximately 1 mg/kg in the earth's crust (Kazantzis, 1979). Tl exists as Tl(II) or the more stable, and soluble, Tl(I) and is soluble over a wide range of pH (Kwan and Smith, 1991). Industrial uses of thallium include alloys, electronic devices, special glass and explosives (Zitko, 1975). Coal-fired power plants are major sources of Tl air pollution due to its presence in flyash (Wallwork-Barber et al., 1985). The international market for thallium is limited, therefore its removal from mining effluent is of low priority (Zitko et al., 1975). Thallium has been used since the 1920s as a rodenticide and is a major primary and secondary source of poisoning for raptors and other predatory mammals (Crabtree, 1962; Robinson, 1948: as cited in Bean and Hudson, 1976). Due to their high toxicity to larger mammals, their use against larger predatory animals was cancelled in 1972 (Zitko, 1975).

Toxicity to Mammals. Thallium sulfate, which has been widely used as a rodenticide, has a rat acute oral LD50 of 16 mg/kg (Ware, 1978). In chronic studies, rats tolerated a dose of 10 mg Tl acetate/ kg, while 30 mg/kg was lethal to males by 15 weeks. All rats fed a daily dose of 0.45 mg Tl/kg died after 4 months (Kazantzis, 1979). Rats exposed to 10 mg/L Tl in drinking water for 2 mo accumulated Tl in testis and exhibited signs of testicular toxicity including reduced sperm motility (Formigli et al., 1986).

Being chemically isomorphic to potassium, Tl interferes with the potassium dependent activities of the cell (Cavanagh et al., 1974). For instance, Tl affinity to ATPase is ten times greater than that of K which puts the active transport systems at high risk to the presence of Tl (Gehring and Hammond, 1966).

Sample et al. (1996) calculated a soil-mammal uptake factor for *Peromyscus peucopus* at 0.027.

Toxicity to Birds. Bean and Hudson (1976) orally dosed 3 golden eagles with 60 and 120 mg Tl_2SO_4/kg bw; the bird receiving 60 mg Tl_2SO_4/kg survived while the two dosed with 120 mg Tl_2SO_4/kg died, suggesting an LD50 between the doses. Oral LD50s for quail, geese, and ducks are 12, 15, and 30 mg/kg respectively (Shaw, 1933). No long-term studies of thallium toxicity to birds are currently available.

Zinc

Zinc (Zn) is an essential trace element in all organisms; it assures the stability of biological molecules and structures such as DNA, membranes, and ribosomes (Eisler, 1993). It is used commercially primarily in galvanized metals and metal alloys, but zinc compounds also have wide applications as chemical intermediates, catalysts, pigments, vulcanization activators and accelerators in the rubber industry, UV stabilizers, and supplements in animal feeds and fertilizers. Zinc compounds are also used in rayon manufacture, smoke bombs, soldering fluxes, mordants for printing and dyeing, wood preservatives, mildew inhibitors, deodorants, antiseptics, and astringents (Lloyd, 1984; ATSDR, 1989a). Zinc phosphide is used as a rodenticide. Zinc makes up about 0.002% of the earth's crust (NAS, 1980) and occurs in many forms in natural waters and aquatic sediments.

In freshwater with pH >4 and <7, the dominant forms of dissolved zinc are the free ion (aquo ion complex) (98%) and zinc sulfate (2%) (Campbell and Stokes, 1985), whereas at pH 9.0, the

dominant forms are the monohydroxide ion (78%), zinc carbonate (16%), and the free ion (6%) (EPA, 1987-Zn).

Zinc occurs in nature as a sulfide, oxide, or carbonate (Eisler, 1993). It is divalent in solution. Zinc interacts with many chemicals, and it may diminish the toxic effects of cadmium and protect against lead toxicosis in terrestrial animals (Eisler, 1993). Background concentrations seldom exceed 0.040 mg/L in water or 200 mg/kg in soil or sediment (Eisler, 1993).

Although it is essential for normal growth and reproduction (Prasad, 1979; Stahl et al., 1989) and important to central nervous system function (Eisler, 1993), the primary toxic effect of zinc is on zinc-dependent enzymes that regulate RNA and DNA. It is most harmful to aquatic life in conditions of low pH, low alkalinity, low dissolved oxygen, and elevated temperature. Zinc is relatively nontoxic in mammals, but excessive intake can cause a variety of effects. It is not known to be carcinogenic by normal exposure routes (Eisler, 1993).

Toxicity to Mammals. Gastrointestinal absorption of zinc is variable (20–80%) and depends on the chemical compound as well as on zinc levels in the body and on dietary concentrations of other nutrients (EPA, 1984). Information on pulmonary absorption is limited and complicated by the potential for gastrointestinal absorption due to mucociliary clearance from the respiratory tract and subsequent swallowing. Pulmonary inflammation and changes in lung function have been observed in inhalation studies on animals (Amur et al., 1982; Lam et al., 1985; Drinker and Drinker, 1928). Zinc is present in all tissues with the highest concentrations in the prostate, kidney, liver, heart, and pancreas. Zinc is a vital component of many metalloenzymes such as carbonic anhydrase, which regulates CO_2 exchange (Stokinger, 1981).

In animals, gastrointestinal and hepatic lesions (Allen et al., 1983; Brink et al., 1959), pancreatic lesions (Maita et al., 1981; Drinker et al., 1927), anemia (ATSDR, 1989a; Fox and Jacobs, 1986; Maita et al., 1981), and diffuse nephrosis (Maita et al., 1981; Allen et al., 1983) have been observed following subchronic oral exposures. Anemia and pancreatitis were the major adverse effects observed in chronic animal studies (Aughey et al., 1977; Drinker et al., 1927; Walters and Roe, 1965; Sutton and Nelson, 1937). Teratogenic effects have not been seen in animals exposed to zinc; however, high oral doses can affect reproduction and fetal growth (Ketcheson et al., 1969; Schlicker and Cox, 1967; Sutton and Nelson, 1937).

Livestock and small mammals are tolerant of extended dietary loadings >100 times the minimum recommended daily zinc requirement (Eisler, 1993). No adverse effects on general health or reproduction were observed in dairy cows fed 1310 mg zinc/kg food (Miller et al., 1989). A diet of 4000–5000 mg zinc/kg food for 18 days resulted in fetotoxicity and poor reproduction in rats (NAS, 1979). Acute oral LD50 doses of 350–800 mg zinc/kg body weight have been reported for rats (Eisler, 1993). Wlostowski et al. (1988) recommended 30 mg zinc/kg in the diet of bank voles.

Dogs on diets with up to 1000 mg zinc/kg of food for up to one year showed no measurable signs of damage (NAS, 1979). Horses ingesting >90 mg zinc/kg body weight daily in the vicinity of a lead-zinc smelter exhibited decreased growth and death (NAS, 1979). No effects were observed in mice fed <682 mg zinc/kg food (<109 mg zinc/kg body weight daily) for 13 weeks, but at 6820 mg zinc/kg food adverse effects on growth and survival were documented (Maita et al., 1981). In a 37-day study involving rats, doses of 97 mg/kg-day administered as zinc carbonate in the diet had no adverse effects on the reproductivity of rats (Kinnamon, 1963). European ferrets (*Mustela putorius furo*) fed up to 500 mg zinc/kg for up to 197 days all survived with no significant histopathologies, but those fed 1500 or 3000 mg/kg diet died within 21 days (Straube et al., 1980;

Reece et al., 1986). Reproduction ceased entirely in female rats ingesting a diet with 500 mg zinc/kg/day (Sutton and Nelson, 1937), possibly a result of zinc-induced anemia.

Toxicity to Birds. Mallards (*Anas platyrhynchos*) fed diets containing >3000 mg zinc/kg for 30 or 60 days suffered leg paralysis, decreased food consumption, and high mortality (Gasaway and Buss, 1972; NAS, 1979). Egg production in Japanese quail (*Coturnix coturnix japonica*) hens fed 15,000 mg zinc (as ZnO)/kg feed for 7 days decreased to near zero within 3 days (Hussein et al., 1988). Seven percent of 14-day old quail fed 600 mg zinc (as zinc phosphide)/kg feed over 5 days died, 53% of those fed 990 mg/kg died, and 93% of those fed 1634 mg/kg died (Hill and Camardese, 1986). Domestic chicken pullets and hens on a diet with 20,000 mg zinc/kg feed for 5 days were lighter weight by day 5 and produced significantly fewer eggs for 4 weeks following treatment (Palafox and Ho-A, 1980). Eggs collected 14–28 days post-treatment had reduced fertility and hatchability. However, normal growth, egg production, fertility, and hatchability was observed 4–12 weeks post-treatment. Acute oral LD50 values for zinc phosphide, a rodenticide, were between 16 and 47 mg/kg body weight in ring-necked pheasants (*Phasianus colchicus*), golden eagles (*Aquila chrysaetos*), mallards, and horned larks (*Eremophila alpestris*) (Hudson et al., 1984), but much of the biocidal action is attributed to the phosphide rather than the zinc (Eisler, 1993).

Diets containing 28, 48, 228, or 2028 mg zinc/kg for 12–44 weeks had no effect on overall egg production by domestic chickens although zinc levels were elevated in hens on the highest zinc diet (Stahl et al., 1990). All day-old chicks fed diets containing 16,000 mg zinc/kg feed for 5 weeks and 80% of those fed 8000 mg/kg died; those on a 4000 mg zinc/kg diet showed no significant reductions in growth or survival (Oh et al., 1979). In a 60-day study, doses of 170 mg/kg-day administered as zinc carbonate in the diet caused increased mortality and altered blood chemistry in mallards (Gasaway and Buss, 1972).

In chickens, adverse effects associated with zinc deficiency have been observed at <38 mg zinc/kg dry weight feed (Blamberg et al., 1960; Westmoreland and Hoekstra, 1969; Stahl et al., 1989), but concentrations of 93–120 mg/kg are suggested as adequate in the diet (Blamberg et al., 1960; Westmoreland and Hoekstra, 1969). Greater than 178 mg/kg dry weight feed is considered excessive (Stahl et al., 1989), and dietary concentrations >2000 mg/kg dry weight feed are considered toxic (NAS, 1979; Oh et al., 1979; Stahl et al., 1990). Turkey poults tolerated zinc levels up to 2000 ppm in starter diets for 21 days with no deleterious effects, but levels ≥4000 ppm resulted in marked growth depression (Vohra and Kratzer, 1968). No mortality was observed in poults on a diet containing 10,000 ppm zinc (Vohra and Kratzer, 1968), but increased mortality has been observed for chickens on diets with 3000 ppm zinc (Roberson and Schaible, 1960).

PCBs

Polychlorinated biphenyls (PCBs) are a family of man-made chemicals consisting of 209 individual compounds with varying toxicity (ATSDR 1989b). Aroclor is the trade name for PCBs made by Monsanto. Because of their insulating and nonflammable properties, PCBs were widely used in industrial applications such as coolants and lubricants in transformers, capacitors, and electrical equipment (ATSDR 1989b). The United States stopped manufacturing PCBs in 1977 due to evidence that they accumulate in the environment. PCBs have become widespread environmental contaminants.

Toxicity to Mammals. Most exposures to PCBs are oral. Absorption of PCBs following oral exposure is often >90% (ATSDR 1989b). PCBs are preferentially stored in adipose tissues in animals. They may cross the placenta or be transferred to offspring through milk. PCBs with higher

chlorine content (the last 2 digits of the Aroclor designation indicate the percent Cl content of the compound) tend to persist in the environment longer than those with lower Cl content, and PCBs are known to bioaccumulate and biomagnify to toxic concentrations in animals (Eisler 1986a, ATSDR 1989b). Chronic exposures are of particular concern. PCBs with high K_{ow} values and high numbers of chlorines in adjacent positions are generally the most toxic. Although relatively insoluble in water, PCBs are generally freely soluble in nonpolar organic solvents and in biological lipids (EPA 1980).

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