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Zinc Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review

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TABLES

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Abstract. Ecological and toxicological aspects of zinc in the environment are reviewed with emphasis on natural resources. Subtopics include sources and uses; chemical and biochemical properties; carcinogenicity, mutagenicity, teratogenicity; background concentrations in biological and nonbiological compartments; effects of zinc deficiency; toxic and sublethal effects on terrestrial plants and invertebrates, aquatic organisms, birds, and mammals; and recommendations for the protection of sensitive resources.

The estimated world production of zinc is 7.1 million metric tons; the United States produces about 4% of the total and consumes 14%. Zinc is used primarily in the production of brass, noncorrosive alloys, and white pigments; in galvanization of iron and steel products; in agriculture as a fungicide and as a protective agent against soil zinc deficiency; and therapeutically in human medicine. Major sources of anthropogenic zinc in the environment include electroplaters, smelting and ore processors, mine drainage domestic and industrial sewage, combustion of solid wastes and fossil fuels, road surface runoff corrosion of zinc alloys and galvanized surfaces, and erosion of agricultural soils.

Zinc has its primary effect on zinc-dependent enzymes that regulate RNA and DNA. The pancreas and bone are primary targets in birds and mammals; the gill epithelium is a primary target site in fish. Dietary zinc absorption is highly variable in animals; in general, it increases with low body weight (BW) and low zinc status and decreases with excess calcium or phytate and by deficiency of pyridoxine or tryptophan. Low molecular weight proteins called metallothioneins play an important role in zinc homeostasis and in protection against zinc poisoning; zinc is a potent inducer of metallothioneins. Zinc interacts with many chemicals to produce altered patterns of accumulation, metabolism, and toxicity; some interactions are beneficial to the organism and others are not depending on the organism, its nutritional status, and other variables. Knowledge of these interactions is essential to the understanding of zinc toxicokinetics.

In natural waters, dissolved zinc speciates into the toxic aquo ion $[Zn(H_2O)_6]^{2+}$, other dissolved chemical species, and various inorganic and organic complexes zinc complexes are readily transported. Aquo ions and other toxic species are most harmful to aquatic life under conditions of low pH, low alkalinity, low dissolved oxygen, and elevated temperatures. Most of the zinc introduced into aquatic environments is eventually partitioned into the sediments. Zinc bioavailability from sediments is enhanced under conditions of high dissolved oxygen, low salinity low pH, and high levels of inorganic oxides and humic substances.

Zinc and its compounds induce testicular sarcomas in birds and rodents when injected directly into the testes; however zinc is not carcinogenic by any other route. Growth of animal tumors is stimulated by zinc and retarded by zinc deficiency. Under some conditions, excess zinc can suppress carcinoma growth, although the mechanisms are imperfectly understood. Organozinc compounds are effective mutagens when presented to susceptible cell populations in an appropriate form; the evidence for the mutagenic potential of inorganic zinc compounds is incomplete. Zinc deficiency can lead to chromosomal aberrations, but excess zinc was not mutagenic in the majority of tests. Excess zinc is teratogenic to frog and fish embryos, but conclusive evidence of teratogenicity in higher vertebrates is lacking. In mammals, excess zinc may protect against some teratogens. Zinc deficiency may exacerbate the teratogenic effects of known teratogens, especially in diabetic animals.

Background concentrations of zinc seldom exceed 40 µg/L in water 200 mg/kg in soils and sediment, or 0.5 $\mu g/m^3$ in air. Environments heavily contaminated by anthropogenic activities may contain up to 99 mg Zn/L in

water, 118 g/kg in sediments, 5 g/kg in soft, and 0.84 µg/m^3 in air. Zinc concentrations in field collections of plants and animals are extremely variable and difficult to interpret. Most authorities agree on six points: (1) elevated concentrations (i.e., >2 g Zn/kg fresh weight [FW]) are normally encountered in some species of oysters, scallops, barnacles, red and brown algae, and terrestrial arthropods; (2) concentrations are usually <700 mg Zn/kg dry weight (DW) tissue in fish, <210 mg Zn/kg DW tissue in birds, and <210 mg Zn/kg DW tissue in mammals; (3) concentrations are higher in animals and plants collected near zinc-contaminated sites than in the same species collected from more distant sites; (4) zinc content in tissue is not proportionate to that of the organism's immediate surroundings; (5) for individual species, zinc concentration varies with age, sex, season, tissue or organ, and other variables; and (6) many species contain zinc loadings far in excess of immediate needs, suggesting active zinc regulation.

The balance between excess and insufficient zinc is important. Zinc deficiency occurs in many species of plants and animals and has severe adverse effects on all stages of growth, development, reproduction, and survival. In humans, zinc deficiency is associated with delayed sexual maturation in adolescent males; poor growth in children; impaired growth of hair, skin, and bones; disrupted vitamin A metabolism; and abnormal taste acuity, hormone metabolism, and immune function. Severe zinc deficiency effects in mammals are usually prevented by diets containing >30 mg Zn/kg DW ration. Zinc deficiency effects are reported in aquatic organism at nominal concentrations between 0.65 and 6.5 µg Zn/L of medium and in piscine diets at <15 mg Zn/kg FW ration. Avian diets should contain >25 mg Zn/kg DW ration for prevention of zinc deficiency effects and <178 mg kg DW for prevention of marginal sublethal effects.

Sensitive terrestrial plants die when soil zinc levels exceed 100 mg/kg (oak and maple seedlings), and photosynthesis is inhibited in lichens at >178 mg Zn/kg DW whole plant. Sensitive terrestrial invertebrates have reduced survival when soil levels exceeded 470 mg Zn/kg (earthworms), reduced growth at >300 mg Zn/kg diet (slugs), and inhibited reproduction at >1,600 mg Zn/kg soil (woodlouse). The most sensitive aquatic species were adversely affected at nominal water concentrations between 10 and 25 µg/L, including representative species of plants, protozoans, sponges, molluscs, crustaceans, echinoderms, fish, and amphibians. Acute LC50 (96 h) values were between 32 and 40,930 µg/L for freshwater invertebrates, 66 and 40,900 µg/L for freshwater teleosts, 195 and >320,000 µg/L for marine invertebrates, and 191 and 38,000 µg/L for marine teleosts. Acute toxicity values were markedly affected by the age and nutrient status of the organism, by changes in the physicochemical regimen, and by interactions with other chemicals, especially copper salts. Pancreatic degeneration occurred in ducks fed diets containing 2,500 mg Zn/kg ration. Ducks died when fed diets containing 3,000 mg Zn/kg feed or when given single oral doses >742 mg Zn/kg BW. Domestic poultry are routinely fed extremely high dietary levels of 20 g Zn/kg ration as a commercial management technique to force the molting of laying hens and the subsequent improvement of long-term egg production that molting produces. However, poultry chicks died at 8 g Zn/kg diet, had reduced growth at 2-3 g Zn/kg diet, and experienced pancreas histopathology when fed selenium-deficient but zinc-adequate (100 mg Zn/kg) diets. Mammals are comparatively resistant to zinc, as judged by their tolerance of extended periods on diets containing >100 times the minimum daily zinc requirement. But excessive zinc through inhalation or ingestation harms mammalian survival, metabolism, and well-being. The most sensitive species of mammals were adversely affected at dietary concentrations of 90 to 300 mg Zn/kg, drinking water concentrations >300 mg Zn/L, daily intakes >9 mg Zn/kg BW, single oral doses >350 mg Zn/kg BW, and air concentrations >0.8 mg Zn/m^3. Humans are comparatively sensitive to excess zinc. Adverse effects occur in humans at >80 mg Zn/kg diet or at daily intakes >2.3 mg/kg BW.

Proposed criteria for protection of aquatic life include mean zinc concentrations of <47 to <59 µg/L in freshwater and <58 to <86 µg Zn/L in seawater. Results of recent studies, however, show significant adverse effects on a growing number of freshwater organisms in the range of 5 to 51 µg Zn/L and on saltwater biota between 9 and 50 µg Zn/L, suggesting that some downward modification in the proposed criteria is necessary.

Although tissue residues are not yet reliable indicators of zinc contamination, zinc poisoning usually occurs in birds when the liver or kidney contains >2.1 g Zn/kg DW and in mammals when concentrations exceed 274 mg Zn/kg DW in kidney, 465 mg Zn/kg DW in liver, or 752 mg Zn/kg DW in pancreas. The proposed air quality criterion for human heath protection is <5 mg Zn/m^3, but guinea pigs were more sensitive and adverse effects were evident at >0.8-4.0 mg/m^3.

Current research needs include the development of protocols to (1) separate, quantitate, and verify the different chemical species of zinc (2) identify natural from anthropogenic sources of zinc; (3) establish toxicity thresholds based on accumulation; (4) evaluate the significance of tissue concentrations in target organs as indicators of zinc stress; and (5) measure the long-term consequences of zinc interactions with other nutrients in animals of various age and nutrient status. (Eisler, R. 1993. Zinc hazards to fish, wildlife, and invertebrates: a synoptic review. U. S. Fish and Wildlife Service Biological Report 10. 106 pp.).

Key words: Zinc, metals, toxicity, deficiency, criteria, residues, agriculture, nutrition, metallothionein, fish, invertebrates, birds, wildlife, livestock.

Zinc is an essential trace element for all living organisms. As a constituent of more than 200 metalloenzymes and other metabolic compounds, zinc assures stability of biological molecules such as DNA and of biological structures such as membranes and ribosomes (Vallee 1959; National Academy of Sciences [NAS] 1979; Casey and Hambidge 1980; Mason et al. 1988; Llobet et al. 1988b; Leonard and Gerber 1989). Plants do not grow well in zinc-depleted soils, and deficiency has resulted in large losses of citrus in California and pecans in Texas (Vallee 1959). Clinical manifestations of zinc deficiency in animals include growth retardation, testicular atrophy, skin changes, and poor appetite (Prasad 1979). The ubiquity of zinc in the environment would seem to make human deficiencies unlikely; however, reports of zinc-associated dwarfism and hypogonadism in adolescent males are now confirmed (Casey and Hambidge 1980) and reflect the fact that much of their dietary zinc is not bioavailable. Zinc deficiency was a major factor in the syndrome of nutritional dwarfism in adolescent males from rural areas of Iran and Egypt in 1961--about 3% of the population in these areas was affected--and a similar syndrome was found in Turkey, Tunisia, Morocco, Portugal, and Panama (Casey and Hambidge 1980). The use of unleavened bread as a major staple food contributed to severe zinc deficiency in the Middle East. Unleavened bread may contain adequate amounts of zinc for nutrition, and intakes may exceed recommended allowances by a wide margin; however, zinc is largely unavailable for absorption because of the high levels of fiber and phytic acid esters in unleavened bread (Casey and Hambidge 1980). Marginal deficiency of zinc in hums is probably widespread and common throughout the world, including the United States (Prasad 1979). Dietary zinc replacement usually reverses the pathologic events of zinc depletion in humans and animals (NAS 1979). But zinc repletion seems to be of little value in rat offspring with congenital malfunctions or behavioral abnormalities associated with zinc depletion (NAS 1979).

Zinc poisoning has been documented in dogs, cats, ferrets, birds, cattle, sheep, and horses, usually as a result of ingesting galvanized metal objects, certain paints and fertilizers, zinc-containing coins, and skin and sunblock preparations containing zinc oxide (Wentink et al. 1985; Ogden et al. 1988; Lu and Combs 1988a; Binnerts 1989; Robinette 1990). Signs of acute poisoning include anorexia, depression, enteritis, diarrhea, decreased milk yield, excessive eating and drinking and, in severe cases, convulsions and death (Ogden et al. 1988). Emissions from zinc smelters at Palmerton, Pennsylvania, destroyed wildlife habitat; reduced prey abundance; poisoned deer, songbirds, and shrews; and eliminated terrestrial amphibians from the mountainside at Lehigh Gap (Beyer et al. 1985; Sileo and Beyer 1985; Beyer 1988). Aquatic populations are frequently decimated in zinc-polluted waters (Solbe and Flook 1975; Everall et al. 1989b). Zinc in the aquatic environment is of particular importance because the gills of fish are physically damaged by high concentrations of zinc (NAS 1979).

Zinc toxicosis in humans is not a common medical problem, although it may appear in some metal workers and others under special conditions (NAS 1979). Industrial processes such as welding, smelting, or fabrication of molten metals can produce ultrafine metal oxides at harmful concentrations. Inhalation of these metal oxides, including oxides of zinc, causes the industrial malady known as metal fume fever (Lain et al. 1985; Lu and Combs 1988a; Llobet et al. 1988b). Symptoms occur several hours after exposure and include fever, chills, perspiration, tachycardia, dyspnea, and chest pains. Recovery is normally complete within 24 h, but susceptible workers can have persistant pulmonary impairment for several days after exposure (Lain et al. 1985). Most reports of human zinc intoxication have been in response to food poisoning from lengthy storage of acidic foods or beverages in galvanized containers (Llobet et al. 1988b; Fosmire 1990).

Historically zinc has been used by humans for industrial, ornamental, or utilitarian purposes for nearly 2,000 years and may have been used as an ointment to treat skin lesions by the ancient Egyptians and other Mediterraneans (NAS 1979). In biblical times, the Romans were known to have produced brass by mixing copper with a zinc ore (Elinder 1986). In its isolated form, zinc was not recognized until the 15th century when smelting occurred accidentally (NAS 1979). The Chinese probably were the first to extract zinc metal, although its first description in 1597 by an occidental traveler, Liborius, related that the process was observed in India (Vallee 1959). Commercial smelting began in the 18th century when it was realized that zinc could be obtained from the calamine ore used to make brass; no reports of zinc toxicosis in any form were recorded from these early accounts (NAS 1979). The first documented use of orally administered zinc was in 1826 to treat discharges from various body orifices (NAS 1979). Zinc was recognized as an essential nutrient for plants and animals in 1869. Its occurrence in biological matter, for example, human liver, was first described in 1877 (Vallee 1959). In 1934, zinc was conclusively demonstrated to be essential to normal growth and development in animals (Prasad 1979).

Zinc composes 0.004% of the earth's crust and is 25th in order of abundance of the elements (Vallee 1959). Uses of zinc include the production of noncorrosive alloys, galvanizing steel and iron products, and the therapeutic treatment of zinc deficiency (Elinder 1986). Zinc is found in coal and many manufactured products such as motor oils, lubricants, tires, and fuel oils (NAS 1979).

Ecological and toxicological aspects of zinc in the environment have been reviewed by many authorities, including Vallee (1959), Skidmore (1964), NAS (1979), Prasad (1979, 1980), U.S. Environmental Protection Agency [EPA] (1980, 1987), Nriagu (1980), Weatherley et al. (1980), Eisler (1981), Spear (1981), Apgar (1985), Elinder (1986), Vymazal (1986), Greger (1989), U.S. Public Health Service [PHS] (1989), and Sorensen (1991).

This report is part of a series of synoptic reviews on hazards of selected chemicals to plants and animals with emphasis on fishery and wildlife resources. It was prepared in response to requests for information on zinc from environmental specialists of the U.S. Fish and Wildlife Service.

Sources and Uses

World production of zinc increased from 0.5 million metric tens in 1900 to 6.1 million metric tons in 1978 (Elinder 1986) and 7.1 million metric tons in 1987 (PHS 1989). The principal ores of zinc are sulfides, such as sphalerite and wurtzite (Elinder 1986). The major world producers include Canada, the former Soviet Union, and Japan--which collectively account for about half the production--and, secondly, the United States, Australasia, Mexico, and Peru (Weatherley et al. 1980; Elinder 1986). Zinc is now available as ingots, lumps, sheets, wire, shot, strips, granules, and powder (PHS 1989). The United States produced 240,000 metric tons of zinc in 1987--mostly from Tennessee, Mississippi, and New York but also from 16 other states--and imported an additional 774,000 metric tons, thus consuming 14% of the world zinc production while producing 3.4% (PHS 1989).

Zinc is mainly used in the production of noncorrosive alloys and brass and in galvanizing steel and iron products. Zinc undergoes oxidation on the surface, thus protecting the underlying metal from degradation. Galvanized products are widely used in construction materials, automobile parts, and household appliances (Elinder 1986). Zinc oxide is used to form white pigments in rubber processing and to coat photocopy paper (EPA 1987; PHS 1989). Zinc sulfate is used as a cooperative agent in fungicides and as a protective agent against zinc deficiency in soils. When incorporated with copper compounds or arsenic-lead wettable powders and applied by spraying, it can minimize the toxic effects of these metals on fruits such as plums, apples, and peaches; in Japan alone, about 250 metric tons of zinc sulfate is sprayed in fields each year (Maita et al. 1981). Zinc is used therapeutically in human medicine in the treatment of zinc deficiency, various skin diseases, wound healing, and to reduce pain in sickle cell anemia patients (Prasad 1979; Spear 1981; EPA 1987; Warner et al. 1988).

Zinc is discharged into the global environment at an estimated yearly rate of 8.8 million metric tons; 96% of the total is a result of human activities (Leonard and Gerber 1989). Major sources of anthropogenic zinc discharges to the environment include electroplaters, smelting and ore processors, drainage from active and inactive mining operations, domestic and industrial sewage, combustion of fossil fuels and solid wastes, road surface runoff, corrosion of zinc alloys and galvanized surfaces, and erosion of agricultural soils (Weatherley et al. 1980; Spear 1981; Mirenda 1986; Llobet et al. 1988a; Buhl and Hamilton 1990). During smelting, large amounts of zinc are emitted into the atmosphere. In the United States alone during 1969, about 50,000 metric

tons of zinc were discharged into the atmosphere during smelting operations (Elinder 1986). Another 20,000 metric tons are discharged annually into U.S. estuaries (Table 1). Zinc is also dispersed from corroded galvanized electrical transmission towers for at least 10 km by runoff and by wind-driven spray and water droplets from the towers (Jones and Burgess 1984). Discharges from placer mining activities usually contain 75-165 µg Zn/L, sometimes up to 882 µg Zn/L in active mines, and these concentrations may represent acute hazards to salmonids in areas downstream of placer mine effluents (Buhl and Hamilton 1990). In Maine, galvanized culverts significantly increased zinc concentrations in stream waters, particularly in newer culverts. Zinc concentrations in culverts were highest during elevated temperatures and low flow; levels of zinc sometimes exceeded the avoidance threshold (0.05 mg/L) of Atlantic salmon (*Salmo salar*); invertebrates seemed unaffected, except for a freshwater sponge, *Spongilla* sp. (Gregory and Trial 1975). Zinc sources implicated in livestock poisonings include galvanized iron wire and troughs and zinc-containing fertilizers and fungicides (Allen et al. 1983; Reece et al. 1986). Zinc toxicosis in humans has been reported from consumption of milk stored in galvanized vessels and from food contaminated with particles of zinc from a zinc pigment plant (Zee et al. 1985). Zinc toxicity is discussed later in greater detail.

Table 1. Estimated annual zinc inputs to U.S. coastal marine ecosystems; study area comprised 116,000 km^2 (Young et al 1980).

Source	Metric tons per year
Rivers	5,950
Atmosphere	4,300
Barged wastes	3,490
Storm channels	3,060
Municipal wastewater	2,500
Direct industrial discharges	710
Vessel protection	360
Dredging release	10
Thermal discharges	2
Groundwater	1
Total	20,383

Chemical and Biochemical Properties

General

Most of the zinc introduced into aquatic environments eventually is partitioned into the sediments. Zinc release from sediments is enhanced under conditions of high dissolved oxygen, low salinity and low pH. Dissolved zinc usually consists of the toxic aquo ion ($Zn(H_2O)_6^{2+}$) and various organic and inorganic complexes. Aquo ions and other toxic species have their greatest effects on aquatic organisms under conditions of comparatively low pH, low alkalinity, low dissolved oxygen, and elevated temperatures.

Zinc has its primary metabolic effect on zinc-dependent enzymes that regulate RNA and DNA. Low molecular weight proteins, metallothioneins, play an important role in zinc homeostasis and in protection against zinc poisoning in animals; zinc is a potent inducer of metallothioneins. The pancreas and bone seem to be primary targets of zinc intoxication in birds and mammals; gill epithelium is the primary target site in fish.

Effects of excess zinc on natural resources are modified by numerous variables, especially by interactions with other chemicals. Interactions frequently produce radically altered patterns of accumulation, metabolism, and toxicity some of which are beneficial to the organism whereas others are harmful.

Chemical Properties

Zinc is a bluish-white metal that dissolves readfly in strong acids. In nature, it occurs as a sulfide, oxide, or carbonate. In solution, it is divalent and can form hydrated Zn^{2+} cations in acids, and zincated anions--probably $Zn(OH)_4^{-2}$-- in strong bases (EPA 1980, 1987). Zinc dust and powder are sold commercially under a variety of trade names: Asarco, Blue powder, CI 77949, CI pigment metal 6, Emanay zinc dust, granular zinc, JASAD Merrillite, L15, and PASCO (PHS 1989). Selected physical and chemical properties of zinc, zinc chloride, and zinc sulfate are listed in Table 2.

Because zinc ligands are soluble in neutral and acidic solutions, zinc is readily transported in most natural waters (EPA 1980, 1987). But zinc oxide, the compound most commonly used in industry, has a low solubility in most solvents (Elinder 1986). Zinc mobility in aquatic ecosystems is a function of the composition of suspended and bed sediments, dissolved and particulate iron and manganese concentrations, pH, salinity, concentrations of complexing ligands, and the concentration of zinc (EPA 1980). In freshwater, zinc is most soluble at low pH and low alkalinity: 10 mg Zn/L of solution at pH 6 that declines to 6.5 mg Zn/L at pH 7, 0.65 mg Zn/L at pH 8, and 0.01 mg/L at pH 9 (Spear 1981). Dissolved zinc rarely exceeds 40 µg/L in Canadian rivers and lakes; higher concentrations are usually associated with zinc-enriched ore deposits and anthropogenic activities. Marine waters usually contain <10 µg Zn/L, most adhering to suspended solids; however, saturated seawater may contain 1.2 - 2.5 mg Zn/L (Spear 1981).

In water the free zinc ion is thought to coordinate with six water molecules to form the octahedral aquo ion $(Zn(H_2O)_6)^{2+}$ in the absence of other complexing or adsorbing agents (Spear 1981). In freshwater zinc exists almost exclusively as the aquo ion at pH >4 and <7 (Campbell and Stokes 1985). In freshwater at pH 6, the dominant forms of dissolved zinc are the free ion (98%) and zinc sulfate (2%); at pH 9 the dominant forms are the monohydroxide ion (78%), zinc carbonate (16%), and the free ion (6%; EPA 1987). In typical river waters, 90% of the zinc is present as aquo ion and the remainder consists of $ZnHCO_3^+$, $ZnCO_3$, and $ZnSO_4$ (Spear 1981).

Table 2. Some properties of zinc, zinc chloride, and zinc sulfate (PHS 1989).

Property	Zinc	Zinc chloride	Zinc sulfate
Formula	Zn	$ZnC1_2$	$ZnSO_4$
CAS number	7440-66-6	7646-85-7	7733-02-0
Molecular weight	65.38	136.29	161.44
Melting point, ° C	419.5	290	Decomposes at 600
Boiling point, °C	908	732	
Density	7.14	2.907	3.54
Physical state	Bluish-white lustrous solid	Solid white granules	Colorless solid
Solubility	Insoluble in water, soluble in acetic acid and alkali	61.4 g/L water, 769 g/L alcohol, 500 g/L glycerol	Soluble in water, slightly soluble in alcohol

Zinc bioavailability and toxicity to aquatic organisms are highest under conditions of low pH, low alkalinity, low dissolved oxygen, and elevated temperatures (Weatherley et al. 1980). Soluble chemical species of zinc are the most bioavailable and most toxic (Spear 1981). The aquo ion predominates over other dissolved species and is suspected of being most toxic; however, aquo ion concentrations decrease under conditions of high

alkalinity, at pH >7.5, and increasing salinity (Spear 1981). Under conditions of high alkalinity and pH 6.5, the most abundant species are $ZnHCO_3^+$, Zn^{2+}, and $ZnCO_3$; at low alkalinity and an elevated pH 8.0, the descending order of abundance was Zn^{2+}, $ZnCO_3$, zinc humic acid, $ZnOH^+$, and $ZnHCO_3^+$ (Spear 1981). Water hardness is the principal modifier of acute zinc toxicity. Increased alkalinity or water hardness decreases toxicity to freshwater organism when all zinc is dissolved; this effect is associated with decreased concentration of aquo ions and is heightened by increased pH. Increased water hardness at pH <8.5 when zinc is in suspension increases toxicity associated with increased suspended $ZnCO_3$. Increased water hardness at pH >8.5 when zinc is in suspension decreases toxicity and increases suspended $Zn(OH)_2$. Suspended zinc carbonate may also be toxic, although its toxicity decreases under conditions suitable to zinc hydroxide formations; suspended $Zn(OH)_2$ is relatively nontoxic. Thus, $ZnCO_3$ composes <1% of the dissolved zinc at low pH and low alkalinity but is the predominant chemical species at high pH and high alkalinity. Organozinc complexes are not stable and under reducing conditions may dissociate, liberating Zn^{2+} (Spear 1981).

In seawater zinc exists in a dissolved state, as a solid precipitate, or adsorbed to particle surfaces. Soluable zinc in seawater exists as uncomplexed free (hydrated) ions, as inorganic complexes (the primary form in the open sea), or as organic complexes (Young et al. 1980). In seawater at pH 8.1, the dominant species of soluble zinc are zinc hydroxide (62%), the free ion (17%), the monochloride ion (6.4%), and zinc carbonate (5.8%). At pH 7, the percentage of dissolved zinc present as the free ion increases to 50% (EPA 1987). In the presence of dissolved organic materials, most of the dissolved zinc is present as organozinc complexes (EPA 1987). In estuaries and other marine environments, the relative abundance of zinc species changes with increasing salinity. At low salinities, $ZnSO_4$ and $ZnC1^+$ predominate; at higher salinities, the aquo ion predominates (Spear 1981). But as salinity decreases, the concentration of free zinc ion increases and the concentration of zinc-chloro complexes decreases, resulting in increased bioavailability of the free metal ion and increased bioconcentration by resident organisms (Nugegoda and Rainbow 1989b).

In solution, zinc is adsorbed by organic agents such as humic materials and biogenic structures (i.e., cell walls of plankton) and by inorganic adsorbing agents such as mineral particles, clays, and hydrous oxides of manganese, iron, and silicon (Spear 1981). Particulate materials in the medium may contain as little as 2% and as much as 100% of the total zinc (Sprague 1986). Formation of zinc-ligand complexes increases the solubility of zinc and probably increases the tendency for zinc to be adsorbed (EPA 1980). Sorption to particulates was lower at higher salinities because of displacement of sorbed zinc ions by alkali and alkaline earth cations (EPA 1987). Increased pH increases zinc sorption to particulates and seems to be independent of water salinity or hardhess (EPA 1987).

Most of the zinc introduced into aquatic environments is sorbed onto hydrous iron and manganese oxides, clay minerals, and organic materials and eventually is partitioned into the sediments (EPA 1987). Zinc is present in sediments as precipitated zinc hydroxide, ferric and manganic oxyhydroxide precipitates, insoluble organic complexes, insoluble sulfides, and other forms. As the sediments change from a reduced to an oxidized state, soluble zinc is mobilized and released however, the bioavailability of different forms of sediment zinc varies substantially and the mechanisms of transfer are poorly understood (EPA 1987). Sorption to sediments was complete at pH >7, but was negligible at pH <6 (EPA 1987). Zinc is dissolved from sediments at low salinities because of displacement of adsorbed zinc ions by alkali and alkaline earth cations that are abundant in brackish waters (EPA 1980). Sulfide precipitation in sediments is an important control of zinc mobility in reducing environment; precipitation of the hydroxide, carbonate, or sulfate may occur when zinc is present in high concentrations (EPA 1980).

Extractable concentrations of sediment-bound zinc positively correlated with zinc concentrations in deposit feeding clams (Luoma and Bryan 1979). Availability of sediment zinc to bivalve molluscs was higher at increased sediment concentrations of amorphous inorganic oxides or humic substances and lower at increased concentrations of organic carbon and ammonium acetatemanganese. Zinc uptake by euryhaline organisms was enhanced at low water salinity (Luoma and Bryan 1979).

Metabolism

Zinc is ubiquitous in the tissues of plants and animals (Rosser and George 1986) and is essential for normal growth, reproduction, and wound healing (Prasad 1979; Stahl et al. 1989a). More than 200 different enzymes require zinc for maximum catalytic activity, including carbonic anhydrase, alkaline phosphatase, alcohol dehydrogenase, acid phosphatase, lactic dehydrogenase, carboxypeptidase, and superoxide dismutase (Prasad 1979, 1980; Casey and Hambidge 1980; Rosser and George 1986; Blesbois and Mauger 1989; Thompson et al. 1989). Zinc has its primary effect on zinc-dependent enzymes that regulate the biosynthesis and catabolic rate of RNA and DNA (Prasad 1979; Casey and Hambidge 1980; Gipouloux et al. 1986; Sternlieb 1988). Zinc exerts a protective effect on liver by inhibiting lipid peroxidation and stabilizing lysosomal membranes (Sternlieb 1988); aids neurotransmission in the brain of fish, birds, reptiles, and mammals (Smeets et al. 1989); prolongs muscular contractions; increases oxygen affinity of myoglobin; is necessary for the growth and differentiation of muscle fiber types (Rosser and George 1986); increases numbers and birthweights of lambs of zinc-supplemented ewes; is essential for wound healing in most studied organisms (Ireland 1986); and is used therapeutically in treating patients with skin diseases, zinc deficiency and other symptoms (Mooradian et al. 1988; Sternlieb 1988).

Zinc enters the gastrointestinal tract as a component of low molecular weight proteins secreted by the salivary glands, intestinal mucosa, pancreas, and liver (Goyer 1986). Usually, only dissolved zinc is sorbed or bound. But zinc dissolution probably occurs in the alimentary tract of animals after ingestion of particulates containing undissolved zinc (EPA 1987). After ingestion, zinc is absorbed across several physiologically active membranes: gut mucosa, alveocapillary membranes, and tissue and organ membranes. The exact transport mechanism are unknown but may be associated with formation of a tetrahedral quadredentate ligand with a small organic molecule (NAS 1979). Some of the zinc taken up by the intestinal epithelial cells is rapidly transferred to the portal plasma where it associates with albumin, 2 macroglobulin, and amino acids; about 67% of the zinc in plasma is bound to albumin and about 3% is stored in the liver (Sternlieb 1988). Soluble organozinc complexes are passively absorbed across the plasma membrane of the mucosa of the intestinal villi; the soluble, nondiffusable complexes are transported in the intestinal products and excreted in feces (NAS 1979). Zinc loss from urine and sweat is usually small (Casey and Hambidge 1980). In a normal human adult about 2 g zinc is filtered by the kidneys daily and about 0.3-0.6 mg is actually excreted each day (Goyer 1986). Zinc homeostasis in rats, unlike in most mammals, is maintained by zinc secretion from the intestines rather than by regulation of zinc absorption (Elinder 1986). Initial uptake of zinc from the rat gastrointestinal tract involves binding to albumin and transport of the zinc-albumin complex from the intestines to the liver (Hoadley and Cousins 1988).

Foods rich in zinc include red meat, milk, gelatin, egg yolks, shellfish, liver, whole grain cereals, lentils, peas, beans, and rice (Sternlieb 1988). About 20-30% of zinc in the diet is absorbed, but this is highly variable and ranges from <10% to >90% (Prasad 1979; Casey and Hambidge 1980; Elinder 1986). Increased zinc absorption, for example, was associated with low body weight (BW), poor zinc status, and various prostaglandins; decreased absorption was caused by excess dietary calcium or phytate and by a deficiency of pyridoxine or tryptophan (Elinder 1986; Goyer 1986). The half-time persistence of zinc in most mammalian tissues is between 100 and 500 days; it is longer in bone and muscle and shorter in the liver (Elinder 1986).

Metallothioneins play an important role in metal homeostasis and in protection against heavy metal toxicity in vertebrates and invertebrates (Engel 1987; Overnell et al. 1987a; Andersen et al. 1989; Olsson et al. 1989; Richards 1989b; Eriksen et al. 1990). Metallothioneins are cysteine-rich (>20%), low (about 6,000) molecular weight proteins with a high affinity for copper, silver, gold, zinc, copper, and mercury. These heat-stable, metal-binding proteins are in all vertebrate tissues and are readily inducible by a variety of agents to which they bind through thiolate linkages. Zinc is a potent inducer of metallothioneins, and a redistribution of zinc from enzymes to metallothioneins is one way to maintain low intracellular zinc concentrations. Metallothioneins also serve as temporary storage proteins for zinc and other metals during early development and may function by maintaining the pool of available zinc at an appropriate concentration. Metallothioneins are quite similar among organisms, that is, all metallothioneins are small proteins of molecular weight 6,000-10,000, rich in sulfur and cysteine, and lack aromatic amino acids (Sprague 1986). Metallothioneins isolated from cattle, sheep, horses, pigs, and other livestock contain 61 amino acids; thioneine, the metal-free protein, is a single chain polypeptide with a molecular weight of about 6,000 (Richards 1989b). Chicken thioneine consists of 63 amino acids, including histidine, an

amino acid not present in mammalian metallothioneins. The unusually high cysteine content enables metallothioneins to selectively bind up to 7 zinc and 12 copper atoms per mole of protein (Richards 1989a).

Metallothioneins are involved in zinc homeostasis in the chick, rat, and calf. When zinc is present at high dietary concentrations, a temporary zinc storage protein aids in counteracting zinc toxicity (Oh et al. 1979). Zinc absorption in mice is directly proportional to intestinal metallothionein levels and implies a significant role of metallothionein in zinc absorption (Starcher et al. 1980). Chick embryo hepatic metallothionein is highly responsive to exogenous zinc introduced into the yolk and increases in a dose-dependent manner; a similar pattern is evident in turkey development (Fleet and McCormick 1988). Zinc protects against subsequent exposure to zinc insult, and protection is believed to be mediated by metallothioneins (Woodall et al. 1988). For example, preexposure of South African clawed frog (*Xenopus laevis*) tadpoles to 5 mg $ZnSO_4$ ($7H_2O$)/L for 96 h resulted in no deaths during subsequent exposure to 15 mg Zn/L for 90 h but in 45% deaths in the nontreated group; at 20 mg Zn/L, 15% died in the pretreated group versus 50% in the nontreated group (Woodall et al. 1988). Metallothioneins are an important factor in zinc regulation during the period of exogenous vitellogenesis in rainbow trout (*Oncorhynchus mykiss*). In female rainbow trout, for example, metallothioneins maintain homeostasis of hepatic zinc during egg formation (Olsson et al. 1989). In plaice (*Pleuronectes platessa*), a marine fish, intraperitoneal injection of zinc raised hepatic metallothionein-ike species by a factor of 15; metallothionein levels remained elevated for the next 4 weeks (Overnell et al. 1987a).

In marine molluscs and crustaceans, excess zinc is usually sequestered by metal-binding proteins and subsequently transported to storage or detoxification sites; soluble proteins and amino acids may contain 20-70% zinc (Sprague 1986). Metallothioneins are actively involved in zinc regulation during normal growth processes in the blue crab (*Callinectes sapidus*), as judged by a decrease in zinc content in the hemolymph and the digestive gland during molting (Engel 1987).

Elevated metallothionein levels are not necessarily indicative of heavy metal insult. Starcher et al. (1980) show that liver metallothionein levels in mice are elevated after acute stress or starvation and that this effect is blocked by actinomycin D, a protein synthesis inhibitor. It is further emphasized that not all zinc-binding proteins are metallothioneins (Webb et al. 1985; Andersen et al. 1989; Richards 1989a; Eriksen et al. 1990). Low molecular weight metal-binding proteins---not metallothioneins--were induced in snails and polychaete annelids in metals-contaminated environments (Andersen et al. 1989). A high molecular weight protein fraction was detected in the plasma of laying turkey (*Meleagris gallopavo*) hens that bound significant amounts of zinc and that coeluted with vitellogenin; vitellogenin, a metalloprotein, from laying hens contained 0.54 mg Zn/kg protein (Richards 1989a). In rock oysters (*Saccostrea cucullata*) collected near an iron-ore shipping terminal, some of the tissue zinc was bound to a high molecular weight (around 550,000), iron-binding protein called ferritin (Webb et al. 1985). Ferritin accounts for about 40% of the protein-bound zinc in rock oysters and most probably in other bivalves containing elevated tissue levels of zinc (Webb et al. 1985); however, this requires verification. In four species of sediment-feeding marine polychaete annelids, zinc was mainly associated with high molecular weight proteins, suggesting that metallothionein-like proteins may not be satisfactory for monitoring purposes and that other cytosolic components should be studied (Eriksen et al. 1990).

High zinc levels induce copper deficiency in rats and interfere with metabolism of calcium and iron (Goyer 1986). Excess zinc interferes with normal metabolism of the pancreas, bone, gall bladder and kidney in mammals and gill in fish. The pancreas is a target organ for zinc toxicity in birds and mammals. Pancreatic alterations are documented from experimentally produced zinc toxicosis in cats, sheep, dogs, calves, chickens, and ducklings and naturally in sheep and calves. Pancreatic changes were limited to acinar cells, specifically cytoplasmic vacuolation, cellular atrophy, and eventually cell death (Lu and Combs 1988a; Kazacos and Van Vleet 1989). Excess zinc may cause stimulation of bone resorption and inhibition of bone formation in chicks, dogs, monkeys, and rats (Kaji et al. 1988). By preferentially accumulating in bone, zinc induces osteomalacia--a softening of the bone caused by deficiency of calcium, phosphorus, and other minerals (Kaji et al. 1988). Zinc plays a role in bone metabolism of aging rats (Yamaguchi et al. 1989b). Normally, the femoral zinc diaphysis content in rats increases from 50 to 150 mg/kg fresh weight (FW) during the first 3 weeks of life and remains constant thereafter. Oral administration of zinc (5-20 mg/kg BW daily for 3-day-old to 28-week-old rats) increased alkaline phosphatase activity and calcium content in the femur and delayed bone deterioration in aging rats (Yamaguchi et al. 1989b). Its high affinity for electrons causes zinc to bind covalently to proteins, mostly at imidazole and cysteine residues. In the mud puppy (*Necturus maculosus*), zinc blocks apical membrane anion exchange in gallbladder epithelium and blocks chloride channels in nerve and muscle cells.

The slow onset and reversal of the effects suggest a covalent modification of the exchanger or an effect requiring Zn^{2+} transport to the cell interior (Kitchens et al. 1990).

Zinc toxicity to aquatic organisms is dependent on the physical and chemical forms of zinc, the toxicity of each form, and the degree of interconversion among the various forms. Aquatic plants and fish are relatively unaffected by suspended zinc, but many aquatic invertebrates and some fish may be adversely affected from ingesting enough zinc-containing particulates (EPA 1987). Zinc toxicosis affects freshwater fish by destruction of gill epithelium and consequent tissue hypoxia. Signs of acute zinc toxicosis in freshwater fish includes osmoregulatory failure, acidosis and low oxygen tensions in arterial blood, and disrupted gas exchange at the gill surface and at internal tissue sites (Spear 1981). Zinc exerts a critical influence on mammalian and piscine immune systems (Ghanmi et al. 1989). Lymphocytes from the pronephros of common carp (*Cyprinus carpio*) were transformed by various mitogenetic agents; zinc added to lymphocyte cultures enhanced thymidine incorporation and inhibited the response of the mitogenetic agents--although Zn^{2+} itself was toxic at these concentrations (650 µgZn^{2+}/L; Ghanmi et al. 1989).

Interactions

Zinc interacts with numerous chemicals. The patterns of accumulation, metabolism, and toxicity from these interactions sometimes greatly differ from those produced by zinc alone. Recognition of these interactions is essential to the understanding of zinc kinetics in the environment.

Cadmium

Calcium-zinc interactions are typical because sometimes they act to the organism's advantage and sometimes not, depending on the organism, its nutritional status, and other variables.

Dietary cadmium accentuates signs of zinc deficiency in turkeys, chicks, rodents, and pigs (NAS 1979). Chicks on a zinc-deficient diet showed an increased frequency of muscle and feather abnormalities when 40 mg Cd/kg diet was added; however, supplementation of the diet with 200 mg Zn/kg for 14-15 days lessened or reversed the adverse effects of cadmium (Supplee 1963). But cadmium promotes the growth of zinc-limited phytoplankton (Price and Morel 1990). Substitution of trace metals or metalloenzymes could be a common strategy for phytoplankton in trace-metal impoverished environments such as the ocean and could result in an effective colimitation of phytoplankton growth by several bioactive elements (Price and Morel 1990). Zinc-deficient marine diatoms (*Thalassiosira weissflogii*), for example, can grow at 90% of their maximum rate when supplied with cadmium (which substitutes for zinc in certain macromolecules); cobalt can also substitute for zinc, although less efficiently than cadmium (Price and Morel 1990).

Zinc diminishes or negates the toxic effects of cadmium. Specifically, zinc protected embryos of the toad (*Bufo arenarum*) and other amphibian embryos against cadmium-induced developmental malformations (Herkovits et al. 1989; Herkovits and Perez-Coll 1990; Rivera et al. 1990). Zinc counteracted adverse effects of cadmium on limb regeneration and on the growth of the fiddler crab (*Uca pugilator*; Weis 1980). Preexposure of a freshwater amphipod (*Gammarus pulex*) to 10 µg Zn/L for 2 weeks increased whole body zinc content from 74 to 142 mg/kg dry weight (DW) and protected against the toxic effects of subsequent cadmium exposure of 500 µg Cd/L for 96 h (Howell 1985). In crickets (*Acheta domesticus*), excess zinc in diets of larvae protected against cadmium toxicity (Migula et al. 1989). Zinc protected rats (*Rattus* sp.) against the toxic effects of cadmium such as testicular lesions, reduced sperm counts, hepatotoxicity, and lung damage (Sato and Nagai 1989; Saxena et al. 1989a). Zinc protected mouse (*Mus* sp.) embryos against cadmium toxicity (Yu and Chan 1988. An effective protection ratio of cadmium to zinc was 1:1 for mouse embryos, but for free living embryos of the toads, this ratio of cadmium to zinc was 1:8 (Belmonte et al. 1989). Zinc reversed the toxic action of cadmium on natural killer cells of mice: 500 mg Zn/L drinking water negated the toxic action of 50 mg Cd/L (Chowdhury and Chandra 1989). The mechanisms of zinc protection against cadmium were variously attributed to metallothionein induction (Sato and Nagai 1989), enhanced detoxification rates of cadmium (Rivera et al. 1990), and competition with cadmium for the same metalloenzyme sites (Yu and Chan 1988; Rivera et al. 1990).

Waterborne solutions of zinc-cadmium mixhires were usually additive in toxicity to aquatic organisms, including freshwater fish (Skidmore 1964) and amphipods (de March B. G. E. 1988), and to marine fish (Eisler and Gardner 1973), copepods (Verriopoulos and Dimas 1988), and amphipods (Ahsanullah et al. 1988).

However, mixtures of zinc and cadmium were less toxic than expected to *Daphnia magna*, as judged by acute lethality studies (Attar and Maly 1982).

Zinc exerted antagonistic effects on uptake of cadmium by gills of the freshwater clam (*Anodonta cygea*) but accelerated cadmium transport from gills towards internal organs (Hemelraad et al. 1987). Cadmium uptake in tissues of *Anodonta* was reduced by about 50% during exposure for 16 weeks to water containing 25 µg Cd/L and 2.5 mg Zn/L (Hemelraad et al. 1987). In a marine prawn (*Pandalus montagui*), cadmium exposure had no effect on tissue zinc levels, but zinc enhanced cadmium uptake in hepatopancreas at the expense of the carcass (Ray et al. 1980). In marine fish, cadmium was taken up more rapidly at elevated seawater zinc levels; however, zinc concentrations in fish tissues decreased with increasing tissue cadmium burdens, suggesting competition between these two metals for the same physiologically active site (Eisler 1981). Zinc concentrations in larval shrimp (*Palaemon serratus*) within its threshold regulation range of 75-525 µg Zn/L were not affected by the addition of 100 µg Cd/L (Devineau and Amiard Triquet 1985). In zebrafish (*Brachydanio rerio*), zinc did not affect cadmium uptake by the whole body or gills but inhibited intestinal uptake and tended to increase gill cadmium elimination rates (Wicklund et al. 1988). Among marine vertebrates, cadmium is selectively accumulated over zinc (Eisler 1984). In ducks, zinc selectively competes with cadmium on high and low molecular weight protein pools in the kidney and liver. Once the high molecular weight protein pool is zinc-saturated excess zinc is stored on metal binding proteins with serious implications for waterfowl stressed simultaneously with cadmium and zinc (Brown et al. 1977). On the other hand, a cadmium-induced disease in bone collagen of chicks was prevented by zinc because of preferential accumulation of zinc (Kaji et al. 1988).

Copper

Mixtures of zinc and copper are generally acknowledged to be more-than-additive in toxicity to a wide variety of aquatic organisms, including oyster larvae (Sprague 1986), marine fish (Eisler and Gardner 1973; Eisler 1984), freshwater fish (Skidmore 1964; Hilmy et al. 1987a) and amphipods (de March 1988), and marine copepods (Sunda et al. 1987; Verriopoulos and Dimas 1988). But zinc-copper mixtures were less-than-additive in toxicity to marine amphipods (*Allorchestes compressa*; Ahsanullah et al. 1988).

Zinc added to the ambient water depressed copper accumulations in tissues of juvenile catfish (*Clarias lazera*), but copper added to the medium depressed zinc uptake Hilmy et al. 1987a). A similar situation was reported in barnacles (*Elminius modestus*); however, simultaneous exposure to copper and zinc resulted in enhanced uptake of both metals (Elliott et al. 1985).

In higher organisms, zinc is a copper antagonist and potentiates the effects of nutritional copper deficiency in rats and chicks. This effect only occurs at extremely high zinc to copper dietary ratios. The addition of copper to the diet of chicks or rats in physiological amounts counteracted all observed signs of zinc intoxication (Tom et al. 1977). No antagonism was evident between dietary copper and zinc fed to channel catfish (*Ictalurus punctatus*) fingerlings; therefore, the high levels of supplemental zinc required in practical feeds should not impair copper status if normal dietary copper levels are present (Gatlin et al. 1989).

High levels of administered zinc limit copper uptake in humans and certain animals (Samman and Roberts 1988) and provides protection against toxicosis produced by copper in pigs and sheep (Allen et al. 1983). Excessive zinc in humans interferes with copper absorption from the intestine, resulting in copper deficiency and eventually in cardiovascular diseases; high zinc intakes also decrease iron bioavailability, leading to a reduction of erythrocyte life span by 67% (Saxena et al. 1989b). Copper deficiency induced by excess dietary zinc is associated with lameness in horses, donkeys, and mules (NAS 1979; Bridges 1990; Ostrowski et al. 1990).

Lead

Lead-zinc mixtures were more-than-additive in toxicity to marine copepods (Verriopoulos and Dimas 1988) and significantly delayed development of mud crab (*Rithropanopeus harrisii*) larvae (EPA 1987). Lead is accumulated up to 10 times more rapidly by marine fish at elevated zinc concentrations in seawater (Eisler 1981).

Among terrestrial animals, zinc protects against lead toxicosis. Dietary zinc reduced the toxic effects of dietary lead to larvae of the house cricket (Migula et al. 1989). Zinc at 100-200 µg/egg (1 mg Zn/kg egg) significantly protected developing white leghorn chicks against lead-induced 50 µg/egg) deformities and death

when injected into the yolk sac on day 7 of incubation (Anwer et al. 1988). Zinc also protects against lead toxicity in horses (Anwer et al. 1988) and against testicular injury induced by lead in rats (Saxena et al. 1989a).

Nickel

Nickel-zinc mixtures were additive in toxicity to marine copepods (Verriopoulos and Dimas 1988) and to the three-spined stickleback (Skidmore 1964).

Oral nickel toxicity in chicks was prevented by increased dietary zinc (Warner et al. 1988). Nickel is a leading cause of allergic contact dermatitis in many industrial nations; about 6% of the general public and about 11% of dermatology clinic patients are sensitive to nickel (Warner et al. 1988). Zinc prevents nickel sulfate-induced allergic contact dermatitis in guinea pigs (*Cavia* spp.) through addition of 100-200 mg Zn/L drinking water for 4 weeks before nickel insult (Warner et al. 1988). Nickel and other metals that cause allergic contact dermatitis penetrate the skin, complex with selected ligands, and stimulate a delayed hypersensitivity. Zinc is thought to block the sites where nickel complexes to the protein (Warner et al. 1988).

Other Chemicals

Zinc interacts with a wide variety of inorganic, organic, and biological agents, but in most cases the available information is fragmentary and the mechanisms of action are unknown. Mice pretreated with zinc at 6.5 mg Zn/kg BW for 9 days showed increased resistance to arsenic toxicosis during a 30-day observation period (Kreppel et al. 1988). Oral zinc therapy was effective in treating biological agents such as infectious pododermatitis in cattle; ovine foot rot in sheep; sporidesmin in sheep, cattle, and rodents; and the toxins of the fungus *Phomopsis leptostromiformis* in sheep (Allen et al. 1983). Calcium modifies zinc toxicity to freshwater aquatic organisms, and increased calcium is associated with decreased acute toxicity (Everall et al. 1989b; Handy et al. 1989). Zinc absorption in the rat gut is decreased after ingestion of phosphorus as polyphosphate or as orthophosphate and high levels of calcium (Greger 1989). Zinc cytotoxicity is blocked by increased calcium or iron but not by magnesium (Borovansky and Riley 1989). Zinc reportedly protects rats against carbon tetrachloride poisoning (Allen et al. 1983).

Various cheating agents, including disodium ethylene diamine tetraacetic acid (EDTA), disodium calcium cyclohexanediamine tetraacetate, D-penicillamine, 2,3-dimercapto-l-propane sulfonic acid, and 2,3-dimercaptosuccinic acid protect mice against zinc acetate poisoning (Llobet et al. 1988b). Zinc protects toad embryos against agents known to produce malformations, including excess Vitamin A, acetazolamide, calcium-EDTA, and acetaminophen (Herkovits et al. 1989). Venom of the jararaca (*Bothrops jararaca*), a venomous Brazilian serpent, contains a zinc metalloprotease called J protease; the proteolytic activity of J protease is inactivated by EDTA and other sequestering agents (Tanizaki et al. 1989).

Chromium-zinc mixtures were more than additive in toxicity to *Tisbe holothuriae*, a marine copepod. Zinc in combination with chromium was more toxic to copepods than mixtures of zinc with copper, lead, nickel, or cadmium (Verriopoulos and Dimas 1988).

Renal tubular absorption of zinc in mice was impaired by certain diuretics and was further influenced by dietary proteins (Goyer 1986).

Zinc absorption in rats was depressed after consumption of high levels of inorganic iron; absorption was normal with organoirons (Greger 1989).

Mercury-zinc mixtures were more-than-additive in toxicity to oyster larvae (Sprague 1986). Preexposure of common mussels (*Mytilus edulis*) to 50 µg Zn/L for 28 days conferred increased tolerance to 75 µg Hg/L (Roesijadi and Fellingham 1987). Zinc inhibited the accumulation of mercury in marine snails and crustaceans (Andersen et al. 1989).

Zinc deficiency places an increased demand on selenium pools in daphnids. As little as 5 µg Se/L in zinc-free water eliminated overt cuticle damage and substantially increased reproduction but did not alter the shortened life span. Cladocerans at the threshold of selenium deficiency become overly selenium-deficient when zinc supplies are lacking (Keating and Caffrey 1989). Insufficient copper introduces cuticle problems in daphnids similar to those introduced by insufficient zinc or selenium, increasing the likelihood of a proposed

relation between glutathione peroxidase (which contains selenium) and copper-zinc superoxide dismutase (Keating and Caffrey 1989).

High levels of dietary tin increased zinc loss from rats (Greger 1989). Zinc prevented toxic effects of vanadium (10 mg/kg BW) on bone metabolism of weanling rats (Yamaguchi et al. 1989a).

Carcinogenicity, Mutagenicity, Teratogenicity

General

When injected directly into the testes, zinc can induce testicular sarcomas in birds and rats but has not been shown to be tumorogenic by any other route. Zinc promotes tumor growth after conditions of zinc deficiency but excess zinc may suppress or inhibit tumor proliferation, although the mechanisms of the action are imperfectly understood. Chromosomal aberrations were observed under conditions of zinc deficiency, but excess zinc was not mutagenic in most tests. Organozinc compounds are effective mutagens when presented to susceptible cell populations in an appropriate form, but the evidence for inorganic zinc is incomplete. Zinc is teratogenic to frog and fish embryos, but conclusive evidence of teratogenicity in mammals is lacking. Zinc may protect against the effects of some mammalian teratogens. Under conditions of mild zinc deficiency, however, diabetes and effects of various teratogens are exacerbated.

Carcinogenicity

Carbamate esters of zinc, zineb, and ziram are carcinogenic and teratogenic in animals, which is, however, attributed to the action of the carbamate esters and not to zinc (Elinder 1986). Results of studies with small mammals showed zinc to be cocarcinogenic with 4-nitroquinoline-N-oxide on oral cancer and with N-ethyl-N-nitrosourea on brain cancer (Leonard and Gerber 1989).

There is conclusive evidence that repeated intratesticular injections of zinc salts can induce testicular sarcomas in birds and rats (NAS 1979; Elinder 1986; Goyer 1986; PHS 1989). Testicular teratomas in roosters were first produced experimentally in 1926 when zinc salts were injected into the testes as a method of practical castration; tumors could be induced only by intratesticular injection during the spring period of gonadal growth (Guthrie 1971). Teratomas of the testes were observed in fowl given testicular injections of 2 mL of 10% $ZnSO_4$ solution (PHS 1989). Teratomas were induced in Japanese quails (*Coturnix coturnix japonica*) by intratesticular injections of 3% zinc chloride solutions during a period of testicular growth stimulated by increased photoperiod; tumors were similar to those of domestic fowl and have histological features in common with spontaneous testicular teratomas in humans (Guthrie 1971). Testicular tumors in rats were produced by direct intratesticular injection of zinc; no other carcinogenic effects were produced by any other route regardless of dose (Goyer 1986). It is emphasized that zinc and zinc compounds are not conclusively carcinogenic except when injected directly into the testes; no field or experimental evidence exists showing zinc to be tumorogenic through any other route (NAS 1979; Phillips and Kindred 1980; Elinder 1986; Leonard and Gerber 1989; PHS 1989).

Zinc is essential for the growth of rapidly proliferating cells such as tumors. The high zinc requirements of these cells in tumor disease can result in latent zinc deficiency. Accordingly, growth of animal tumors is stimulated by zinc and retarded by zinc deficiency (Prasad 1979; Leonard and Gerber 1989). In mouse fibrosarcoma cells, zinc inhibits endonucleases, subsequently blocking DNA fragmentation and tumor cell lysis, allowing tumors to grow (Flieger et al. 1989). There is no evidence that zinc deficiency causes cancer (NAS 1979), although deficiency was associated with decreasing tumor growth (Prasad 1979; Phillips and Kindred 1980). Malignant human tissues, for example, frequently contained less zinc than normal tissue, that is, 78 mg/kg FW in a normal liver versus 18 mg/kg FW in a cancerous liver (Phillips and Kindred 1980).

Zinc can also inhibit tumor growth (NAS 1979), although the mechanisms of zinc suppression of carcinomas are imperfectly understood (Phillips and Kindred 1980). Zinc inhibits the growth of mouse melanoma cells at concentrations between 8.2 and 9.9 mg Zn/L culture medium (Borovansky and Riley 1989). The addition of 100 mg $ZnSO_4$/L to drinking water of hamsters inhibited formation of dimethylbenzanthracene-induced carcinomas (Phillips and Kindred 1980). High zinc diets of 500 mg/kg ration reduced growth of a chemically induced hepatoma in rats (Phillips and Kindred 1980). Intramuscular injections of zinc oxide or zinc acetate administered together with nickel sulfide--a potent muscle carcinogen--delayed but did not prevent 100% tumor incidence in rats during a 66-week observation period (Kasprzak et al. 1988). Administration of zinc slows the carcinogenic

process induced by nickel from the production of water-soluble and water-insoluble zinc compounds, despite markedly different retention times in muscle of zinc compounds (Tb½ ZnO = 24 days, zinc acetate = 2.5 days, Ni_3S_2 = 21 days). Zinc in either form exerted no measurable influence on nickel retention at the injection site or early local cellular reactions to nickel (Kasprzak et al. 1988). Testicular tumors in rats caused by injection of cadmium were suppressed by zinc injection (Leonard and Gerber 1989) when the zinc to cadmium molar ratio was about 100:1 (Phillips and Kindred 1980). Inhibition of cadmium carcinogenesis by zinc is a complex phenomenon, depending on dose, route, and target site (Waalkes et al. 1989). For example, the number of cadmium-induced testicular tumors in rats was reduced by 50% during a 2-year period after three subcutaneous injections of 65.4 mg Zn/kg BW given within 18 h of the initial cadmium insult, although unlike controls, this group had a marked elevation in prostatic tumors; tumor number was reduced by 92% when rats were given 100 mg Zn/L in drinking water (Waalkes et al. 1989).

Mutagenicity

Results of mutagenicity studies with whole organisms were usually negative because homeostatic controls of absorption and protein binding preclude the likelihood of zinc being genotoxic under standard feeding conditions (Thompson et al. 1989). However, zinc is an effective mutagen and clastogen when presented to a susceptible cell population in an appropriate form (Thompson et al. 1989). Zinc acetate produced dose-related positive responses in the mouse lymphoma assay and also in a cytogenetic assay with Chinese hamster ovary cells; however, results of mutagenicity assays with inorganic zinc were negative in the *Salmonella* mutation assay and in unscheduled DNA synthesis on primary cultures of rat hepatocytes (Thompson et al. 1989). Organozinc compounds have mutagenic potential, as judged by the positive responses with zinc 2,4-pentanedione and *Salmonella* (Thompson et al. 1989).

Structural chromosome aberrations, particularly chromatid gaps and increased frequency of fragment exchange, were observed in rat bone marrow cells after 14 days of exposure to 240 mg Zn/L drinking water (Kowalska-Wochna et al. 1988). Chromosomal aberrations were observed in bone marrow cells of mice fed diets equivalent to 650 mg Zn/kg BW daily in mice exposed to zinc oxide by inhalation, and in mice maintained on a low calcium diet (PHS 1989). Aberrations in bone marrow of mice given 5,000 mg Zn/kg diet may be associated with calcium deficiency (Leonard and Gerber 1989). Calcium is displaced by zinc in calcium-depleted conditions, leading to chromosomal breaks and interference in the repair process (PHS 1989).

Zinc chloride induces chromosomal aberrations in human lymphocytes in vitro (Elinder 1986). A higher incidence of chromosome anomalies in leukocytes occurs among workers exposed to zinc (Elinder 1986), but these aberrations are probably due to other (unspecified) mutagenic factors in the work environment (Leonard and Gerber 1989).

Zinc inhibits the mutagenic action of some carcinogens because it is a constituent of mutagen detoxifying enzymes or because it acts directly on the microsomal monooxygenases forming the ultimate carcinogen (Leonard and Gerber 1989). Zinc significantly reduced a genotoxic effect of lead in rat bone marrow cells (500 mg Pb/L drinking water followed by 240 mg Zn/L for 2 weeks) and also protected against lead accumulations in erythrocytes and lead-induced inhibition of delta-amino levulinic acid dehydratase (Kowalska-Wochna et al. 1988). Zinc deficiency can lead to chromosomal aberrations, but excess zinc was not mutagenic in the majority of tests for DNA damage--except for zinc-containing fungicides wherein the organic dithiocarbamate constituents were the mutagenic agents and for zinc chromate wherein the chromate ion was the active agent (Leonard and Gerber 1989). Frequencies of sister chromatid exchanges in calves with hereditary zinc deficiency, also known as Lethal Trait A46, are lower than in healthy normal cows, suggesting a fundamental association between disturbed zinc metabolism and the low incidence of sister chromatid exchanges in A46 cattle (Bosma et al. 1988).

Teratogenicity

Excess zinc is teratogenic to frog and fish embryos, possibly by inhibition of DNA synthesis (Dawson et al. 1988; Fort et al. 1989). Zinc at 150 mg/kg in rat diets was associated with inhibited fetal implantation but this needs confirmation (Elinder 1986). No conclusive evidence now exists demonstrating that excessive zinc produces any teratogenic effect in mammals (NAS 1979; Dawson et al. 1988; Leonard and Gerber 1989).

Excess zinc may protect against some teratogens, such as calcium EDTA (Leonard and Gerber 1989). Also, teratogenic effects of cadmium salts in golden hamsters was reduced by simultaneous administration of zinc salts (NAS 1979).

Zinc deficiency is clearly teratogenic in mammals (Dawson et al. 1988; Leonard and Gerber 1989). Severe maternal zinc deficiency is known to be teratogenic in rats. Fetal malformations--especially calcification defects--from maternal zinc deficiency affect almost every tissue (Ferreira et al. 1989). Skeletal malformations are most common, possibly because of a reduction in cellular proliferation and in activity of bone alkaline phosphatase (Leonard and Gerber 1989). Human zinc deficiency may act teratogenically, either directly or indirectly through other toxic agents (Jameson 1980). Zinc deficiency may exacerbate effects of several teratogenic agents such as thalidomide; there is also the possibility that zinc deficiency may increase the incidence of spina bifida and anencephaly, but this needs verification (Leonard and Gerber 1989). Diabetes during pregnancy can amplify the effects of a mild maternal zinc deficiency. In one study, diabetic and nondiabetic rat strains were fed a low zinc diet (4.5 mg Zn/kg diet), an adequate zinc diet (24.5 mg/kg), or a high zinc diet (500 mg/kg) throughout gestation. Fetuses from diabetic dams were smaller, weighed less, and had less calcified skeletons and more malformations than fetuses from control dams. In controls, maternal dietary zinc had a minor effect on fetal malformation frequency. In diabetic strains, however, the low zinc diet had a strong teratogenic effect (Uriu-Hare et al. 1989).

Background Concentrations

General

Total zinc concentrations in nonbiological samples seldom exceed 40 µg/L in water, 200 mg/kg in soils and sediments, or 0.5 µg/m^3 in air. Environments heavily contaminated by anthropogenic activities may contain up to 99 mg Zn/L in water 118 g/kg in sediments, 5 g/kg in soil, and 0.84 µg/m^3 in the atmosphere. Zinc was detectable in all samples of plants and animals measured. Grossly-elevated (i.e., >4 g/kg DW) concentrations were normally encountered in selected tissues of marine bivalve molluscs, barnacles, and polychaete annelids. In general, zinc concentrations were elevated in organisms collected near anthropogenic point sources of zinc contamination but were modified substantially by the organism's diet, age, reproductive state, and zinc-specific sites of accumulation as well as by inherent interspecies differences.

Nonbiological

Zinc concentrations in freshwater, seawater, groundwater, sewage sludge, sediments, and soils are listed in Table 3. These data are considered reliable, although newer clean laboratory techniques suggest that dissolved zinc concentrations in nonpolluted rivers may be 10 to 100 times lower than previously reported (Shiller and Boyle 1985).

Zinc concentrations in water seldom exceed 40 µg/L except near mining, electroplating and similar activities--where concentrations between 260 and 954 µg/L were frequently recorded. Drinking water usually contains <10 µg Zn/L, although concentrations >2 mg/L may occur after passage through galvanized pipes (Goyer 1986). Zinc-contaminated streams in the Platte River Basin sometimes contain up to 99 mg Zn/L and in Arkansas up to 79 mg/L (Mirenda 1986). Zinc concentrations in water downstream of placer mining activities in Alaska sometimes exceed the concentrations that are toxic to the Arctic grayling, *Thymallus arcticus* (Buhl and Hamilton 1990). The disappearance of the stone loach (*Noemacheilus barbatulus*) in the United Kingdom from streams receiving industrial wastes was attributed directly to zinc concentrations in the stream rising from 1 mg/L to a lethal 5 mg/L (Solbe and Flook 1975).

Table 3. Zinc concentrations (milligrams of zinc per kilogram fresh weight [FW] or dry weight [DW] in representative nonbiological materials.

Material	Concentration[a] (mg/kg or mg/L)	Reference[b]
Earth's crust	40 DW	11
Freshwater		
Canada		
Normal	<0.04 FW	1
Acidic mine tailings	0.9 FW, Max. 3.3 FW	2
wastes, Sudbury, Ontario		
United States		
Alaska		
Contaminated streams	0.029-0.882 FW	3
Downstream of placer	0.125 (0.075-0.165) FW	3
mining activities		
Nationwide	0.0005-0.010 FW	4
Worldwide, rivers	0.021 FW	2,5
Groundwater, near Lake Erie	Max 0.954 FW	1
Seawater		
Australia (polluted)	0.134 FW	6
Canada	0.01-0.04 FW	1
Irish Sea		
Coastal	0.007 FW	6
Near shore	0.003 FW	6
Offshore	0.003 FW	6
Open ocean		
Deep water	0.0006 FW	6
Surface	0.000002-0.0001 FW	4
United Kingdom		
Clyde estuary	0.006 FW	7
Heavily polluted	0.026 FW	6
Polluted	0.007-0.012 FW	6
Severn estuary	0.022 FW	7
United States, San Diego		
Coastal	0.0005 FW	6
Harbor	0.0026 FW	6
Western Mediterranean		
Coastal	0.0015-0.002 FW	6
Estuary	Max 0.010 FW	6
Near Shore	0.0036 FW	6

Sediments		
Australia	35 DW; Max. 280	8
Canada		
Lakes	55-160 DW	1
Marine	64-180 DW	1
Streams and rivers	50-138 DW	1
Mediterranean	5-20 DW	S
Sweden and Norway	Usually <130 DW; Max. 118,000	8
United Kingdom	70-245 DW; Max. 825 DW	8
United States		
Corpus Christi, Texas		
Bay	10-229 DW	9
Harbor	229-11,000 DW	9
New York Bight		
Uncontaminated site	18 DW	9
Sewage dump site	252 (54-416) DW	9
Northeast	15-20 DW; Max. 1,500 DW	8
Puget Sound	65 DW; Max. 185 DW	8
Rhode Island, near electroplaters		
Narragansett Bay	110 (53-168) DW	9
Providence River	490 DW	9
Southern California Bight	55-75 DW; Max. 2,800	8
Sewage Sludge		
United Kingdom, Glasgow	1,125 DW	7
United States		
Average	1,409 DW	10
Missouri	1,200 (170-13,000) DW	10
Soils		
United States	54 (<25-2,000) DW	10
Uncontaminated	10-300 DW	11
Near smelters	5,000 DW	11

[a]Concentrations are shown as means, range (in parentheses), and maximum (Max.).

[b] 1. Spear 1981; 2. Mann et al. 1989; 3. Buhl and Hamilton 1990; 4. EPA 1987; 5. Mann and Fyfe 1988; 6. Sprague 1986; 7. Nugegoda and Rainbow 1988b; 8. Young et al. 1980; 9. Eisler et al. 1977; 10. Beyer 1990; 11. Elinder 1986.

Concentrations of zinc in sediments and soils usually do not exceed 200 mg/kg but can range between 3 and 118 g/kg as a result of human activities (Table 3). Atmospheric zinc levels were almost always <1 µg/m^3, although they tended to be higher over industrialized areas (Goyer 1986). Average zinc concentrations were <0.001 µg/m^3 atmosphere at the South Pole, 0.01-0.02 µg,/m^3 atmosphere in rural areas of the United States, <0.01-0.84 µg/m^3 atmosphere in U.S. cities, and 0.06-0.35 µg/m^3 atmosphere at various locations in the United Kingdom (Elinder 1986).

Biological

Zinc measurements in field collections of plants and animals (Table 4) show several trends. (1) Zinc is present in all tissues of all organisms measured. (2) Concentrations are elevated in organisms near anthropogenic point sources of zinc contamination. (3) Concentrations are normally grossly elevated (>4 g/kg FW soft parts) in bivalve molluscs and barnacles. (4) Zinc-specific sites of accumulation include the frond in algae; the kidney in molluscs; the hepatopancreas in crustaceans; the jaws in polychaete annelids; the viscera, gonad, and brain in fish; the liver, kidney, and bone in birds; and the serum, pancreas, feces, liver, kidney, and bone in mammals. (5) Interspecies variations in zinc content are considerable, even among taxonomically closely-related species. (6) Intraspecies differences in zinc content vary with age, size, sex, season, and other modifiers. (7) Many species regulate zinc within a threshold range of concentrations.

Additional information on background concentrations of zinc is given in Vallee (1959), NAS (1979), Young et al. (1980), and Eisler (1980, 1981).

Terrestrial Plants and Invertebrates

Zinc concentrations in forest plants vary considerably. Some species of oaks (*Quercus* spp.), for example, are accumulators whereas others may be termed discriminators. In descending order of concentration zinc is in the roots, foliage, branches, and trunk of individual species (Van Hook et al. 1980). Small lateral roots accumulate zinc to much greater levels than other vegetation components and are probably most sensitive to changes in zinc inputs. Half-time persistence of zinc in forest ecosystems varies from about 3 years in organic matter components to >200 years in large soil pools (Van Hook et al. 1980).

Table 4. Effects of zinc on representative terrestrial plants and invertebrates.

Taxonomic group, organism, and other variables	Concentration[a] (mg/kg)	Reference[b]
Aquatic plants		
Euglena sp., from acidic mine tailings waste discharges (0.9 mg Zn/L, Max. 3.3 mg/L)	143 DW; Max. 410 DW	1
Aquatic moss, *Fontinalis squamosa*		
Contaminated river, Wales, 1985	Max. 2,810 DW	2
Uncontaminated site	<400 DW	2
Marine plants		
Phytoplankton	38 DW	3
Seaweeds	90 DW	3
Eelgrass, *Zostera marina*		
Leaf	Max. 195 DW	4
Rhizome	Max. 70 DW	4
Root	Max. 155 DW	4
Stem	Max. 85 DW	4
Terrestrial plants and invertebrates		
Honey bee, *Apis mellifera*, Czechoslovakia, 1986-87		
Drones	77-89 DW	5

Honey	0.6-4.5 DW	5
Pollen in combs	39-55 DW	5
Wax	11-249 DW	5
Workers, whole		
Foragers, spring	116-204 DW	5
Dead overwintering	8-13 DW	5
Young	83-160 DW	5
Grey field slug, *Deraceras reticulatum*, near lead-zinc mine		
Digestive gland	3,968 DW	6
Foot-head	308 DW	6
Gonads	118 DW	6
Intestine	380 DW	6
Whole	800 DW	7
Earthworms, north-eastern United States, whole		
From uncontaminated soils (23-200 mg Zn/kg DW), 6 species	120-650 DW	8
From mining sites (100-2,500 mg Zn/kg DW), 5 species	200-950 DW	8
From industrial sites (24-320 mg Zn/kg DW soil), 6 species	320-1,600 DW	8
Near galvanized towers (28-270 mg Zn/kg DW soil), 1 species	340-690 DW	8
Earthworms, whole, gut empty		
Dendrodrilus rubidus	(308-1,683) DW	9
Lumbricus rubellus	(394-3,873) DW	9
Gastropods, whole, near abandoned mine, soil contained 1,377 mg Zn/kg DW		
Arion ater	900 DW	7
Arion hortensis	600 DW	7
Arion subfuscus	1,200 DW	7
Derocerus caruanae	1,000 DW	7
Lichen, *Lasallia papulosa*		
Near zinc smelter	2,560 DW	10

Control population	214 DW	10
Isopod, *Oniscus asellus*, whole, from soil containing various concentrations of zinc (mg Zn/kg soil DW)		
<0.3Max. 150 DW		11
1-10	Max. 350 DW	11
>50	Max. >500 DW	11
Plants, terrestrial	Average 100 DW	114
Woodlouse, *Porcellio scaber*		
Near metal smelter of maximum soil zinc of 24,900 mg/kg DW, and soil litter of 4,150 mg/kg DW		
Hepatopancreas	Max. 13,500 DW	12
Whole	Max. 1,500 DW	12
From soil containing various concentrations of zinc (mg Zn/kg soil DW), whole organism		
<0.3	Max. 350 DW	11
1-10	Max. 550 DW	11
>50	Max. >1,000 DW	11
Protozoans, marine	63-279 DW	13
Coelenterates		
Soft coral, *Alcyonia alcyonium*, whole	9.6 FW	14
Plumose anemone, *Metridium senile*, whole	18 FW	14
Various species, whole		
Uncontaminated areas	50 DW	3
Noncontaminated areas	<80 FW; <120 DW	13
Contaminated areas	Max. 603 DW	13
Molluscs, aquatic		
Abalones, soft parts	55 (38-100) DW	17
Bivalves		
Kidney granules	10,000-43,320 DW	15
Soft parts	91-660 DW	16
Cephalopods		

Soft parts	81-150 DW; Max. 580 DW	16,17
Whole	250 DW	3
Chitons, soft parts	290-700 DW	17
Clams, soft parts	81-115 DW; Max. 510 DW	17
Sydney rock oyster, *Crassostrea commercialis*, soft parts, Southeast Asia	800 (64-1,920) DW	18
American oyster, *Crassostrea virginica*, soft parts		
Chesapeake Bay	3,975 (60-12,800) DW	18
Gulf of Mexico	2,150 (485-10,000) DW	18
South Carolina	2,410 (280-6,305) DW	18
United States	1,018-1,641 (204-4,000) FW	19
Drills, soft parts	536-3,470 DW	17
Gastropods, soft parts	84-763 DW	16
Limpets, soft parts		
18 species	112 (14-760) DW	17
7 species	196 (86-430) DW	17
Clam, *Macoma balthica*, adults, San Francisco Bay, soft parts	200-600 DW	20
Mussels, soft parts	109-267 DW; Max. 7,700 DW	17
Common mussel, *Mytilus edulis*		
Soft parts, 0.43 g DW		
Visceral mass	34-100 DW	21
Gills and palps	47-94 DW	21
Remainder	48-110 DW	21
Soft parts, 0.22 g DW		
Visceral mass	28-112 DW	21
Gills and palps	38-158 DW	21
Remainder	40-130 DW	21
Kidney, Newfoundland		
October 1984	144 (50-427) DW	22
April 1985	828 (94-3,410) DW	22
Oyster drill, *Ocenebra erinacea*, soft parts	1,451-2,169 DW	23
European flat oyster, *Ostrea edulis*, soft parts		
Contaminated site	10,560 (4,700-12,640) DW	24
Clean site	98 DW	24
Oysters		
Sort parts	1,960-7,270 DW; Max. 49,000 DW	17
Soft parts	100-271 FW	19

Scallop, *Pecten* sp.		
Kidney	32,000 DW	17
Kidney granules	120,000 DW	17
Soft parts	200 DW	17
Scallops, soft parts	105-212 DW; Max. 462 DW	
Green-lipped mussel,		
Perna viridis, Hong Kong		
Soft parts, 1986-87	56-134 DW	25
Soft parts, 1986		
March	63-150 DW	26
May	77-94 DW	26
Clam, *Pitar morrhuana*, soft parts, near electroplating plant, Rhode Island, 1973	Max. 276 DW	27
Rock oyster, *Saccostrea cucullata*, soft parts, Hong Kong, 1986		
March	2,082-3,275 DW	26
May	2,210-2,863 DW	26
Whelks, soft parts	198 (13-650) DW	17
Crustaceans		
Amphipods, marine, whole, western British coastal waters		
Orchestia gammarellus	104-392 DW	28
Orchestia mediterranea	120-506 DW	28
Talitrus saltator	178-306 DW	28
Talorchestia deshayesii	199-208 DW	28
Amphipods, *Themisto* spp., whole	76 (72-81) DW	29
Barnacle, *Balanus amphitrite*, soft parts	Max. 1,937 DW	30
Barnacle, *Balanus balanoides*, soft parts	1,028-3,438 FW	31
Crustaceans, marine		
Northeast Atlantic ocean, July 1985, whole		
Decapods	35-57 DW	32
Euphausids	44-96 DW	32
Mysids	24-44 DW	32
Soft parts		
Amphipods	73-109 DW	16
Barnacles	690-27,837 DW	16
Barnacles	1,050-5,140 DW; Max. 113,000 DW	17

Copepods	60-170 DW	16
Copepods	164-177 DW; Max. 1,300 DW	17
Crabs	68-102 DW; Max. 340 DW	17
Euphausids	53-83 DW	16
Isopods	94 DW	16
Shrimps	14-69 DW; Max. 150 DW	17
Various species		
Blood	0.2-87 FW	19
Excretory organs	Max. 29 FW	19
External eggs	24-107 FW	19
Gills	8-69 FW	19
Hepatopancreas	34-169 FW	19
Muscle		
Leg	15-68 FW	19
Abdominal	10-24 FW	19
Shell	5-17 FW	19
Stomach fluid	1-92 FW	19
Ovary	26-82 FW	19
Vas deferens	13-30 FW	19
Urine	Max. 2.2 FW	19
Whole	18-54 FW	19
Hermit crab, *Eupagurus bernhardus*, whole	282 FW	19
Euphausid, *Euphausia superba*, whole	68 (42-75) DW	29
Euphausid, *Meganyctiphanes norvegica*, whole		
Firth of Clyde	43 (27-62) DW	29
Northeast Atlantic Ocean	102 (40-281) DW	29
Euphausids, whole	13 FW	33
American lobster, *Homarus americanus*		
Gill	102-126 DW	34
Green gland	114-148 DW	34
Hepatopancreas	70-135 DW	34
Muscle		
Pincer	100-127 DW	34
Tail	80 DW	34
Crayfish, *Orconectes virilis*, collected 12-150 km from metal smelter		
Hepatopancreas		
12 km	190 DW	35
30 km	166 DW	35
150 km	92 DW	35
Digestive tract		
12 km	154 DW	35

30 km	100 DW	35
150 km	111 DW	35
Muscle		
12 km	93 DW	35
30 km	97 DW	35
150 km	80 DW	35
Grass shrimp, *Palaemonetes pugio*		
From sediments containing 627 mg Zn/kg DW		
Exoskeleton	58 FW	36
Muscle	55 FW	36
From sediments containing 8 mg Zn/kg DW		
Exoskeleton	18 FW	36
Muscle	30 FW	36
Prawn, *Pandalus montagui*		
Cuticle	57 DW	37
Eye	70 DW	37
Gill	106 DW	37
Hepatopancreas	30 DW	37
Muscle	57 DW	37
Whole	58 DW	37
Pink shrimp, *Penaeus brasiliensis*, adults, whole	(47-75) DW; (181-290) FW	38
Insects, marine, whole	110-197 DW	13
Chaetognaths, whole	76-90 DW	13
Annelids, aquatic		
Annelids, marine		
Jaws		
Total	5,000-24,000 DW	13
Basal section	1,790 DW	13
Distal section	34,950 DW	13
Whole body	22-1,564 DW	13
Lugworm, *Arenicola*, *marina*, whole	1.8 FW	14
Freshwater leech, *Erpobdella octoculata*, adults, whole body	Upstream (18 µg Zn/L) from zinc-polluted mine waste discharge, whole body content of 1,439-1,559 DW; reproduction normal. Downstream (180 µg Zn/L), concentration after 19-month exposure was 1,932-2,432 DW; reproduction impaired	116
Sandworm, *Nereis diversicolor*		

Head	843-995 DW	39
Parapodia	216-418 DW	39
Trunk	158-218 DW	39
Echinoderms, various species, **whole**	Usually 100 DW or lower, frequently >100 DW; Max. 245 FW, 1,500 DW	3,13
Tunicates, whole	200 DW; Max. 64 FW, 370 DW	3, 13
Fish		
Catostomids, 3 species, Missouri, blood		
Site contaminated with mine tailings	10.9-13.4 FW, 94-119 DW	40
Uncontaminated site	8.7-11.2 FW, 76-86 DW	40
White sucker, *Catostomus commersoni*		
From metals-contaminated lake (400 µg Zn/L)		
Eggs	83-158 DW	41
Larvae	511 DW	41
Ovaries		
Prespawning	114 DW	41
Postspawning	290 DW	41
Testes, postspawning	89 DW	41
From control lake (2.7 µg Zn/L)		
Eggs	69-108 DW	41
Larvae	163 DW	41
Ovaries		
Prespawning	84 DW	41
Postspawning	317 DW	41
Testes, postspawning	163 DW	41
New Brunswick, whole	92-93 DW	42
Nova Scotia, whole	98-122 DW	42
African sharptooth catfish, *Clarias gariepinus*, age 4-8 years, South Africa, 1988-89, lake sediments contained 1,104 mg Zn/kg DW (595-2,189)		
Brain	335 DW	43
Fat	5O DW	43
Gill	177 DW	43
Gonad	126 DW	43
Heart	196 DW	43
Intestine	143 DW	43
Kidney	143 DW	43
Liver	143 DW	43
Muscle	59 DW	43
Spleen	163 DW	43

Vertebrae	75 DW	43
Baltic herring, *Clupea harengus*, liver	23 FW	14
Freshwater fish, various species		
Great Lakes		
Whole, less intestines, 4 species	12-20 FW	19
Liver, 10 species	11-48 FW	19
Greece, 1987-88, muscle, 11 species	7 (3-37) FW	44
United States, nationwide, whole		
1978-79	25 (8-168) FW	45
1980-81	24 (9-109) FW	45
1984		
Geometric mean	21.7 FW	121
85th percentile	34.2 FW	121
Maximum	118.4 FW	121
From metals-contaminated (636 µg dissolved Zn/L) lake, Indiana, whole		
Bowfin, *Amia calva*	93 DW	46
White sucker, *Catostomus commersoni*	102 DW; Max. 152 DW	46
Brown bullhead, *Ictalurus nebulosus*	127 DW; Max. 139 DW	46
Warmouth, *Lepomis gulosus*	140 DW; Max. 166 DW	46
Orangespot sunfish, *Lepomis humilis*	248 DW	46
Redear sunfish, *Lepomis microlophus*	477 DW; Max. 820 DW	46
Largemouth bass, *Micropterus salmoides*	119 DW; Max. 207 DW	46
Golden shiner, *Notemigonus crysoleucas*	160 DW; Max. 171 DW	46
Yellow perch, *Perca flavescens*	160 DW; Max. 171 DW	46
Black crappie, *Pomoxis nigromaculatus*	123 DW	46
From metals-contaminated stream, Missouri, muscle, 5 species	3.1-24 FW	47,115
Shortfin mako, *Isurus oxyrinchus*, vertebrae	36 (5-127) DW	48
Marine fish, various species		
Muscle		
54 species	0-5 FW	19
32 species	5.1-10 FW	19
7 species	10.1-15 FW	19
4 species	15.1-20 FW	19

2 species	20.1-25 FW	19
Whole	80 DW	3
Red Sea, 1980-82		
Triggerfish, *Balistoides viridiscens*		
Muscle	66 DW	49
Liver	154 (81-227) DW	49
Ovaries	291 (287-792) DW	49
Surgeonfish, *Ctenochaetus strigosus*, muscle	29 (11-43) DW	49
Halfbeak, *Hemiramphus marginatus*, muscle	32 DW	49
Labrids, 3 species, muscle	33 (19-51) DW	49
Lethrinids *Lethrinus* spp.		
Muscle	33 (13-112) DW	49
Liver	95 (43-146) DW	49
Ovaries	146 (72-259) DW	49
Testes	152 (141-164) DW	49
Snapper, *Lutianus fulviflamma*, muscle	48 (25-70) DW	49
Parrotfish, *Scarys gyttatus*		
Liver	17 DW	49
Muscle	62 DW	49
Serranids, 4 species		
Muscle	51 (8-112) DW	49
Liver	130 (78-183) DW	49
Rabbitfish, *Siganus oramin*		
Muscle	55 (18-195) DW	49
Liver	179 (68-611) DW	49
Sparids, 2 species, muscle	56 (34-76) DW	49
Goatfish, *Upeneus tragula*, muscle	51 (37-68) DW	49
Pacific hake, Merluccius productus		
Muscle	4 (3-6) FW	33
Whole	12 FW	33
Catfish, *Mystus gulio*, juveniles, whole, India		
From contaminated estuary (100-120 µg Zn/L, 120-145 mg Zn/kg sediment DW)	160-180 DW	50
From uncontaminated estuary (10 µg Zn/L, 30 mg Zn/kg sediment)	15 DW	50
Yellow perch, *Perca flavescens*, whole		

New Brunswick	81-103 DW	42
Nova Scotia	68-85 DW	42
Blue shark, *Prionace glauca*, vertebrae	95 (32-210) DW	45
Atlantic salmon, *Salmo salar*		
Eggs		
Hatchery	20-35 FW	51
Native	19-28 FW	51
Liver, juveniles		
Hatchery	29-41 FW	51
Native	34 FW	51
Muscle	13 DW	52
Ovaries	166 DW	52
Spines	79-219 DW	52
Stomach contents	78 DW	52
Brook trout, *Salvelinus fontinalis*, whole		
New Brunswick	87-158 DW	42
Nova Scotia	90-110	42
Atlantic mackerel, *Scomber scombrus*, liver	31 FW	14
King mackerel, *Scomberomorus cavalla*, otolith		
Age <1 year	16 DW; Max. 50 DW	53
Age 2 years	11 DW	53
Age 10 years	8 DW	53
Lesser spotted dogfish, *Scyliorhinus caniculus*, liver	8.7 FW	14
Monkfish, *Squatina squatina*, liver	8 FW	14
Reptiles		
American alligator, *Alligator mississippiensis*, eggs (less shell), Florida, 1984	4.9-9.2 FW	54
Birds		
Blue-winged teal, *Arias discors*, Texas, 1983		
Muscle		
Males	13.8 FW	55
Females	11.3 FW	55
Liver		
Autumn	41.4 FW	55
Spring	33.7 FW	55
Mallard, *Anas platyrhynchos*, liver	54 FW	56
Canvasback, *Aythya valisineria*, Chesapeake Bay liver	41 FW	56

Nicobar pigeon, *Caloenas nicobarica*, zinc-poisoned		
Kidney	2,107 DW	57
Liver	3,575 DW	57
Ovary	654 DW	57
Turkey vulture, *Cathartes aura*, California, 1980-81		
Liver	21-44 FW	58
Kidney	16-24 FW	58
Feather	81-110 DW	58
Common raven, *Corvus corax*, California, 1980-81		
Liver	14-45 FW	58
Kidney	17-33 FW	58
Feather	110-160 DW	58
Trumpeter swan, *Cygnus buccinator*, USA, 7 western states, 1976-87, found dead		
Liver, kidney, femur	96 (61-160) FW	118
Blood	5.2 (3.7-8.8) FW	118
Dutch Wadden Sea		
Knots, 3 species, recently-formed primary feathers		
Juveniles	100-400 DW	59
Adults	Max. 977 DW	59
Geese, 3 species, feather vane	93-164 DW; Max. 330 DW	59
Little egret, *Egretta garzetta*, France, found dead		
Bone	100 DW	60
Feather	80 DW	60
Gizzard	140 DW	60
Kidney	70 DW	60
Liver	120 DW	60
Lung	50 DW	60
Muscle	70 DW	60
Stomach	65 DW	60
Chicken, *Gallus* sp.		
Egg yolk	64 DW	61
Kidney	70 DW	61
Liver	69 DW	61
Liver	32 (25-56) FW	62
Pancreas	88 DW	61
Seminal plasma		

Age 30 weeks	9.8 FW	63
Age 60 weeks	9.8-25 FW	63
California condor, *Gymnogyps californianus*, dead on collection, 1980-86		
Nestlings (died from handling shock)		
Liver	22 FW	64
Kidney	17 FW	64
Juveniles (died from cyanide poisoning)		
Liver	33 FW	64
Feather	99-100 DW	64
Subadults (died from lead poisoning)		
Liver	30 FW	64
Kidney	33 FW	64
Feather	85 DW	64
Adults (died from lead poisoning), liver	27-250 FW	64
California, 1980-81, feather	46-130 DW	58
Kern County, California, 1976		
Liver	49 FW	65
Kidney	16 FW	65
White-tailed eagle, *Haliaeetus albicilla*		
Blood, clotted	7.5 FW	66
Brain	20 FW	66
Feather	88 DW	66
Femur	284 (175-390) DW	66
Heart	28 (21-39) FW	
Intestine	50 (27-76) FW	66
Kidney	43 (35-60) FW	66
Liver	68 (38-100) FW	66
Lung	14 (11-17) FW	66
Muscle	55 (42-80) FW	66
Stomach	25 (20-30) FW	66
Bald eagle, *Haliaeetus leucocephalus*, egg, 1968		
Wisconsin	30-56 DW; 4-8 FW	67
Maine	32-52 DW; 4-7 FW	67
Florida	36-65 DW; 5-8 FW	67
Glaucous gull, *Larus hyperboreus*		
Liver	32 (26-47) FW	68
Kidney	46 (37-57) FW	68
Turkey, *Meleagris gallopavo*		
Laying hens		
Serum	6.9 FW	117

Liver	75 DW	117
Nonlaying hens		
Serum	1.6 FW	117
Liver	39 DW	117
Red-breasted merganser, *Mergus serrator*, egg, Lake Michigan, 1978	15 (12-20) FW	69
Black-crowned night-heron, *Nycticorax nycticorax*, liver, prefledglings, 1979		
Massachusetts	602 (482-784) DW	70
North Carolina	649 (479-857) DW	70
Rhode Island	503 (246-885) DW	70
Osprey, *Pandion haliaetus*		
Eastern United States, 1975-82, liver		
Iowa	98 FW	71
Maryland	19-34 FW	71
Massachusetts	89 FW	71
New Jersey	63-120 FW	71
North Carolina	69 FW	71
South Carolina	73 FW	71
Wisconsin	59 FW	71
Virginia	27-150 FW	71
Eastern United States, 1964-73, liver		
Florida	27-36 FW	56
Maryland	18-93 FW	56
New Jersey	22 FW	56
Ohio	60-80 FW	56
All ospreys, liver		
Immatures	67 FW	56
Adults	38 FW	56
Brown pelican, *Pelecanus occidentalis*		
Egg contents		
South Carolina, 1971-72	6.4 (5.5-8.0) FW	119
Florida, 1969-70	6.4 (4.3-8.3) FW	119
Liver		
Found dead		
South Carolina, 1973	26 FW	119
Florida, 1972-73	41-50 FW	119
Georgia, 1972	33 FW	119
Shot		
Florida, 1970	32-55 FW	119

South Carolina, 1973	31-38 FW	119
Greater flamingo, *Phoenicopterus ruber*		
Bone	123 (103-145) DW	60,72
Feather		
Inner barbs	66 (38-105) DW	60,72
Outer barbs	101 (45-190) DW	60,72
Kidney	115 (90-167) DW	60,72
Liver	758 (525-963) DW	60,72,73
Lung	43 (33-56) DW	60,72
Muscle	53 (38-78) DW	60,72
Seabirds		
Albatrosses, 3 species		
Liver	(29-86) FW	68
Kidney	(31-65) FW	68
Fulmars, 2 species		
Liver	36-95 FW	68
Kidney	32-96 FW	
Penguins, 4 species		
Liver	(27-73) FW	68
Kidney	(25 -71) FW	68
Petrels, 7 species		
Liver	(28-81) FW	68
Kidney	(15-78) FW	68
Shearwaters, 2 species		
Liver	(28-54) FW	68
Kidney	(27-88) FW	68
Skuas, 3 species		
Liver	(21-51) FW	68
Kidney	(22-53) FW	68
South Atlantic Ocean, adults, 15 species		
Kidney	28-63 (15-88) FW	74
Liver	22-67 (18-86) FW	74
Spain, infertile eggs, 1985-86		
Golden eagle, *Aquila chrysaetos*	8.4 (5.5-11.9) FW	75
Buzzard, *Buteo buteo*	14 FW	75
White stork, *Ciconia ciconia*	9.8 (6.2-19.2) FW	75
Peregrine, *Falco peregrinus*	11.8 (8.8-16.7) FW	75
Booted eagle, *Hieraetus pennatus*	9.4 (7.7-13.0) FW	75
Black kite, *Milvus migrans*	12.6 (6.4-29.4) FW	75
Common blackbird, *Turdus merula*, from metals-contaminated area (1,750 mg Zn/kg DW soil), feathers of various age (days), feathers washed or unwashed before analysis		

4, unwashed	(100) DW	76
400, unwashed	(546) DW	76
26, washed	(90) DW	76
150, washed	(100) DW	76
400, washed	(162) DW	76
Hoopoe, *Upupa epops*, nestling feathers, age (days)		
7	(200) DW	77
21	(600) DW	77
35	(1,000) DW	77
Mammals		
Antelopes, zoo animals, 7 species, blood serum	4.6-9.4 (1.9-12.9) FW	78
Cattle, cow, *Bos* spp.		
Brain, fetus	50-86 DW	79
Feces		
Normal	220 DW	57
Zinc-poisoned	8,740 DW	57
Food items		
Cereal grains, normal	20-30 DW	80
Grasses, normal	25-60 DW	80
Turnips, beets, chicory roots, potatoes	67-390 DW	80
Hair, distance from Czechoslovakian power plant		
6 km	167 (114-199) FW	81
26 km	32 (21-43) FW	81
Heart, fetus	78-160 DW	79
Kidney		
Adult	92-133 DW	79
Age 2+ years	16 (13-17) FW	82
Fetus	83-251 DW	79
Normal	18 (11-56) FW; 80 DW	57, 82
Zinc-poisoned	670 DW	57
Liver		
Adult	116-150 DW	79
Age 2+ years	40 (27-49) FW	82
Fetus	548-703 DW	79
Normal	135 DW	57
Zinc-poisoned	2,000 DW	57
Milk, days postpartum		
0	21 FW	83
1	12 FW	83
30	6 FW	83

150	4 FW	83
Muscle, Age 2+ years	49 (28-80) FW	82
Dog, *Canis familiaris*		
Serum		
Normal	1.7 (0.6-2.0) FW	84
Zinc-poisoned	29 FW	84
Seminal plasma	1,750 DW	85
Spermatozoa		
Ejaculated	1,040 DW	85
Nonejaculated	150-180 DW	85
Goat, *Capra* sp., milk, days postpartum		
0	17-25 FW	83
1	8-15 FW	83
90	5-6 FW	83
150	3-5 FW	83
Red deer, *Cervus elaphus*, Germany		
Kidney	131 DW	86
Kidney cortex	33 (20-184) FW	87
Liver	111 DW	86
Bank vole, *Clethrionomys glareolus*		
Diet		
Spring	56-70 DW	88
July-December	37-43 DW	88
Bone	145-199 DW	88
Heart	69-74 DW	88
Kidney	79-91 DW	88
Liver	78-103 DW	
Muscle	44-51 DW	
Testes		
December-September	126-163 DW	88
October-November	ND	88
Hooded seal, *Cystophora cristata*, liver	57 FW	89
Indian elephant, *Elephas maximas*, serum		
Young, age <15 years	2.0 FW	90
Adult females	2.8 FW	90
Big brown bat, *Eptesicus fuscus*, captive colony, guano	340 DW	91
Horse, *Equus caballus*, near zinc smelter versus control location		
Kidney	150 DW vs. 17 DW	92
Liver	402 DW vs. 23 DW	92

Pancreas	788 DW vs. 7 DW	92
Serum	2.65 FW vs. 0.8-1.2 FW	92
Kidney cortex	41 FW	87
Plasma, mares, Australia		
All	0.5-1.2 FW	93
Thoroughbreds	0.47 FW	94
Farm horses		
Pregnant	0.52 FW	94
Lactating	0.44 FW	94
Northern sea lion, *Eumetopias jubata*		
Brain	(33-51) DW	95
Heart	(94-101) DW	9'5
Kidney	(99-202) DW	95
Liver	(102-247) DW	95
Lung	(42-69) DW	95
Muscle	(90-140) DW	95
Pancreas	(78-262) DW	95
Spleen	(56-117) DW	95
Long-finned pilot whale, *Globicephala melaena*, Newfoundland, Canada, stranded, 1980-82		
Blubber	1.5 (0.6-3.0) DW	96
Kidney	99 (58-139) DW	96
Liver	234 (68-716) DW	96
Muscle	62 (38-80) DW	96
Gorilla, *Gorilla gorilla gorilla*, captives, plasma	2.4 (0.9-7.3) FW	97
Gray seal, *Halichoerus grypus*		
Blubber	5 FW	14
Kidney	37 FW	14
Liver	84 FW	14
Muscle	43 FW	14
Human, *Homo sapiens*		
Diet		
Protein-rich foods (meat, seafood)	10-50 FW	113
Grains	10-100 FW	113
Vegetables, fruits	<5 FW	113
Erythrocytes	10. 1 - 13.4 FW	98
Hair	>105 FW	98
Milk	3 FW	113
Plasma	0.7-1.6 FW	97,98
Prostate	100 FW	113
Semen	100-350 FW	19
Skin	20-1,000 DW	19

White-beaked dolphin, *Lagenorhynchus albirostris*, Newfoundland, Canada, ice-entrapped, 1980-82, 2-6 years old		
Kidney	85 (68-112) DW	96
Liver	100 (43-136) DW	96
Muscle	53 (36-89) DW	96
Rhesus monkey, *Macaca mulatta*, plasma	0.66-0.98 FW	99
Marine mammals		
Pinnipeds, 9 species		
Liver	(27-97) FW; (123-406) DW	68
Kidney	(11-78) FW; (146-353) DW	68
Muscle	(14-49) FW	68
Cetaceans, 9 species		
Liver	(18-109) FW	68
Kidney	(4-86) FW	68
Muscle	(7-51) FW	68
Sirenians		
Liver	(58-1,101) FW	68
Kidney	(14-54) FW	68
Muscle	(8-28) FW	68
Southeastern bat, *Myotis austroriparius*, Florida, 1981-83, liver		
Near battery salvage plant	31 (27-35) FW	91
Noncontaminated site	28 (26-30) FW	91
Gray bat, *Myotis grisescens*, Florida, 1981-83, guano		
Near battery salvage plant	640 DW	91
Distant sites	390-530 DW	91
Mule deer, *Odocoileus hemionus*, Montana		
Kidney	97 FW	86
Liver	113 FW	86
White-tailed deer, *Odocoileus virginianus*		
Illinois, liver	70 DW	86
Pennsylvania, various distances from zinc smelter		
< 8 km		
Feces	577 (185-1,797) DW	86
Kidney	310 (211-454) DW	86
Liver	167 (137-205) DW	86

10-20 km		
Feces	574 (1,384) DW	86
Kidney	274 (212-355) DW	86
Liver	167 (137-205) DW	86
>100 km		
Feces	185 (77-445) DW	86
Kidney	145 (103-205) DW	86
Liver	132 (95-182) DW	86
Sheep, *Ovis* sp., kidney	22 (14-38) FW	62
Ringed seal, *Phoca hispida*.		
Liver	176 (121-576) DW	100
Kidney	209 (104-441) DW	100
Muscle	79 (52-135) DW	100
Harbor porpoise, *Phocoena phocoena*		
Blubber	4 FW	14
Liver	37 FW	14
Muscle	22 FW	14
Dall's porpoise, *Phocoenoides dalli*		
Adults		
Bone, skin	270-296 FW	101
Heart, liver, pancreas, kidney, whole body	25-51 FW	101
Brain, lung, testes	11-20 FW	101
Blubber, blood, muscle	4-9 FW	101
Fetus		
Liver	82 FW	101
Other tissues	<6 FW	101
Rat, *Rattus* sp., spermatozoa		
Ejaculated	890 DW	85
Nonejaculated	860 DW	85
Striped dolphin, *Stenella coeruleoalba*		
Blubber	16 FW	14
Muscle	11 FW	14
Pig, *Sus* spp., adults		
Kidney	22 (16-33) FW	62,82
Liver	74 (28-160) FW	82
Muscle	24 (8-53) FW	82
Bottle-nosed dolphin, *Tursiops truncatus*		
Blubber	20 FW	14
Muscle	11 FW	14
Polar bear, *Ursus maritimus*		
Kidney	33 (20-49) FW	120
Liver	58-63 (33-100) FW	58,120

Integrated studies		
Electrical transmission towers		
(corroded, galvanized), Ontario, Canada		
Soils		
Near towers	11,480 DW	102
1 km	10,431 DW	102
2 km	10,869 DW	102
5 km	362 DW	102
10 km	160 DW	102
25-50 km	54-70 DW	102
Plants, 5 species, roots and shoots		
Near towers	Max. 1,535 DW	102
1-5 km	Max. 297 DW	102
12-25 km	Max. 55 DW	102
Estuary, Calcasieu River, Louisiana		
Invertebrates		
Periphyton, whole	264 (49-1,300) DW	103
Zooplankton, whole	330 (31-3,550) DW	103
Ctenophores, whole	31-64 DW	103
Hooked mussel,	61 (39-86) DW	103
Brachidontes exustus, soft parts		
American oyster,	3,300 (1,000-7,794) DW	103
Crassostrea virginica, soft parts		
Blue crab, *Callinectes sapidus*, muscle	112 (106-213) DW	103
Brown shrimp, *Penaeus aztecus*, whole	46-61 DW	103
White shrimp, *Penaeus setiferus*, whole	44-62 DW	103
Fish, muscle		
Gulf menhaden, *Brevoortia patronus*	115 DW	103
Gizzard shad, *Dorosoma cepedianum*	25 DW	103
Threadfin shad, *Dorosoma petenense*	29 DW	103
Blue catfish, *Ictalurus furcatus*	35 (16-61) DW	103
Spot, *Leiostomus xanthurus*	22 (217-31) DW	103
Spotted gar, *Lepisosteus oculatus*	(22-239) DW	103
Atlantic croaker, *Micropogonias undulatus*	31 (15-95) DW	103
White mullet, *Mugil curema*	86 DW	103
Southern flounder, *Paralichthys lethostigma*	24 DW	103
Flotation mill (lead-zinc),		
Greenland		
Near outfall		
Suspended particulates	11,600 (1,058-25,700) FW	104
Sediments	Max. 6,799 FW	104
Water	0.035 FW	104

Mussel, *Mytilus edulis*, soft parts	502 (340-813) FW	104
Seaweed, *Fucus disticus*	300 FW	104
Control site		
Suspended particulates	123 FW	104
Sediments	129 FW	104
Water	0.0002 FW	104
Mussel	100 FW	104
Seaweed	8 FW	104
Freshwater lake, India		
Water	0.2 FW	105
Sediment	540 FW	105
Phytoplankton	11-15 FW	105
Zooplankton	60 FW	105
Fish, whole	10 FW	105
Grassland ecosystem		
On a revegetated mine tailings dam		
Soil (1-8 cm depth)	1,915-2,160 DW	106
Vegetation		
Live	157-201 DW	106
Dead	303-646 DW	106
Invertebrates, whole		
Herbivores	355-746 DW	106
Carnivores	403-515 DW	106
Detritivores	769-1,275 DW	106
Field vole, *Microtus agrestis*		
Bony tissues	183-226 DW	106
Soft tissues	160-281 DW	
Common shrew, *Sorex araneus*		
Bony tissues	438-547 DW	
Soft tissues	160-281 DW	106
Control grassland ecosystem		
Soil (1-8 cm depth)	52-62 DW	106
Vegetation		
Live	23-41 DW	106
Dead	24-56 DW	196
Invertebrates, whole		
Herbivores	133-299 DW	106
Carnivores	277-372 DW	106
Detritivores	248-1,095 DW	106
Field vole		
Bony tissues	178-249 DW	106
Soft tissues	53-121 DW	106

Common shrew		
Bony tissues	847-420 DW	106
Soft tissues	145-204 DW	106
Lead smelter, South Australia, marine outfall, whole organisms		
Samples collected 2.5-5.2 km from source		
Sediments	1,270 DW; Max. 16,700 DW	107
Seagrasses, 5 species	823 DW; Max. 3,540 DW	107
Crustaceans, 5 species	148 DW; Max. 767 DW	107
Tunicate, *Polycarpa pediculata*	153 DW; Max. 345 DW	107
Bivalve molluscs, 5 species	4,880 DW; Max. 20,300 DW	107
Carnivorous fish, 8 species	163 DW; Max. 440 DW	107
Omnivorous fish, 3 species	222 DW; Max. 619 DW	107
Herbivorous fish, six-lined trumpeter, *Siphamia cephalotes*	310 DW; Max. 480 DW	107
Samples collected 18-18.8 km from outfall		
Sediments	21 DW	107.
Seagrasses	72 DW	107
Crustaceans	68 DW	107
Tunicate	98 DW	107
Bivalve molluscs	2,590 DW	107
Carnivorous fish	78 DW	107
Omnivorous fish	105 DW	107
Herbivorous fish	97 DW	107
Metals-contaminated forest versus control location, Poland		
Yellow-necked field mouse, *Apodemus flavicollis*		
Liver	119 DW vs. 109 DW	108
Kidney	220 DW vs. 87 DW	108
Hair	179 DW vs. 122 DW	108
Carcass	109 DW vs. 98 DW	108
Bank vole, *Clethrionomys glareolus*		
Liver	120 DW vs. 116 DW	108
Kidney	156 DW vs. 143 DW	108
Hair	243 DW vs. 169 DW	108
Carcass	148 DW vs. 153 DW	108
Old-field community, Ohio, treated with sewage sludge for 10 consecutive years		
Treated area		
Sludge	866 DW	109
Soil	107 DW	109
Perennial plant, *Rubus frondosus*	41 DW	109
Giant foxtail, *Setaria faberii*	97 DW	109

Earthworm, *Lumbricus rubellus*	615 DW	169
Bluegrass, *Poa* spp.	85 DW	109
Japanese brome, *Bromus japonicum*	80 DW	109
Control area		
Perennial plant	14 DW	109
Bluegrass	35 DW	109
Japanese brome	35 DW	109
Zinc smelter, Palmerton, Pennsylvania		
Site 2 km downwind of smelter		
Soil	24,000 DW	110
Foliage, 8 species	660 DW	110
Acorns and berries, 4 species	59 DW	110
Fungi, 4 species	320 DW	110
Moths, 6 species	250-480 DW	110
Beetle, *Dendroides* sp.	1,450 DW	110
Caterpillar, *Porthetria dispar*	280 DW	110
Birds, 10 species, carcasses	140 (93-210) DW	110
White-footed mouse, *Peromyscus leucopus*, carcass	192 DW	110
Short-tailed shrew, *Blarina brevicauda*, carcass	377 DW	110
Site 10 km upwind of smelter		
Soil	960 DW	110
Foliage	118 DW	110
Acorns and berries	27 DW	110
Fungi	120 DW	110
Moths, 9 species	140-340 DW	110
Beetles, 2 species	470 DW	110
Caterpillar, *P. dispar*	170 DW	110
Birds, 10 species, carcasses	120 (78-170) DW	110
White-footed mouse, carcass	145 DW	110
Short-tailed shrew, carcass	201 DW	110
Zinc smelter, Peru, South America, 1980-84		
Soil, kilometers from smelter		
1	575 DW	111
13	183 DW	11]
27	154 DW	111
33	52 DW	111
35-55	16-29 DW	111
Domestic sheep, *Ovis aries*, liver, kilometers from smelter		
13	305 DW	111
29	165 DW	111

>100	77 DW	111
Zinc smelters, various		
Soils	Max. 80,000 DW	112
Trees, foliage	Max. 4,500 DW	112

[a] Concentrations are shown as means, range (in parentheses), maximum (Max.), and nondetectable (ND).

[b] 1. Mann et al. 1989; 2. Mason and Macdonald 1988; 3. Young et al. 1980; 4. Brix and Lyngby 1982; 5. Veleminsky et al. 1990; 6. Greville and Morgan 1989a; 7. Greville and Morgan 1989; 8. Beyer and Cromartie 1987; 9. Morgan and Morgan 1988; 10. Nash 1975; 11. Hopkin et al. 1989; 12. Hopkin et al. 1986; 13. Eisler 1981; 14. Morris et al. 1989; 15. Sullivan et al. 1988; 16. White and Rainbow 1985; 17. Sprague 1986; 18. Prestey et al. 1990; 19. NAS 1979; 20. Cain and Luoma 1986; 21. Amiard et al. 1986; 22. Lobel 1986; 23. Amiard-Triquet et al. 1988; 24. Bryan et al. 1987; 25. Chan 1988a; 26. Chu et al. 1990; 27. Eisler et al. 1978; 28. Weeks and Moore 1991; 29. Rainbow 1989; 30. Anil and Wagh 1988; 31. Walker et al. 1975; 32. Ridout et al. 1989; 33. Cutshall et al. 1977; 34. Waiwood et al. 1987; 35. Bagatto and Alikhan 1987; 36. Khan et al. 1989; 37. Nugegoda and Rainbow 1998b; 38. Shrestha and Morales 1987; 39. Fernandez and Jones 1989; 40. Schmitt et al. 1984; 41. Munkittrick and Dixon 1989; 42. Peterson et al. 1989; 43. Bezuidenhout et al. 1990; 44. Lazos et al. 1989; 45. Lowe et al. 1985; 46. Murphy et al. 1978; 47. Schmitt and Finger 1987; 48. Vas et al. 1990; 49. Hanna 1989; 50. Joseph 1989; 51. Craik and Harvey 1988; 52. Poston and Ketola 1989; 53. Grady et al. 1989; 54. Heinz et al. 1991; 55. Warren et al. 1990; 56. Wiemeyer et al. 1980; 57. Zee et al. 1985; 58. Wiemeyer et al. 1986; 59. Goede 1985; 60. Cosson et al. 1988; 61. Williams et al. 1989; 62, Ellen et al. 1989; 63. Blesbois and Mauger 1989; 64. Wiemeyer et al. 1988; 65. Wiemeyer et al. 1983; 66. Falandysz et al. 1988; 67. Krantz et al, 1970; 08. Thompson 1990; 69. Haseltine et al. 1981; 70. Custer and Mudhern 1983; 71. Wiemeyer et al. 1987; 72. Cosson et al. 1988a; 73. Cosson 1989; 74. Muirhead and Furness 1988; 75. Hernandez et al. 1988; 76. Weyers et al. 1988; 77. Kaur 1989; 78. Vahala et al. 1989; 79. Gooneratne and Christensen 1989; 80. Binnerts 1989; 81. Pisa and Cibulka 1989; 82. Jorhem et al. 1989; 83. Park and Chukwu 1989; 84. Latimer et al. 1989; 85. Saito et al. 1967; 86. Sileo and Beyer 1985; 87. Holterman et al. 1984; 88. Wlostowski et al. 1988; 89. Nielsen and Dietz 1990; 943. Sreekumar and Nirmalan 1989; 91. Clark et al. 1986; 92. Gunson et al. 1982; 93. Auer et al. 1988b; 94. Auer et al. 1988a; 95. Hamanaka et al. 1982; 96. Muir et al. 1988; 97. McGuire et al. 1989; 98. Casey and Hambidge 1980; 99. Keen et al. 1989; 100. Wagemann 1989; 101. Fujise et al. 1988; 102. Jones and Burgess 1984; 103. Ramelow et al. 1989; 104. Loring and Astound 1989; 105. Prahalad and Seenayya 1989; 106. Andrews et al. 1989; 107. Ward et al. 1986; 108. Sawicka-Kapusta et al. 1987; 109. Levine et al. 1989; 110 Beyer et al. 1985; 111. Reif et al. 1989; 112. Buchauer 1971; 113. Elinder 1986; 114. Vymazal 1986; 115. Dwyer et al. 1988; 116. Willis 1985a; 117. Richards 1989; 118. Blus et al. 1989; 119. Blus et al. 1977; 120. Norheim et al. 1992; 121. Schmitt and Brumbaugh 1990.

Terrestrial plants growing beneath corroded galvanized fencing have been poisoned by zinc (Jones and Burgess 1984). Vegetables are relatively low in zinc, but growing plants can accumulate zinc applied to soils (Geyer 1986). High soil level of zinc is the primary cause of vegetation damage near zinc smelters (Buchauer 1971; Leonard and Gerber 1989). Elevated zinc concentrations in soils near zinc smelters inhibit seedling root elongation and probably prevent establishment of invader species in denuded areas (Buchauer 1971). Lichen species richness and abundance were reduced by about 90% in lichen communities near a Pennsylvania zinc smelter; elevated zinc concentrations were the probable cause of the impoverished lichen flora (Nash 1975). Soils and vegetation surrounding zinc smelters in Palmerton, Pennsylvania were grossly contaminated with zinc, cadmium, and lead. Zinc was primarily responsible for the destruction of trees and subsequent erosion of the soil, reductions in moss and lichen flora, reductions in litter arthropod populations, and reductions in species diversity of soil fungi and bacteria; zinc residues were elevated in slugs and millipedes (Sileo and Beyer 1985; Beyer 1988). Soil litter invertebrates were rare or absent 2 km downwind of the smelter; unlike soil litter invertebrates from more distant sites, invertebrates collected up to 10 km upwind of the smelters had significantly elevated zinc concentrations (Beyer et al. 1985).

The maximum zinc concentration in earthworms collected from a contaminated site was 1,600 mg/kg DW whole animal; for uncontaminated sites it was 650 mg/kg (Beyer and Cromartie 1987). Whole body zinc concentrations in earthworms (*Dendrodrilus rubidus*, *Lumbricus rubellus*) tended to reflect zinc concentrations in

soil, although zinc accumulations in both species seem to be physiologically regulated when soil zinc values exceeded 1,000 mg/kg DW (Morgan and Morgan 1988).

Whole body zinc content of terrestrial isopods seems to reflect soil zinc levels and may be a useful indicator of soil contamination (Hopkin et al. 1989). *Porcellio scaber*, a terrestrial isopod known as a woodlouse, is recommended as a biological indicator of zinc contamination because of the positive correlation between zinc content in soil or leaf litter and woodlouse hepatopancreas. Zinc content in *Porcellio*, litter, and soil near a zinc smelter was >1,000 mg/kg DW in whole isopod, >9,000 mg/kg DW in hepatopancreas, > 10,000 mg/kg DW in litter, and >50,000 mg/kg DW in soil (Hopkin et al. 1986).

Interspecies variability in zinc content of terrestrial invertebrates is large and governed by numerous modifiers. For example, whole body zinc content in closely-related species of terrestrial gastropods collected from a single contaminated site was between 600 and 1,200 mg/kg DW (Greville and Morgan 1989a). In grey field slugs (*Deroceras reticulatum*), zinc was highest in late spring and lowest in summer and positively correlated with tissue cadmium concentrations; starvation for 16 days had no effect on body zinc concentrations (Greville and Morgan 1989b). Zinc tends to concentrate in mechanical structures of various invertebrates, such as mandibular teeth. High concentrations of zinc are reported in jaws of polychaete worms, cutting edges of the mandibles of herbivorous insects, mandibles of various species of beetles, copepod mandibles, chaetognath teeth and spines, mandibular teeth of ants, and fangs of spiders (Schofield and Lefevre 1989). Honey bees (*Apis mellifera*) collected near a lead smelting complex at East Helena, Montana, had depressed whole body zinc concentrations despite increased ambient air zinc values; however, whole body burdens of arsenic, cadmium, copper, and lead were significantly elevated and may have influenced zinc kinetics (Bromenshenk et al. 1988). Also, pollen was usually the most indicative source of zinc and other heavy metals in bees (Veleminsky et al. 1990).

Aquatic Organisms

Concentrations of zinc in tissues of aquatic organisms are usually far in excess of that required for normal metabolism. Much of the excess zinc is bound to macromolecules or present as insoluble metal inclusions in tissues (Eisler 1981, 1984; EPA 1987). Diet is the most significant source of zinc to aquatic organisms and is substantially more important than uptake from seawater (Eisler 1981, 1984). In general, zinc concentrations in sediments and tissues of aquatic organisms are elevated in the vicinity of smelters and other point sources of zinc and decrease with increasing distance (Ward et al. 1986; Table 4).

Freshwater algae in Canadian mine tailing environments heavily concentrate zinc and other metals and may retard metal dispersion through the water column (Mann and Fyfe 1988). Zinc levels in field collections of marine algae and macrophytes are usually at least several orders of magnitude higher than zinc concentrations in the surrounding seawater (Eisler 1981). In general, concentrations in marine aquatic flora were high when seawater zinc concentrations were elevated, although the relation was not linear. Marine flora, especially red and brown algae, are among the most effective marine zinc accumulators. Increasing accumulations of zinc in marine algae were associated with decreasing light intensity, decreasing pH, increasing temperature, decreasing levels of DDT, and increasing oxygen. Ionic zinc was accumulated more rapidly than other forms of zinc (Eisler 1981). Many species of marine algae had zinc concentrations >1 g/kg DW (Eisler 1980). These grossly elevated levels were usually associated with nearby industrial or domestic outfalls containing substantial amounts of zinc (Eisler 1981). In eelgrass (*Zostera marina*), zinc concentrations increased with age of leaf (Brix and Lyngby 1982).

In the Fal estuary, England, long-term metal pollution during the past 120 years resulted in zinc sediment levels between 679 and 1,780 mg/kg DW, producing benthic communities that favor zinc-tolerant organisms, such as oysters and nereid polychaetes, and a general impoverishment of mussels, cockles, non-nereid polychaetes, and gastropods (Bryan et al. 1987).

Zinc in molluscs is usually associated with high molecular weight proteins, with diet (as opposed to ambient water zinc concentrations), from collection locales with elevated sediment zinc burdens, and with particulate matter from dredging and storm perturbations (Eisler 1981). Zinc levels in molluscs were highest in animals collected near anthropogenic point sources of zinc. Excess zinc accumulations do not seem to affect normal molluscan life processes, and zinc is frequently accumulated far in excess of the organism's immediate needs (Eisler 1981). American oysters (*Crassostrea virginica*), for example, may naturally contain up to 4 g Zn/kg FW

soft parts; this is comparable to accumulations observed in oysters exposed to 0.2 mg Zn/L for 20 weeks (NAS 1979). Zinc tends to accumulate in the molluscan digestive gland and stomach as excretory granules and in the kidney as concretions (Eisler 1981; Sprague 1986; Sullivan et al. 1988). The preferred storage site in mussels and scallops is the kidney and in oysters, the digestive gland (Sprague 1986). In oysters, granules may contain up to 60% of the total body zinc, explaining, in part, how some shellfish can exist with such high body burdens (Sprague 1986).

Zinc in molluscan tissues is usually elevated under conditions of increasing water temperature and pH and decreasing salinity (Eisler 1981); however, zinc accumulation kinetics in molluscs vary considerably among species (Chu et al. 1990). Variations in zinc content of clam tissues were associated with seasonal changes in tissue weights (Cain and Luoma 1986). Unlike conspecifics collected at more distant sites, gastropods nearest a ferronickel smelter had elevated zinc concentrations in the hepatopancreas; however, there were no consistent seasonal variations (Nicolaidu and Nott 1990). Fluctuations in zinc content of common mussels (*Mytilus edulis*) related to size or season of collection were sufficient to conceal low chronic or short-term pollution (Amiard et al. 1986). Diet, which is the primary route of zinc accumulation in most molluscs, had no significant effect on whole body zinc content of certain predatory marine gastropods. Whole body zinc concentrations of gastropod oyster drills (*Ocenebra erinacea*) were between 1,451 and 2,169 mg/kg DW and remained unchanged after feeding for 6 weeks on Pacific oysters (*Crassostrea gigas*) containing 1,577 mg Zn/kg DW or common mussels (*Mytilus edulis*) containing 63 mg Zn/kg DW (Amiard-Triquet et al. 1988).

High zinc concentrations in crustaceans are usually associated with industrial contamination. In barnacles (*Balanus* spp.), high (>3.3 g/kg DW soft parts) levels are attributed to inorganic granules that contain up to 38% zinc and that accumulate in tissue surrounding the midgut (Eisler 1980, 1981). In descending order of chemical abundance, the granules consist of phosphorus, zinc, potassium, sulfur, and chlorine (Thomas and Ritz 1986). These insoluble, membrane-limited spheres form in response to high zinc levels in the ambient seawater within 12 days of exposure and concentrate in specified cells around the gut: the stratum perintestinale (Walker et al. 1975; Sprague 1986; Thomas and Ritz 1986). Zinc granules in barnacles represent a detoxification mechanism for surplus zinc (Thomas and Ritz 1986). Older barnacles have greater whole body zinc accumulations than younger stages, and accumulations change seasonally (Anil and Wagh 1988). Zinc concentrations in marine crustacean tissues are usually <75 mg/kg FW or <100 mg/kg DW; exceptions include hepatopancreas, molts, eggs, fecal pellets, and barnacles (Table 4). In crustaceans, zinc is slightly elevated in hepatopancreas but in most tissues only 2 to 3 times higher than in muscle (Sprague 1986). For marine crustaceans, the highest concentration recorded in muscle was 57 mg Zn/kg FW in the king crab, *Paralithodes camtschatika* (NAS 1979), and was associated with two metal binding proteins of molecular weight 11,500 and 27,000 (Eisler 1981). In crustacean tissues, zinc levels were higher in summer at lower salinities and in young animals (Eisler 1981), although young amphipods had higher zinc residues than older stages (Rainbow 1989). Seasonal accumulations of whole body zinc in the shrimp (*Palaemon serratus*) during spring and summer and loss in winter seem to reflect water zinc concentrations in the range of 0.0 to 9.0 µg/L (Alliot and Frenet-Piron 1990). Zinc is present in crustacean serum at concentrations >1,000 times greater than in ambient seawater in serum, it serves primarily as a cofactor of carbonic anhydrase--the principal enzyme involved in calcification. Serum zinc concentrations in crustaceans seem to be independent of season and water temperature or salinity (Sprague 1986).

Molting results in a 33-50% loss of total zinc in marine crustaceans; molts, together with fecal pellets, constitute an important vehicle of zinc transfer in marine ecosystems (Eisler 1981). The freshwater opossum shrimp (*Mysis relicta*) can transport zinc from sediments into the water column and in the reverse during their migratory cycle. *Mysis relicta* and other benthic invertebrates play an important role in determining the concentration of zinc and other metals in lake sediments (Van Duyn-Henderson and Lasenby 1986). Unlike decapod crustaceans, marine amphipods do not regulate body zinc concentrations; amphipod body burdens of zinc may reflect sediment total zinc levels and suggest that certain groups may be suitable bioindicators (Rainbow et al. 1989). Molting had no effect on body zinc concentration in four species of adult marine amphipods (Weeks and Moore 1991), and this forces a reexamination of the role of cast exuviae in zinc transport.

In annelids, zinc content was highest in nonselective deposit feeders, omnivores, and carnivores and from animals collected from sediments with elevated zinc levels (Eisler 1981). Freshwater tubificid worms have the potential to increase zinc concentrations in the water column, particularly during short episodes of high

burrowing activity (Krantzberg and Stokes 1985). A high zinc content seems to be a structural characteristic of jaws of marine nereid worms (Table 4). In the marine polychaete worm *Nereis diversicolor*, zinc is localized in the gut wall, epidermis, nephridia, and blood vessels; most of the body zinc is present in wandering amoebocytic cells of excretory organs. Zinc in *Nereis* may be present as insoluble granules in membrane bound vesicles; excretion is through exocytosis with the aid of amoebocytes (Fernandez and Jones 1989). Unlike the insoluble zinc phosphate granules of molluscs and crustaceans, zinc granules in *Nereis* were very soluble and retained only by sulfide precipitation (Pirie et al. 1985).

Marine vertebrates, including fish and elasmobranchs, have lower zinc concentrations in tisues (6-400 mg/kg DW) than marine plants and invertebrates (Eisler 1980, 1981, 1984). Highest concentrations in muscle of marine fish (20.1-25.0 mg/kg FW) were recorded in the northern anchovy (*Engraulis mordax*) and the Atlantic menhaden (*Brevoortia tyrannus*; NAS 1979). The highest zinc concentrations measured in whole freshwater fish in the conterminous United States in 1978-79 were in common carp (*Cyprinus carpio*) from Utah; concentrations in carp from Utah were between 70 and 168 mg Zn/kg FW versus an average of 63 mg Zn/kg FW for this species collected elsewhere (Lowe et al. 1985). Zinc concentrations in fish tend to be higher near urban areas (Peterson et al. 1989); highest in eggs, viscera and liver (Eisler and LaRoche 1972; Eisler 1981); lowest in muscle (Eisler 1981); positively correlated with metallothionein concentrations (Overnell et al. 1987b); lower in all tissues with increasing age and growth (Eisler and LaRoche 1972; Eisler 1981, 1984; Grady et al. 1989); and relatively unaffected by water salinity, temperature, or copper concentrations (Eisler and LaRoche 1972; Eisler 1981). Zinc residue data from marine fish that were dead on collection are of limited worth because dead fish accumulate zinc from seawater at a substantially higher rate than living teleosts (Eisler 1981).

Zinc concentrations in fish and other aquatic vertebrates are modified by diet, age of the organism, reproductive state, and other variables. In fish, diet is the major route of zinc uptake and juveniles accumulate zinc from the medium more rapidly than embryos or larvae (Cutshall et al. 1977; Eisler 1981). Because the diet of many teleost carnivores changes drastically with age and because upper trophic level vertebrates are frequently used as indicators of water quality, more research into zinc burdens in prey organisms is needed (Eisler 1984). A reduction in serum zinc during egg formation in a flatfish (*Pleuronectes platessa*) may represent a transfer of zinc to eggs (Overnell et al. 1987b). High (>35 mg/kg FW) zinc concentrations in eggs of Atlantic salmon are sometimes associated with increasing mortality, although low (14 mg/kg FW) concentrations seem to have no adverse effect on survival (Craik and Harvey 1988). Zinc concentrations in Atlantic salmon milt ranged from 0.5 to 5.5 mg Zn/kg and was linearly proportional to spermatozoan abundance (Poston and Ketola 1989). In lakes containing 1,150 mg Zn/kg sediment and 209-253 µg Zn/L water column, white sucker (*Catostomus commersoni*) females did not grow after sexual maturity and had increased incidences of spawning failure. Alterations in growth and reproduction were related, in part, to nutritional deficiencies as a result of chronic effects of elevated sediment zinc on the food base of the sucker, that is, invertebrate fauna were absent in the uppermost 7 m (Munkittrick and Dixon 1988). Eggs of the white sucker incubated at a metals-contaminated site (400 µg Zn/L), but not eggs of conspecifics, at a noncontaminated (2.7 µg Zn/L) site, produced larvae with a decreased tolerance to copper and with elevated zinc body burdens; larval size and fertilization rate were the same at both sites (Munkittrick and Dixon 1989).

Birds

Zinc residues were elevated in birds collected near zinc smelters (Beyer 1988). In general, the highest concentrations of zinc in birds are in the liver and kidney and the lowest in muscle (Eisler 1981, 1984). In giant Canada geese (*Branta canadensis maxima*), more zinc is contained in red muscle than in white muscle and more in slow contracting muscle than in fast muscle (Rosser and George 1986). Zinc concentrations in marine birds normally are between 12 mg/kg FW in eggs and 88 mg/kg FW in the liver. The highest recorded concentration of zinc in a marine bird was 541 mg/kg DW in the liver of a booby (*Sula* sp.) that died from polychlorinated biphenyl poisoning. Elevated zinc levels in these birds may have been a manifestation of toxicant-induced stress (i.e., breakdown in osmoregulatory processes), as in other taxonomic groups (Eisler 1981). Seabirds with high zinc concentrations in the liver and kidney tend to have high cadmium levels in these tissues (Muirhead and Furness 1988). In flamingos, zinc in the liver positively correlated with copper levels in the liver and kidney and with metallothionein levels in the kidney (Cosson 1989). In egrets, zinc positively correlated with metallothionein protein levels in the liver (Cosson 1989). In blue-winged teals (*Anas discors*), zinc concentrations were higher in the liver than in muscle, higher in males than in females, and higher in

autumn than in spring (Warren et al. 1990). Zinc concentrations in the liver of black-crowned night-herons (*Nycticorax nycticorax*) were usually higher in younger birds, although weight and sex had no direct effect on zinc content (Custer and Mulhern 1983). Zinc concentrations in tissues and feathers of dead California condors (*Gymnogyps californianus*) that had died from a variety of causes (Table 4) were similar to those in turkey vultures (*Cathartes aura*), common ravens (*Corvus corax*), and ospreys (*Pandion haliaetus*) and are considered normal (Wiemeyer et al. 1988). The highest recorded concentration in condor liver of 250 mg/kg FW approaches those in livers of mallards (*Anas platyrhynchos*) that died from high dietary loadings of zinc (Wiemeyer et al. 1988). Zinc concentrations in the liver of ospreys were similar between age groups and sexes (Wiemeyer et al. 1987). With the onset of egg production in turkeys (*Meleagris gallopavo*), serum zinc in hens increased from 1.6 to 6.9 mg/L and remained significant elevated throughout egg laying; during this same period, zinc concentration in the liver declined from 75 to 39 mg/kg DW, although total zinc in the liver increased because of an increase in liver weight (Richards 1989a).

Zinc concentrations in the sediments of the Rhine River increased about 6 times between 1900 and 1950 and have remained stable since then. But migratory waterfowl from this collection locale do not have elevated zinc concentrations in their primary feathers (Goede 1985). Zinc content in feathers of the hoopoe (*Upupa epops*) increased from 200 mg/kg DW at age 7 days to 1,000 mg/kg DW at age 35 days (Kaur 1989). Hoopoe populations are declining in India and this decline is said to be associated with increasing zinc concentrations in feathers (Kaur 1989). Feathers of the greater flamingo (*Phoenicopterus ruber*) are proposed indicators of atmospheric zinc contamination: the average zinc content was 53% more in outer barbs of the black primary feathers exposed to air pollution than in inner barbs (Cosson et al. 1988a). More research into the use of feathers as indicators of zinc contamination is needed.

Zinc concentrations in seminal plasma are about 100 times lower in domestic chickens (*Gallus* sp.) than in humans and most other mammals, except sheep. Concentrations of zinc in fowl seminal plasma after in vivo storage of spermatozoa for 24 h at 4°C were near the threshold values toxic to spermatozoa (Blesbois and Mauger 1989), suggesting that poultry spermatozoa normally function near their lower lethal zinc threshold.

Mammals

White-tailed deer (*Odocoileus virginianus*) collected near a zinc smelter, but not conspecifics from more distant sites, had elevated tissue zinc concentrations. Deer with zinc concentrations of 150 mg/kg FW (750 mg/kg DW) in the renal cortex potion of the kidney had swollen joints, lameness, and joint lesions similar to those of zinc-posioned horses from the same area (Sileo and Beyer 1985). Zinc was elevated in the kidney cortex of red deer (*Cervus elaphus*) and older deer tended to have higher concentrations (as high as 184 mg/kg DW) than younger deer (as low as 20 mg/kg FW); in older deer, zinc was associated with the metallothionein fraction (Holterman et al. 1984). Zinc residues were usually elevated in rodents near smelters (Beyer et al. 1988). Rodents from metals-contaminated forests had zinc loadings in tissues similar to those from control locations, although lead and cadmium were significantly elevated in the contaminated zone (Sawicka-Kapusta et al. 1987). Elevated zinc concentrations in mine tailings reportedly do not represent a notable contamination hazard to the invading mammalian fauna, although zinc concentrations in invertebrates, especially earthworms, and vegetation were elevated (Andrews et al. 1989; Table 4).

Otters (*Lutra lutra*) were found only on a single unpolluted tributary of a river system contaminated by zinc mine drainage waste, suggesting that a contaminated food supply may be responsible for the avoidance by otters of otherwise suitable habitat (Mason and Macdonald 1988).

Marine mammals collected near heavily urbanized or industrialized areas or near zinc pollution point sources, but not individuals of the same species and of similar age from relatively pristine environments, usually had elevated zinc concentrations (Eisler 1984). Zinc concentrations in tissues of the ringed seal (*Phoca hispida*) were essentially the same in animals near a lead-zinc mine and in animals in a distant reference site, although lead and selenium burdens were elevated in the vicinity of the mine site (Wagemann 1989). Concentrations of zinc in tissues of the Northern sea lion (*Eumetopias jubata*) were highest in the liver and pancreas and next highest in descending order in the kidney, muscle, heart, spleen, and lung; this rank order is comparable to that in human tissues (Hamanaka et al. 1982). There is considerable variation among species in tissue zinc concentrations; threefold differences are not uncommon for the same tissue in different species of marine mammals (Muir et al. 1988). Marine mammals contained the lowest zinc concentrations (2-505 mg/kg DW, elevated in the liver) of all groups of marine organisms examined. Because zinc is usually available in sufficient

quantity in the marine environment and is usually accumulated in excess of the organism's immediate needs, it remains unclear why zinc is comparatively depressed in tissues of marine mammals (Eisler 1981).

Zinc toxicosis in horses near a zinc smelter was characterized by lameness, swollen joints, and unkempt appearance, particularly in foals. Zinc concentrations in afflicted foals, but not in foals at more distant sites, were elevated in the pancreas, liver, kidney, and serum (Gunson et al. 1982). Foals born near the smelter had joint swellings that were attributable to generalized osteochondrosis; lesions were similar to those induced experimentally in animals fed high zinc diets and may have been the result of a zinc-induced abnormal copper metabolism (Gunson et al. 1982). Concentration of zinc in tissues of horses from farms near the Palmerton smelter were extremely high and approaching lethal thresholds in some cases; zinc poisoning was a cause of debility and death of foals (Sileo and Beyer 1985). Grazing mares managed with standard husbandry had significant monthly variations in plasma zinc because, in part, of dietary factors such as nutritional supplementation and seasonal variations in the quality of grazing pasture (Auer et al. 1988b). Peak plasma zinc levels in horses positively relate to age (in weanlings age 22-52 weeks) and to summer diets (Cymbaluk and Christison 1989).

Dairy cattle near a lead and zinc ore processing facility did not have elevated blood or hair zinc levels, although daily zinc intake was 5.6 mg/kg BW versus 1.2 mg/kg BW daily by cattle in a control area (Milhaud and Mehannaoui 1988). In cattle, proximity to zinc refineries did not result in significant elevation of zinc concentrations in the liver and kidney (Spierenburg et al. 1988). However, cows living within 6 km of a power plant in Czechoslovakia, but not a herd at a 26-km distance, had elevated zinc loadings in hair and poor reproduction (Pisa and Cibulka 1989). In adult bovines, zinc reserves are usually small and located primarily in the skeleton and muscle, although appreciable hepatic accumulations can occur in the fetus. At 270 days of gestation, for example, 30% of zinc in fetal cattle is in the liver; zinc concentration is about 4 times higher in the fetal than in the maternal liver (Gooneratne and Christensen 1989). Liver concentrations >120 mg Zn/kg DW in cattle are frequently associated with elevated dietary zinc loadings (Binnerts 1989). Concentrations of zinc in milk of cows and goats varied significantly between breeds and with zinc level in diet and declined markedly after parturition (Park and Chukwu 1989).

A normal 70-kg human male contains 1.5-2.0 g zinc or about 21-29 mg Zn/kg BW; normal zinc uptake is 12-15 mg daily, equivalent to 0.17-0.21 mg/kg BW (Prasad 1979). Foods rich in zinc are seafoods, meats, grains, dairy products, nuts, and legumes (Goyer 1986). About 90% of the total body zinc is in the musculoskeletal system (Rosser and George 1986). Highest zinc concentrations of 100-200 mg/kg occur in the prostate, eye, brain, hair, bone, and reproductive organs; intermediate concentrations of 40-50 mg/kg occur in the liver, kidney, and muscle (NAS 1979; Casey and Hambidge 1980). In blood, about 80% of the total zinc is in red cells where it is associated with carbonic anhydrase. The mean plasma zinc level is about 0.9 mg/L; about half is in a freely-exchangeable form loosely bound to albumin; most of the remainder is tightly bound to macroglobulins and amino acids, especially histidine and cysteine (Casey and Hambidge 1980; Goyer 1986). The greatest zinc concentration in the human body is in the prostate and may be related to the elevated levels of acid phosphatase, a zinc-containing enzyme in that organ (Goyer 1986). The prostate gland contributes zinc to spermatozoa in dogs--a necessary process for canine fertility and fecundity; in rats, however, the prostate does not contribute to zinc in spermatozoa, and its function is not essential for reproduction in rats (Saito et al. 1967).

Zinc Deficiency Effects

General

Zinc is important in the metabolism of proteins and nucleic acids and is essential for the synthesis of DNA and RNA. Zinc deficiency has been reported in humans and a wide variety of plants and animals--with severe effects on all stages of reproduction, growth, and tissue proliferation in the young. In early gestation, zinc deficiency may cause severe congenital abnormalities. Later in gestation, deficiency can cause growth inhibition and brain growth impairment, leading to altered behavioral development after birth. Feeding a low zinc diet to lactating dams produces signs of zinc deficiency in suckling pups. In humans, zinc deficiency is associated with delayed sexual maturation in adolescent males; poor growth in young children; impaired growth of hair, skin, and bone; disrupted Vitamin A metabolism; and abnormal taste acuity, hormone metabolism, and immune function.

Terrestrial Plants

Zinc deficiencies in citrus groves in California, pecan trees in Texas, and various crops in Australia resulted in large crop losses (Vallee 1959). Applications of zinc salts were effective under acidic soil conditions. But neutral or alkaline soils rendered zinc salts insoluble and zinc therapy ineffective. Zinc salts sprayed on leaves or injected into tree trunks overcame the problems of soil solubility and have generally been successful (Vallee 1959). Zinc is usually bound strongly in plants, particularly in grains, markedly decreasing its availability to animal consumers. Binding is attributed mainly to high content of phytate and also to high levels of fiber hemicelluloses, and amino acid-carbohydrate complexes (Casey and Hambidge 1980). Whole-grain cereals and legumes are considered rich sources of zinc (Casey and Hambidge 1980).

Aquatic Organisms

Nutritional zinc deficiency is rare in aquatic organisms (Spear 1981), although reports are available of experimentally-induced zinc deficiency in algae, sponges, daphnids, echinoderms, fish, and amphibians.

Experimental zinc deficiency in euglenoids (*Euglena gracilis*) was associated with arrested growth and abnormal cell differentiation and development, leading to extensive teratological abnormalities. Zinc-deprived *Euglena* survived for extended periods through decreased metabolism (Falchuk et al. 1985; Falchuk 1988). Marine algae stopped growing when ambient zinc concentrations fell below 0.7 µg/L, and zinc-deficient cultures of freshwater algae were unable to metabolize silicon (Vymazal 1986).

A freshwater sponge (*Ephydatia fluviatilis*) grew normally at a concentration of 0.65 µg Zn/L, but growth was reduced at lower concentrations (Francis and Harrison 1988).

Daphnids (*Daphnia pulex*, *Daphnia magna*) reared for six brood cycles in zinc-free water showed reduced survival, inhibited reproduction, and cuticle damage (Keating and Caffrey 1989).

Zinc is important in pH regulation of sperm of marine invertebrates. Zinc reduction in semen to <6.5 µg/L adversely affected sperm pH and motility in sea urchins (*Strongylocentrotus purpuratus*, *Lytechnicus pictus*), horseshoe crab (*Limulus polyphemus*), and starfish (Clapper et al. 1985a, 1985b).

Rainbow trout fry fed diets containing 1-4 mg Zn/kg ration had poor growth, increased morality, cataracts, and fin erosion; supplementing the diet to 15-30 mg Zn/kg alleviate these signs (Spry et al. 1988). Spry et al. (1988) also fed rainbow trout fry diets containing 1, 90, or 590 mg Zn/kg ration and simultaneously exposed them to a range of waterborne zinc concentrations of 7, 39, 148, or 529 µg Zn/L. After 16 weeks, the 7 µg Zn/L plus 1 mg/kg diet group showed clear signs of deficiency including a significantly reduced plasma zinc concentration (which was evident as early as the first week of exposure), reduced growth (with no growth after week 12), decreased hematocrit, and reduced plasma protein and whole body zinc concentration. Elevating waterbone zinc to 39 or 148 µg Zn/L partially corrected the deficiency but did not restore plasma or whole body zinc to initial levels or in fish raised for 16 weeks on a zinc-adequate diet of 90 mg Zn/kg ration. There were no toxic effects at any other dietary-waterborne zinc mixture. It was concluded that zinc uptake from water was independent of uptake from diet because at any dietary zinc level, an increase in the waterborne zinc resulted in an increase in whole body zinc. In freshwater, where waterborne concentrations of <10 µg Zn/L are most commonly encountered, waterborne zinc contributions to whole body zinc loadings are probably insignificant. When dietary zinc was adequate (i.e., 90 mg Zn/kg ration), the contribution of waterborne zinc was significant in the case of rainbow trout (Spry et al. 1988). In marine teleosts, diet is the major zinc source when seawater contained <15 µg Zn/L; at higher ambient concentrations of 600 µg Zn/L, waterborne zinc contributed up to 50% of the total body zinc burden (Spry et al. 1988).

Experimentally-produced zinc deficiency in toad embryos resulted in adults with abnormal ovarian development, altered meiotic and ovulation processes, and embryos with a high incidence of congenital malformations (Herkovits et al. 1989).

Birds

Zinc deficiency in the chicken, turkey, and Japanese quail is characterized by low survival, reduced growth rate and food intake, poor feathering, shortening and thickening of long bones of legs and wings, reduced egg production and hatchability, skeletal deformities in embryos, an uncoordinated gait, reduced bone alkaline

phosphatase activity, and increased susceptibility to infection (Blamberg et al. 1960; NAS 1979; Prasad 1979; Apgar 1985; O'Dell et al. 1989); Stahl et al. 1989a).

Laying hens (*Gallus* sp.) had low egg hatchability on diets that contained 6 mg Zn/kg and produced chicks that were weak and poorly feathered; these chicks usually died within a few days on 8-9-mg Zn/kg diets (Blamberg et al. 1960). Zinc-deficient chicks (13-16 mg Zn/kg DW diet for 4 weeks) had pathological defects in epiphyseal cartilage; no interference with calcification was noted in controls fed diets containing 93-96 mg Zn/kg feed (Westmoreland and Hoekstra 1969). Pullets fed diets containing 28 mg Zn/kg for 4 months and then 4 mg Zn/kg ration for 4.5 months produced few hatchable eggs after 4 months; prevalent malformations included faulty trunk and limb development, missing vertebrae, missing limbs and toes, abnormal brain morphology, small eyes, and skeletal malformations (Blamberg et al. 1960). Most zinc deficiency effects were reversed by increasing dietary zinc concentrations to 96-120 mg/kg (Blamberg et al. 1960).

Chicks of the Japanese quail fed an excess of zinc (25-30 mg Zn/kg diet) during their first week of life were protected during a subsequent period of zinc deprivation (1 mg Zn/kg diet for 1 week). Birds that received an initial intake of zinc in excess of requirements grew significantly better than birds on a minimal amount of zinc. Japanese quails may store excess zinc in bones; this zinc store may become available during a subsequent period of zinc deprivation, especially during a period of rapid bone growth (Harland et al. 1975); but this requires verification.

Egg production constitutes a major loss of zinc and other trace metals by the laying hen. Vitellogenin mediates the transfer of zinc from the liver to the maturing oocyte, ultimately resulting in deposition into yolk of the newly formed egg (Richards 1989a). More research into the role of zinc in avian reproduction seems needed.

Mammals

Compared with zinc toxicity, zinc deficiency is a much more frequent risk to mammals (Leonard and Gerber 1989). Zinc is required in all stages of the cell cycle, and deficiency adversely affects metabolism of DNA, RNA, proteins, and activity of carbonic anhydrase, lactic dehydrogenase, mannosidase, and other enzymes (NAS 1979; Prasad 1979, 1980; Apgar and Everett 1988). In zinc deficiency, the activity of various zinc-dependent enzymes are reduced in testes, bone, esophagus, and kidney of rats, and alkaline phosphatase activity is reduced in bone and plasma of zinc-deficient rats, pigs, and cows (Prasad 1979; Vergnes et al. 1990). Deficiency leads to loss of appetite and taste, skin disturbances, slow wound healing, impaired brain development, deficient immune system, and disrupted water metabolism (Binnerts 1989). Zinc deficiency adversely affected testicular function in humans and animals and seems to be essential for spermatogenesis and testosterone metabolism (Prasad 1980). Zinc deficiency in young men with very low zinc intakes resulted in testicular lesions and reduced accessory gland weights, primarily from reduced food intake and growth (Apgar 1985). Zinc deficiency during pregnancy produced low birth weight, malformations, and poor survival in rats, lambs, and pigs; the role of zinc in human reproductive problems is still unclear (Apgar 1985). Zinc-deficient diets for ruminants and small laboratory animals usually contain <1 mg Zn/kg ration, although rats show deficiency at <12 mg Zn/kg ration (Elinder 1986). Zinc deficiency has been documented in humans, small laboratory animals, domestic livestock, minks, and monkeys; signs of severe zinc deficiency in mammals include decreased food intake, growth cessation, fetal malformations, testicular atrophy, swelling of feet, excessive salivation, dermal lesions, parakeratosis of the esophagus, impaired reproduction, hair loss; unkempt appearance, stiffness, abnormal gait, skin and organ histopathology, and hypersensitivity to touch (NAS 1979; Jameson 1980; Elinder 1986; Gupta et al. 1988; O'Dell et al. 1989). Selected examples of zinc deficiency in various species follow.

Zinc deficiency in humans is rare and usually associated with severe malabsorption, parenteral alimentation lacking zinc, or geophagia (Sternlieb 1988). Symptoms of zinc deficiency depend in part on age, acuteness of onset, duration and severity of the zinc depletion, and the circumstances in which deficiency occurs. Many of the features of zinc deficiency observed in humans are similar to those in zinc-deficient animals (Casey and Hambidge 1980). Simple nutritional deficiency from marginal zinc intake may be common even in the United States (Casey and Hambidge 1980). Factors of zinc deficiency include inadequate dietary intake (protein-calorie malnutrition), decreased availability (high fiber-phytate diets), decreased absorption, excessive losses (increased sweating, burns), increased requirements (rapid growth, pregnancy, lactation), as well as old age, alcoholism, and possible genetic defects (Casey and Hambidge 1980). Zinc deficiency may also occur as a

result of liver or kidney disease, gastrointestinal disorders, skin disorders, parasitic infections, diabetes, and genetic disorders, such as sickle cell disease (Prasad 1979). Clinical disorders aggravated by zinc deficiency include ulcerative colitis, chronic renal disease, and hemolytic anemia (Goyer 1986). In the 40 years since human zinc deficiency was demonstrated, it has been observed in a wide variety of geographic areas and economic circumstances. Severe zinc deficiency occurs in some areas of the Middle East and North Africa and is frequently associated with the consumption of unrefined cereals as a major part of the diet (Casey and Hambidge 1980). Chronic zinc deficiency in humans is associated with dwarfism, infantile testes, delayed sexual maturity, birth defects, poor appetite, mental lethargy, immunodeficiency, skin disorders, night blindness, impotence, spleen and liver enlargement, defective mobilization of vitamin A, delayed wound healing, impaired taste acuity, abnormal glucose tolerance, impaired secretion of luteinizing hormone, and iron and folate deficiency (Prasad 1979, 1980; Casey and Hambidge 1980; Elinder 1986; Goyer 1986; Sternlieb 1988; Mackay-Sim and Dreosti 1989). A deficiency of zinc in the growing age period results in growth retardation; a severe zinc deficiency may be fatal if untreated (Prasad 1980). Zinc-deficient humans excrete <100 µg zinc daily in urine rather than a normal daily >300 µg zinc (Goyer 1986). Zinc deficiency may exacerbate impaired copper nutrition; interactions with cadmium and lead may modify the toxicity of these metals (Goyer 1986). Acrodermatitis enterohepatica is a disease characterized by skin eruptions, gastrointestinal disorders, and low serum zinc levels. One causative factor is poor intestinal absorption of zinc; a complete cure was accomplished by oral administration of 135 mg zinc daily as 600 mg zinc sulfate (Elinder 1986). Using radiozinc-65, it was shown that afflicted individuals had a greater turnover of plasma zinc, a smaller pool of exchangeable zinc, and a reduced excretion of zinc in stool and urine (Prasad 1979). Zinc deficiency in humans is usually treated by oral administration of 1 mg Zn/kg BW daily (Casey and Hambidge 1980). However, zinc-deficient humans given daily intravenous injections of 23 mg zinc experienced profuse sweating blurred vision, and hypothermia (Saxena et al. 1989b). An endemic zinc deficiency syndrome among young men has been reported from Iran and Egypt and is characterized by retarded growth, infantile testes, delayed sexual maturation, mental lethargy, anemia, reduced concentration of zinc in plasma and red cells, enlarged liver and spleen, and hyperpigmentation; oral supplementation of 30 mg zinc daily had a prompt beneficial effect (Prasad 1979; Elder 1986). A zinc deficiency syndrome during human pregnancy includes increased maternal morbidity, abnormal taste sensations, prolonged gestation, inefficient labor, atonic bleeding, and increased risks to the fetus (Jameson 1980). Pregnant women with initially low and subsequently decreasing serum zinc levels had a high frequency of complications at delivery, including congenital malformations in infants. (Jameson 1980). Multiple severe skeletal abnormalities and organ malformations in human fetuses have been attributed to zinc deficiency (Casey and Hambidge 1980). In newborns, zinc deficiency is manifested by growth retardation, dermatitis, hair loss, impaired healing, susceptibility to infections, and neuropsychologic abnormalities (Casey and Hambidge 1980; Goyer 1986).

Hereditary zinc deficiency occurs in certain strains of cattle (*Bos* spp.) and affects the skin and mucous membranes of the gastrointestinal tract. The disease, Lethal Trait A46, is caused by failure of a single autosomal recessive gene regulating zinc absorption from the intestine. Affected animals die within a few months from secondary bacterial infections unless treated daily with high oral doses of zinc compounds (Bosma et al. 1988). Certain imported breeds of cattle in the western Sudan with low zinc serum levels (i.e., <0.6 mg/L) showed signs of zinc deficiency, including stunted growth, weakness, skin lesions, and loss of hair pigment (Damir et al. 1988). Cows fed a low (25 mg/kg ration) but adequate zinc diet had liver zinc concentrations below the expected 125 mg Zn/kg DW; increasing the total zinc dietary loading to 45 or 50 mg/kg DW is recommended for counteracting reduced zinc absorption in diets with soybean products (Binnerts 1989). Cows and calves fed low zinc diets of 25 mg Zn/kg ration showed a decrease in plasma zinc from 1.02 mg/L at start to 0.66 mg/L at day 90; cows fed 65 mg Zn/kg diet had a significantly elevated (1.5 mg Zn/L) plasma zinc level and increased blood urea and plasma proteins (Ramachandra and Prasad 1989). Biomarkers to identify zinc deficiency in bovines include zinc concentrations in plasma, unsaturated zinc-binding capacity, ratio of copper to zinc in plasma, and zinc concentrations in other blood factors; indirect biomarkers include enzyme activities, red cell uptake, and metallothionein content in the plasma and liver (Binnerts 1989).

Domestic goats (*Capra* sp.) fed a zinc-deficient diet (15 mg Zn/kg developed skin histopathology and alopecia (hair loss) after 177 days; zinc-deficient diets lacking vitamin A hastened the process, and signs were evident between 46 and 68 days (Chhabra and Arora 1989). No signs were evident in goats fed vitamin A-adequate diets containing 80 mg Zn/kg ration (Chhabra and Arora 1989).

Guinea pigs (*Cavia* spp.) fed a zinc-deficient diet (1.25 mg Zn/kg FW) for 60 days had significant reductions in zinc concentration in the serum (0.5 mg/L), kidney (10 mg/kg FW), testes (9.5 mg/kg FW), and liver (9.4 mg/kg FW). Guinea pigs fed 1.25 mg Zn/kg FW diet for 45 days followed by a zinc-replete diet of 100 mg/kg FW for 15 days had normal concentrations of zinc in serum (1.6-2.0 mg/kg FW), kidney (18-20 mg/kg FW), testes (19-27 mg/kg FW), and liver (15-17 mg/kg FW; Gupta et al. 1988). Zinc-deficient guinea pigs (<3 mg Zn/kg diet, 1 mg Zn/L drinking water), but not zinc-adequate animals (<3 mg Zn/kg diet, 15 mg Zn/L), exposed from day 30 of gestation to term on day 68 produced young with a low birth weight and severe skin lesions, were sensitive to handing and slow in recovering balance when turned on side, and had a peculiar stance; fetal zinc concentrations were depressed 15-33% in the liver and placenta (Apgar and Everett 1988). Disrupted immunocompetence responses and disordered protein metabolism were found in guinea pigs fed a zinc-deficient diet of 1.25 mg/kg FW ration for 45 days; marked, although incomplete, restoration occurred when this group was switched to 100 mg Zn/kg ration for 15 days (Verma et al. 1988). Neuromuscular pathology was evident in weanling guinea pigs fed a zinc-deficient diet (<1 mg Zn/kg) for 4 weeks, as judged by abnormal posture, skin lesions, and disrupted vocalizations; signs became severe after 5-6 weeks, but a single intraperitoneal injection of 1.3 mg Zn/kg BW (as $ZnSO_4$) caused remission within 7 days (O'Dell et al. 1989). Acute experimental allergic encephalomyelitis was induced in guinea pigs maintained on low (6 mg/kg), normal (20 mg/kg), and high (200 mg/kg) levels of zinc in the diet. Acute experimental allergic encephalomyelitis is usually a fatal disease of the central nervous system induced by inoculation with protein found in myelin of the central nervous system. Those on the zinc-deficient diet exhibited the expected signs of zinc deficiency but, unlike other groups, did not develop neurological signs of acute experimental allergic encephalomyelitis (Scelsi et al. 1989). Experimental allergic encephalomyelitis suppression in the zinc-deficient guinea pigs is ascribed to the influence of zinc deficiency of the T-cell function. A model of autoimmune central nervous system disease such as experimental allergic encephalomyelitis that requires a prominent T-lymphocyte sensitization can be altered or suppressed when the immunoregulatory mechanisms are impaired by zinc deficiency (Scelsi et al. 1989).

Unlike conspecifics on diets containing 100 mg Zn/kg, rhesus monkeys (*Macaca mulatta*) fed a marginally deficient zinc diet (4 mg Zn/kg diet) between age 5.5 and 30.0 months had lower plasma zinc levels, delayed onset of accelerated weight gain and linear growth, and no loss of subcutaneous fat--typical of early adolescence (Golub et al. 1988). Marginal dietary zinc deprivation also depressed immune function in rhesus monkeys by about 30% and impaired both learning and reversal of a visual discrimination task by 33-66% (Golub et al. 1988). When pregnant rhesus monkeys are fed a diet marginally deficient in zinc (4 mg/kg), perturbations in the mother's immune system can occur. Their infants, but not controls (100 mg Zn/kg diet), had reduced immune responsiveness despite the absence of marked differences in plasma or soft tissue zinc concentrations (Keen et al. 1989). Infant rhesus monkeys from zinc-deprived (4 mg Zn/kg ration) pregnant dams and subsequently fed the same low zinc diet showed delayed skeletal maturation during their first year. The condition was most severe at age 6 months but began to return to normal despite continuation of the marginally zinc-deficient diet (Leek et al. 1988).

Mice (*Mus* sp.) fed a zinc-deficient diet of 0.7 mg Zn/kg ration for 40 days, unlike mice fed a zinc-adequate diet of 36.5 mg Zn/kg, had a reduced growth rate, impaired phagocytic function, increased susceptibility to lead poisoning, and reduced zinc content in the blood (0.7 mg/L vs. 1.0-1.1 mg/L) and liver (12 mg Zn/kg FW vs. 17-19 mg Zn/kg FW; Tone et al. 1988). Zinc deficiency during early development affects neural tube development through arrested cell growth (Mackay-Sim and Dreosti 1989). Zinc deficiency in mice may disrupt olfactory function through interference with zinc-containing neurons in higher olfactory centers. Adult mice fed a zinc-deficient diet of 5 mg Zn/kg ration for 42 days, unlike mice given 100 mg Zn/kg diet, could not distinguish odors, although olfactory epithelia seemed normal (Mackay-Sim and Dreosti 1989).

Mink (*Mustela vison*) kits fed a zinc-deficient diet of 4.1 mg Zn/kg FW ration for 4 days retained 0.49 mg Zn/kit and lost weight. Kits fed a zinc-adequate diet (35-45 mg Zn/kg FW, 100-150 mg/kg DW) retained 2.5 mg Zn/kit, and those fed 83 mg Zn/kg FW diet retained 7.8 mg Zn/kit. Kits on low doses ate less than other groups. The most important excretory route was urine in the zinc-deficient group and feces in higher dose groups (Mejborn 1989).

Domestic sheep (*Ovis aries*) fed a low zinc diet (2.2 mg Zn/kg DW diet) for 50 days, unlike sheep fed a zinc-adequate diet (33 mg Zn/kg DW diet), excreted less zinc (<4 mg daily vs. 23-25 mg), consumed less food (409 g daily vs. 898 g), and had lower plasma zinc concentrations (0.18 mg/L vs. 0.53-0.58 mg/L); a reduction in

plasma alkaline phosphatase activity and an increase in plasma zinc binding capacity were also noted (Khandaker and Telfer 1990). Sensitive indicators of zinc deficiency in lambs include significant reductions in plasma alkaline phosphatase activity and plasma zinc concentrations; signs were clearly evident in lambs fed 10.8 mg Zn/kg DW diet for 50 to 180 days (Vergnes et al. 1990). A normal diet for lambs contains 124-130 mg Zn/kg DW ration and 33 for adults (Vergnes et al. 1990). One recommended treatment for zinc-deficient sheep is ruminal insertion of zinc-containing boluses every 40 days; bolus zinc release is about 107 mg daily (Khandaker and Telfer 1990).

Zinc-deficient pregnant laboratory white rats (*Rattus* sp.) have reduced litter size, a high frequency of fetal deformities, low birth weight, and a prolonged parturition; dams are inactive and seem indifferent toward young (Harland et al. 1975). Fetal skeletal defects are prominent in rats fed zinc-deficient diets of 10 mg/kg ration during a 21-day gestation period. About 91% of zinc-deficient fetuses had multiple skeletal malformations, but controls fed 76 mg Zn/kg diet had none (Ferreira et al. 1989). Zinc-deficient (1.5 mg Zn/kg diet) pregnant rats also had increased iron levels in the liver, kidney, and spleen; depleted liver glycogen: and reduced levels of zinc in the pancreas and duodenum (Mamba et al. 1989). Zinc deficiency causes testicular atrophy and hypogonadism in rats; the effects include spermatic arrest, histopathology of seminiferous tubules and interstitial cells, reduced serum testicular testosterone levels, and reduced testicular zinc concentrations (Hafiez et al. 1990). Zinc is required in Leydig cells for normal testosterone activity. Calcitonin inhibits transmembrane influx of zinc in the isolated rat Leydig cell, but these effects usually take >2 days and are critical only in states of borderline zinc deficiency (Chausmer et al. 1989). Zinc deficiency during pubertal development of rats depresses the activity of dipeptidyl carboxypeptidase in the testes and epididymis; this enzyme is required for maturation and development of sperm cells and reduced activity may cause suppression of sexual maturity (Reeves 1990). Laboratory white rats fed zinc-deficient diets for 20 days show an aversion to the zinc-deficient diet. They readily consumed a familiar zinc-adequate diet for 15 days, but the previously deficient animals continued to avoid zinc-deficient diets when given a choice (Cannon et al. 1988). Zinc deficiency in rats (<1 mg Zn/kg diet for 26 days) significantly reduced blood pressure and this correlated positively with serum angiotensin converting enzyme activity; increasing the dietary intake of calcium had no effect on these responses (Reeves and O'Dell 1988). During zinc deficiency, zinc is mobilized from bone in young immature animals and may be available for metabolic processes including growth (Calhoun et al. 1978). Diabetic rats are at risk of developing zinc deficiency because of zinc's role in modulating immune system dysfunction in diabetes mellitus (Mooradian et al. 1988). Cadmium toxicity is related to the zinc status of the body. Zinc-deficient rats (<1 mg Zn/kg diet) and zinc-adequate rats (40 mg/kg) were both challenged with cadmium. The zinc-deficient group had accelerated zinc loss from the kidneys; enlarged liver, kidneys, spleen and lungs; and increased distribution of cadmium in tissues (Sato and Nagai 1989). Other signs in zinc-deficient laboratory white rats included decreased food intake and loss of body weight (Vallee 1959; Cannon et al. 1988; Reeves and O'Dell 1988; Dib et al. 1989; Ferreira et al. 1989; Mamba et al. 1989; Mansour et al. 1989; Sato and Nagai 1989); reduced serum zinc (Calhoun et al. 1978; Reeves and O'Dell 1988); altered cholesterol metabolism (Samman and Roberts 1988); increased serum magnesium (Reeves and O'Dell 1988); lowered bone (femur) zinc concentrations (Calhoun et al. 1978); degenerated olfactory epithelium (Mackay-Sim and Dreosti 1989); reduced serum total proteins (Mansour et al. 1989); decreased activity of glutamate, glycine, methionine, arginine, lysine, and proline (Bettger 1989); and increased dental caries (Goldberg et al. 1990).

Zinc deficiency in domestic pigs (*Sus* sp.) is associated with a condition known as porcine parakeratosis, characterized by dermatitis, diarrhea, vomiting, anorexia, severe weight loss, and eventually death; the condition is exacerbated by high calcium levels (Vallee 1959).

Lethal and Sublethal Effects

General

Significant adverse effects on growth, reproduction, and survival are documented for sensitive marine and freshwater species of aquatic plants, invertebrates, and vertebrates at nominal water concentrations between 10 and 25 µg Zn/L. Sensitive terrestrial plants died when soil zinc concentrations were >100 mg/kg and showed decreased photosynthesis when total plant contained >178 mg Zn/kg DW. Representative soil invertebrates showed reduced growth at 300-1,000 mg Zn/kg diet and reduced survival at 470-6,400 mg Zn/kg soil. Domestic poultry and avian wildlife had reduced growth at >2,000 mg Zn/kg diet, and reduced survival at >3,000 mg Zn/kg diet or at a single oral dose >742 mg Zn/kg BW; younger stages (i.e., chicks, ducklings) were least resistant.

Sensitive species of livestock and small laboratory animals were adversely affected at >0.8 mg Zn/m^3 air, 90-300 mg Zn/kg diet, >90 mg Zn/kg BW daily, >300 mg Zn/L drinking water, and >350 mg Zn/kg BW single oral dose.

Terrestrial Plants and Invertebrates

Sensitive terrestrial plants die when soil zinc levels exceed 100 mg/kg or when plant zinc content exceeds 178 mg/kg DW (Table 5). The phytotoxic zinc level for barley (*Hordeum vulgare*) is not known, but zinc content of barley leaf rarely exceeds 100 mg/kg DW (Chang et al. 1983). Uptake of zinc from soils by plants is dependent on soil type; for example, uptake is lower in coarse loamy soils than in fine loamy soils (Chang et al. 1983). Zinc uptake by barley leaf is greater with increasing rate of sludge application, but the relation is not proportional (Table 5).

Among terrestrial invertebrates, adverse effects on earthworm survival were documented at 470-662 mg/kg soil, slugs had reduced food consumption at 300 mg Zn/kg diet and reduced growth at 1,000 mg Zn/kg diet, and woodlice had impaired reproduction at 1,600 mg Zn/kg soil and reduced survival at 5,000 mg Zn/kg diet or 6,400 mg Zn/kg soil (Table 5).

High zinc concentrations in soils are responsible for reductions in populations of soil invertebrates near brass mills and zinc smelters (Beyer 1990). Soils in the vicinity of zinc smelters contained up to 35 g Zn/kg and had decreased populations of arthropods; experimentally, 20 g of total zinc per kilogram of soil could account for the decreased survival (Beyer et al. 1984). Zinc concentrations exceeding 1,600 mg/kg soil litter are associated with reduced natural populations of decomposer organisms in contaminated forest soil litter, and this has been verified experimentally (Beyer and Anderson 1985). Poisoning of decomposer organisms, such as the woodlouse (*Porcellio scaber*), may disrupt nutrient cycling and reduce the number of invertebrates available as wildlife food (Beyer and Anderson 1985). The woodlouse contains higher concentrations of zinc than other terrestrial invertebrates: up to 152 mg Zn/kg DW whole organism (Hopkin and Martin 1985). It is speculated that the large zinc stores in *P. scaber* repels predators that find zinc distasteful (Hopkin and Martin 1985).

Table 5. Effects of zinc on representative terrestrial plants and invertebrates.

Organism, dose, and other variables	Effect	Reference[a]
Plants		
Fir, *Abies pindrow,* wooden stakes coated with 10% zinc oxide	Protects wood against termite damage for 5 years compared with 4 years for copper sulfate, 2 years for calcium carbonate, and <6 months for untreated wood	1
Red maple, *Acer rubrum*, 100 mg Zn/kg culture medium	Lethal to seedlings	2
Lichen, *Cladonia uncialis*, whole plant zinc content	Depressed photosynthesis when whole lichen burden is >178 mg Zn/kg DW; decreased respiration at >3,550 mg Zn/kg DW	3
Barley, *Hordeum vulgare*, leaf, from soil treated with sludge for 3 years		
No sludge	21-25 mg/kg DW	4
80 kg Zn/ha/year	26-47 mg/kg DW	4
160 kg Zn/ha/year	29-56 mg/kg DW	4
320 kg Zn/ha/year	41-57 mg/kg DW	4

Lichen, *Lasallia papulosa*, whole plant zinc content	Significant depression in photosynthesis at >308 mg Zn/kg DW and in respiration at >3,300 mg Zn/kg DW	3
Oak, *Quercus rubra*, culture medium contained 100 mg Zn/kg	Lethal to seedlings	2
Corn, *Zea mays*, grown on sludge amended loam plots; soil contains a maximum of 460 mg Zn/kg DW	Leaf contains a maximum of 293 mg Zn/kg DW (60 mg Zn/kg for controls); grain contains a maximum of 65 mg Zn/kg DW (32 mg Zn/kg for controls)	5
Invertebrates		
Earthworm, *Aporrectodea tuberculata*; concentrations of zinc in soil ranged from 28 mg/kg DW to 470 mg/kg DW versus concentrations in whole worms (less gut contents)	At soil zinc concentration of 28 mg/kg DW (control), worms contained 320 mg Zn/kg DW. At soil zinc levels of 97, 110, 190, and 320 mg/kg DW, whole worms contained 810, 1,300, 1,100, and 650 mg Zn/kg DW, respectively. No worms were found at soil zinc levels of 470 mg/kg DW	6
Slug, *Arion ater*, fed diets containing 10, 25, 50, 100, 300, or 1,000 mg Zn/kg ration for 27 days	No deaths in any group. Significantly reduced food consumption in 300 and 1,000 mg/kg diets. All groups weighed less than controls at day 27, but growth was statistically impaired only in the 1,000 mg/kg group	7
Slug, *Arion ater*, fed diets containing up to 1,000 mg/kg feed for 30 days	No adverse effects except for glycogen depression at 1,000 mg/kg diet	8,9
Spider, *Dysdera crocata*, fed woodlice (*Porcellio scaber*) at rate of one every 3 days for 36 days		
Woodlice from uncontaminated site (87 mg Zn/kg DW whole organism)	Whole spider contains 182 mg Zn/kg DW	10
Woodlice from contaminated site (152 mg Zn/kg DW whole organism)	Whole spider contains 118 mg Zn/kg DW (116 mg Zn/kg DW in starved spiders)	10
Earthworm, *Eisenia foetida*		
10-12 µg Zn/cm^2 applied to epidermis	LC50 (48 h)	11
662 mg Zn/kg artificial soil (95% C.I. 574-674)	LC50 (2 weeks)	11
Woodlice, *Porcellio scaber*, fed soil litter containing up to 12,800 mg Zn/kg for 64 weeks	Soil litter containing ≥1,600 mg Zn/kg had adverse effects on reproduction; adult survival was reduced at >6,400 mg Zn/kg litter	12

Woodlice, *Porcellio scaber*, fed diets containing up to 20,000 mg Zn/kg feed for 8 weeks		Decreased survival at ≥5,000 mg/kg	13

[a] 1. Roomi et al. 1990; 2. Buchauer 1971; 3. Nash 1975; 4. Chang et al. 1983; 5. Hinesly et al. 1977; 6. Beyer et al. 1987; 7. Marigomez et al. 1986; 8. Recio et al. 1988a; 9. Recio et al. 1988b; 10. Hopkin and Martin 1985; 11. Neuhauser et al. 1985; 12. Beyer and Anderson 1985; 13. Beyer et al. 1984.

Slugs (*Arion ater*) are resistant to high dietary zinc intakes (1,000 mg/kg feed) for 30 days, although zinc accumulations occur in excretory and calcium cells of the digestive gland (Recio et al. 1988a, 1988b). Histochemical detection of zinc in digestive glands of *Arion* is an indication of high levels of zinc in the environment (Recio et al. 1988a). Zinc elimination in *Arion* occurs directly from lipofuscin material of excretory cells and from spherules of calcium cells; excretion of lipofuscin material through feces is the major excretory route (Recio et al. 1988a).

Zinc normally aids wound healing in terrestrial invertebrates. Wounding of the optic tentacle, foot tissue, and partial shell removal in *Helix aspersa*, a terrestrial gastropod, resulted in deposition of zinc in the wound area after 2 to 5 days. Increased zinc in *Helix* wound areas may be necessary to promote protein synthesis, collagen, formation, and mitotic cell division (Ireland 1986).

Aquatic Organisms

Significant adverse effects of zinc on growth, survival, and reproduction occur in representative sensitive species of aquatic plants, protozoans, sponges, molluscs, crustaceans, echinoderms, fish, and amphibians at nominal water concentrations between 10 and 25 µg Zn/L (Table 6).

Table 6. Effects of zinc on representative aquatic plants and animals. Concentrations are in micrograms of zinc per liter of medium.

Taxonomic group, organism, and other variables	Concentration (ppb)	Effects	Reference[a]
Plants			
Alga, *Amphidinium carteri*	400	Growth inhibition	1
Aquatic plants, various	30->200,000	Adverse effects	2
Brown alga, *Ascophyllum nodosum*	100	No effect on growth in 10 days	2
Ascophyllum nodosum	250	Decreased growth in 10 days	2
Coccolithophorid, *Cricosphaera carterae*	77	Growth reduced 50% in 4 days	2
Freshwater algae, 11 species	140-800	Growth inhibition	3
Freshwater algae, most species	>1,000	Growth inhibition	1
Brown macroalgae, *Fucus serratus*	9.5	Bioconcentration Factor (BCF) of x10,770 in 140 days	2
Fucus serratus	8.8	Altered lipid metabolism	2
Marine macroalgae, *Fucus vesiculosus*	3,500	No adverse effects	2
Fucus *vesiculosus*	7,000	Growth retardation	2
Dinoflagellate, *Glenodinium halli*	20	Chlorophyll reduced 65% in 2 days	2
Dinoflagellate, *Gymnodinium*	110-392	Chlorophyll reduced about 65%	2

splendens		in 2 days in temperature range 16-30° C	
Gymnodinium *splendens*	100	Growth inhibition in 38 days	3
Alga, *Isochrysis galbana*	74	Chlorophyll reduced 65% in 48 h at 16 ppt salinity, 20° C	2
I. galbana	430	Chlorophyll reduced 65% in 48 h at 16° C, 16 ppt salinity	2
Kelp, *Laminaria digitata*	100	Growth inhibition in 24 days	1
Brown macroalga, *Laminaria hyperborea*	250 8-10 days	Reduced growth of sperophytes in	2
Marine algae, 4 species	50-500	Decrease in cell numbers	1
Marine algae, 5 species	100	Growth inhibited in 48 h	2
Marine macroalgae, 4 species	100	No adverse effects	2
Marine macroalgae, 4 species	1,400	Growth reduction	2
Diatom, *Nitzschia closterium*	271-300	50% growth inhibition in 4 days	2
Diatom, *Nitzschia longissima*	100	Growth stimulated during exposure for 1-5 days	2
Dinoflagellate, *Procentrum micans*	319	50% growth inhibition in 4 days	2
Diatom, *Phaeodactylum tricornutum*	250	BCF of x1,800 in 3 days	2
P. tricornutum	4,800	6.7% increase in growth during 12-day exposure	2
Phytoplankton	15	Primary productivity reduced in 14 days	2
Marine alga, *Rhizosolenia* sp.	15-25	Photosynthesis reduction	3
Alga, *Scenedesmus quadricauda*	2	Adverse effects	4
S. quadricauda	64	Growth inhibition in 14 days	2
S. quadricauda	300	Lethal	4
Diatom, *Schroederella schroederi*	19	Growth inhibited 50% in 48-96 h	2
Freshwater alga, *Selenastrum capricornutum*	30	Some growth inhibition in 7 days	1
S. cupricornutum	40-68	95% growth inhibition in 14 days	1
S. capricornutum	100	100% growth inhibition in 7 days	1
Diatom, *Skeletonema costatum*	19.6	Adverse effects	4
S. costatum	50-100	Growth reduced 20-23% in 10-15 days	2
S. costatum	200	Growth stimulated in 1-5 days	2
S. costatum	265	Metabolic disruption in 3 days	2
Diatom, *Thalassiosira pseudonana*	65	Adverse effects	4
T. pseudonana	500	Growth reduced 41% in 11-15 days	2
T. pseudonana	823	Growth reduced 50% in 72 h	2
Green macroalga, *Ulva lactuca*	65	BCF of x255 in 6 days	2
Protists			
Protozoan, *Cristigera* sp.	50-125	Growth reduced in 5-h exposure	1,2
Bacterium, *Escherichia coli*	650-1,400	Growth inhibition	3
Microorganisms, various	650-1,100	Growth inhibition, usually	3

Paramecium, *Paramecium multi-micronucleatum*	560-10,000	LC50 (3 h)	3
Bacterium, *Pseudomonas* sp.	1,000-10,000	Growth inhibition	3
Protozoan, *Vorticella convallaria*	50	LC50 (48 h)	3
Porifera			
Freshwater sponge, *Ephydatia fluviatilis*			
Adults	6.5	No effect on growth; no tolerance developed with long-term exposure	5
Adults	26	After exposure for 10 days, tissue deterioration and death during 3-week postexposure period	5
Rotifers			
Rotifer, *Philodena acutiformis*			
Adults	500	LC50 (48 h), 25° C	
Adults	1,550	LC50 (48 h), 5° C	2
Molluscs			
Freshwater snail, *Ancylus fluviatilis*			
Juvenile	80	LC50 (100 days), shell length <2 mm	6
Adult	100	No adverse effect on reproduction in 100 days	6
Juvenile	130	LC50 (100 days), shell length >3 mm	6
Adult	180	Reproduction reduced in 100 days	6
Bay scallop, *Argopecten irradians*			
Larvae	50	Growth rate reduced 22% in 9 days	7
Larvae	109	Growth reduced 50% in 9 days	7
Larvae	120	LC50 (9 days), increased shell deformities	7
Larvae	150-200	All dead at metamorphosis	7
Juvenile	2,250	LC50 (96 h)	8
Freshwater snail, *Biomphalaria glabrata*	500	By day 33 of exposure, embryo survival was reduced 50% and adult growth and reproduction inhibited	9
Asiatic clam, *Corbicula fluminea*	<20	Residues were 169 mg/kg dry weight (DW) parts after feeding on periphyton containing 393-1,327 mg/kg DW for 30 days	11
C. fluminea	25	Normal growth during exposure for 30 days	10
C. fluminea	34	Residues were 433 mg/kg DW soil parts in 30 days after feeding on periphyton containing 956-4,369 mg Zn/kg DW;	11

		growth reduced; cellulase enzyme activity reduced	
C. fluminea	50-500	Growth inhibited between days 20 and 30 of exposure	10
C. fluminea	218	BCF of x126 in 28 days	2
C. fluminea	1,000	After exposure for 30 days, about 30% died. Survivors had osmoregulatory impairment and residues of 2,000 mg Zn/kg DW soft parts (200 mg Zn/kg DW soft parts in controls). Depuration complete by day 17 postexposure, and growth rate returns to normal	10
Pacific oyster, *Crassostrea gigas*			
Larvae	10-20	Reduced larval settlement in 20 days	1
Larvae	30-35	Reduced larval settlement in 6 days	2
Larvae	50	Normal growth and development in 5 days	12
Larvae	70	Abnormal shell development in 48 h	1
Larvae	75	No deaths in 48 h	1
Larvae	80-95	Growth reduced 50% in 4 days	2
Larvae	119-310	LC50 (48 h)	1,13
Larvae	125	Substrate attachment inhibited in 5 days	1,2
Larvae	200	No growth in 5 days	12
Embryo	233	LC50 (96 h)	2
Larvae	250	Increasing incidence of abnormal development and mortality	12
Sperm	444	Fertilization success reduced 50% in 60 min	2
Larvae	500	All died in 48 h	1
American oyster, *Crassostrea virginica*			
Adult	100	Whole body concentration of 2,560-2,708 mg Zn/kg fresh weight (FW) soil parts after 20-week exposure (1,036-1,708 mg Zn/kg FW soil parts in controls)	14
Adult	200	After exposure for 20 weeks, residues were 3,185-3,813 mg Zn/kg FW soft parts	14
Embryo	230	LC50 (96 h)	2
Larvae	340	LC50 (48 h)	13
Red abalone, *Haliotis rufescens*			

Larvae	19	No adverse affects after 9-day exposure	13
Larvae	41	Normal development during 48-h	13
Larvae	50	50% abnormal development during exposure for 9 days	13
Larvae	68	50% abnormal development in 48-h exposure	13
Marine gastropod, *Littorina littorea*			
Adult	0.2 (controls)	Zinc concentrations in all tissues were <185 mg/kg DW, except kidney, which was 372 mg/kg DW	15
Adult	10	After exposure for 42 days, tissue zinc residues were: head-foot 120 mg/kg DW, gills 255 mg/kg DW, whole soft parts 605 mg/kg DW, viscera 1,322 mg/kg DW, stomach 1,918 mg/kg DW, and kidney 2,153 mg/kg DW	15
Freshwater pond snail, *Lymnaea luteola*, adult	1,680	LC50 (96h)	85
Hard shell clam, *Mercenaria mercenaria*			
Larvae	50	5% died in 12 days	1
Larvae	168	50% dead or abnormal in 48 h	1
Embryo	195	LC50 (96 h)	2
Larvae	195-341	LC50 (10-12 days)	1
Larvae	279	All died in 48 h	1
Softshell clam, *Mya arenaria*			
Adult	10	Soft parts contained 9.5 mg Zn/kg FW after 16 weeks at 0-10° C, and 11 mg/kg after 2 weeks at 16-22° C	14
Adult	200	BCF of x85-135 in 50 days	2
Adult	500	Soft parts contained 31-48 mg Zn/kg FW after exposure for 6-16 weeks at 0-10° C, and 59-82 mg/kg after 1-2 weeks exposure at 16-22° C	14
Adult	900	No deaths in 7 days at 22° C	16
Adult	1,550	LC50 (7 days) at 22° C	16
Adult	25,000	All dead after 70-day exposure at 0-10° C; at exposure temperature of 16-22° C, all dead by day 14	17
Common mussel, *Mytilus edulis*			
Adult	25	Maximum kidney zinc residue after 18 days was 14.1 g/kg DW (4.9 g/kg in controls)	18

Adult	60	Shell growth rate reduced 50% in 2-6 days of exposure	2
Embryo	96-314	Development inhibited 50% in 72 h	2
Adult	100	No accumulations in tissues after 4-day exposure	19
Adult	230-860	In 7-h exposure, pumping rate decreased with increasing zinc, and was completely stopped at >470 µg/L; recovery on return to background levels	20
Adult	1,000	After exposure for 24 h, zinc concentration in soil parts rose from 150 mg/kg DW to 252 mg/kg DW and remained elevated for at least 6 weeks postexposure	21
Larvae	1,752	LC50 (48 h)	13
Adult	1,800	Reduced byssal thread production	2
Adult	5,000	LC50 (7 days)	2
Adult	5,000	LC100 (16 days)	19
Adult	20,800	LC50 (24-h exposure plus 6 weeks postexposure); none dead during exposure	22
Sperm	65,400	Respiration inhibited 50% in 20 min	23
Mud snail, *Nassarius obsoletus*			
Adult	200	Decreased oxygen consumption in 72 h	1
Egg	650	Abnormal veliger development	24
Adult	5,000	No deaths in 168 h	25
Green-lipped mussel, *Perna virdis*			
Adult	<178-362	Maintains constant body concentration over 21-day exposure period	26
Adult	>362	Accumulation in tissue	26
Adult	6,090	LC50 (96 h)	26
Freshwater snail, *Physa heterostropha*, juvenile	241	LC50 (96 h)	2
Surf clam, *Spisula solidissima*, juvenile	2,950	LC50 (96 h)	8
Bryozoans			
Bryozoan, *Bugula neritina*, larvae	200	LC50 (5 h)	3
Bryozoan, *Watersipora cucullata*, larvae	650	LC50 (5 h)	3
Crustaceans			

Copepod, *Acartia tonsa*	290	50% immobilized in 48 h	
A. tonsa	294	LC50 (96 h)	2
Amphipod, *Allorchestes compressa*	580-2,000	LC50 (96 h)	3,27
Brine shrimp, *Artemia* sp.	14-1,360	Egg hatching significantly reduced in dose-dependent manner; no effect on survival of prenauplii larvae	28
Cladoceran, *Ceriodaphnia reticulata*	51	LC50 (96 h)	2
Hermit crab, *Clibanarius olivaceous*			
Larvae	1-90	Molting delayed in dose-dependent manner	29
Larvae	100	LC50 (96 h)	29
Larvae	125	LC100 (96 h)	29
Daphnid, *Daphnia galeata mendotae*	15	BCF of x9,400 in 2 weeks	2
D. g. mendotae	30	BCF of x5,833 in 2 weeks	2
D. g. mendotae	60	BCF of x6,333 in 2 weeks	2
Daphnid, *Daphnia magna*	5-14	LC50 (72 h) at 30° C	2
D. magna	25	No effect in soft water (50 mg $CaCO_3$/L) in 50 days	30
D. magna	42-52	MATC [b]; water contains 104-211 mg $CaCO_3$/L	1,2
D. magna	68-655	LC50 (96 h)	31
D. magna	70	Reproduction reduced 16% in 21 days	2
D. magna	100	LC50 (48 h), starved	84
D. magna	250	Nonlethal in 6 weeks when sediments present in test container. Final sediment value of 13,400 mg/kg DW (600 mg/kg DW in controls). Organisms had whole body residues of 450 mg/kg DW	32
	"		
D. magna	280	LC50 (48 h), fed	3
D. magna	560	LC50 (24 h) at 25° C	84
D. magna	560	50% immobilized in 48 h	33
D. magna	2,300	LC50 (24 h) at 5° C	3
Daphnid, *Daphnia pulex*	253	LC50 (96 h)	2
D. pulex	280	LC50 (48 h)	
D. pulex	500	LC50 (24 h) at 25° C	3
D. pulex	1,550	LC50 (24 h) at 5° C	3
Copepod, *Eudiaptomus padanus*	500	LC50 (48 h)	
Amphipod, *Gammarus duebeni*			
Natural population	>100	Survival reduced in 7 days	34
Natural population	1,000	All dead in 7 days at 10 ppt	

		salinity, 84% dead at 30 ppt	34
Zinc-tolerant population	1,000	50% dead in 14 days at 10 ppt salinity, 33% dead at 30 ppt	34
American Lobster, *Homarus americanus*			
Larvae	130	LC50 (17 days)	3
Larvae	381	LC50 (96 h)	2
Adult	13,000	LC50 (11 days)	3
Mysid, *Mysidopis bahia*	120-230	MATC[b]	1,2
M. bahia	499	LC50 (96 h)	2,13
Crayfish, *Orcenectes virilis*	130,000	No deaths in 10 days	35
Hermit crab, *Pagurus longicarpus*			
Adult	200	LC50 (168 h)	25
Adult	400	LC50 (96 h)	1,2
Prawn, *Palaemon elegans*	562	LC67 (21 days)	36
Shrimp, *Pandalus montagui*	65	BCF of x 3.7 in 14 days	2
Mysid, *Praunus flexuosus*	2,000	LC50 (192 h), 5^{o}C, 4.5 ppt salinity	37
Mudcrab, *Rithropanopeus harrisii*, larvae	50	Delayed development in 16-days exposure	1
Copepod, *Tisbe holothuriae*			
Life Cycle	7	No effect on population size after exposure for 4 generations	38
Life Cycle	10	Some deaths in fourth generation	38
Life Cycle	70	All dead by end of first generation	38
Copepodid	421	LC50 (48 h)	39
Adults	620-700	LC50 (48 h)	38,40
Females with egg sacs	713	LC50 (48 h)	39
Copepod, *Tropocyclops praisinus mexicanus*, Quebec lakes, uncontaminated	52-26	LC50 (48 h) in soft water	41
T.P. mexicanus, Quebec lakes, contaminated	2,934	LC50 (48 h) in hard water lake; metal preexposure protective effect hypothesized	41
Aquatic insects			
Mayfly, *Epeorus latifolium*			
Larvae	30	Gradual decrease in growth rate in 4-week exposure; some deaths before emergence	42
Larvae	100-300	Growth inhibited after 2 weeks; all dead before emergence	42
Midge, *Tanytarsus dissimilis*, embryo through third instar	37	LC50 (10 days)	1,2

Annelids			
Polychaete worm, *Capitella capitata*			
Larvae	50-100	Abnormal development during 16 day exposure	2
Adult	1,250	LC50 (28 days)	2
Adult	10,700	LC50 (48 days)	13
Leech, *Erpobdella octoculata*			
Juveniles	60	LC50 (70 days)	43
Adults	100	LC50(70 days)	43
Adults	180	High frequency of abnormal eggs produced in 60-day exposure	43
Adults	320	Inhibited reproduction in 60-day exposure	43
Juveniles	390	LC50 (40 days)	43
Juveniles	2,100	LC50 (96 h)	43
Adults	4,800	LC50 (40 days)	43
Adults	8,800	LC50 (96 h)	43
Polychaete, *Neanthes arenaceodentata*, juveniles	900	LC50 (28 days)	3
Sandworm, *Nereis diversicolor*			
Adults	1,500	No deaths in 168 h	25
Adults	2,600	LC50 (168 h)	25
Adults	10,000	Whole body zinc concentration in survivors after exposure for 34 days was 2,500 mg/kg DW (180 mg/kg DW in controls)	14
Adults	10,000	After 96-h exposure at; 6° C, zinc residues were 1,031 mg/kg DW in head (843 mg/kg DW in controls), 366 mg/kg DW in trunk (158 mg/kg DW in controls), and 455 mg/kg DW in parapodia (275 mg/kg DW in controls); uptake was higher at 12° and 20° C	44
Adults	20,000	No death in 96 h	44
Adults	40,000	LC50 (47 h) for nontolerant strains; LC50 (70 h) for zinc-tolerant strains	45
Worm, *Spirorbis lamellora*, larvae	350	LC50 (3 h)	3
Echinoderms			
Sea urchin, *Anthocidarius crassispina*			
Egg	65	No effect on fertilization membrane formation or development in eggs transferred 1 min after insemination	46
Egg	326	Irreversible inhibition of	

		fertilization membrane formation in eggs transferred 10 s after insemination	46
Starfish, *Asterias rubens*			
Adult females	240	Increased steroid metabolism in pyloric caeca after 21 days	47
Adults	1,000	No deaths in 168 h	25
Adults	2,300	LC50 (168 h)	25
Sand dollar, *Dendraster excentricus*, sperm	28	Fertilization success reduced 50% in 60 min	2
Echinoderms, 3 species, embryos	60-200	Embryonic development inhibited	46
Red sea urchin, *Strongylocentrotus franciscanus*, sperm	313	Fertilization success reduced 50%	2
Purple sea urchin, *Strongylocentrotus purpuratus*, embryos	23	Development inhibited 50% in 5 days	2
Fish			
Longfin dace, *Agosia chrysogaster*,	228	LC50 (96 h)	2
Murrel, *Channa punctatus*, fingerlings, 31-day exposure	12,000	Growth rate reduced by day 19; liver RNA and proteins decreased by day 20; muscle RNA and proteins reduced by day 30	49,50
Texas cichlid, *Cichlasoma cyanoguttatum*, adults, exposure for 4 weeks	40 (control), 65, or 90	Residues were 0.8, 28, and 34 mg Zn/kg FW in muscle; 6, 56, and 25 mg Zn/kg FW in viscera; 6, 59, and 98 mg Zn/kg FW in gills; and 12, 66, and 92 mg Zn/kg FW in bone	51
Air-breathing catfish, *Clarias lazera*, juveniles	26,000-52,000	LC50 (96 h) at 25.1° C (26,000) through 9.3° C (52,000); at 88,000 µg/L and 18.5° C, 50% died and survivors had BCF of x544 in gill, x425 in liver, and x250 in muscle	52
Baltic herring, *Clupea harengus*, eggs exposed from fertiliza-tion through hatching	500, 2,000, 6,000, or 12,000	Histopathology of epidermis and kidney in larvae at >6,000 µg/L; no measurable effects at < 2,000 µg/L	53,54
Atlantic herring, *Clupea harengus harengus*, embryos and larvae	50	Significant increase in incidence of jaw and branchial abnormalities	2
Freshwater fish, 4 species, adults	4,600-17,300	LC50 (5 days)	55
Mummichog, *Fundulus heteroclitus*			
Adults	810	BCF of x16 in whole fish after 56 days	2
Adults	10,000	Zinc concentration in scale, rose from 229 mg/kg DW at start to 746 mg/kg DW	56

		after 45 days and to 1,608 mg/kg DW after 94 days	
Adults	10,000	Zinc content in scale after 45 days exposure and 21 to 49 days in uncontaminated water fell from 746 mg/kg DW to 422-498 mg/kg DW	56
Adults	43,000	No deaths in 8 days; no significant increase in tissue zinc levels	57
Adults	52,000-66,000	LC50 (8 days)	25,57
Adults	71,000-153,000	LC50 (48 h)	58
Mosquitofish, *Gambusia affinis*, adults, muscle	18,000	After 24 h, zinc increased from 82 to 134 µg/kg FW; significant increases in glycogen, total lipids, phospholipids, and cholesterol; decreases in RNA and proteins	59
Flagfish, *Jordanella floridae*			
Life cycle	26-51	MATC [b]	2
Larvae	85	LC80 (30 days)	3
Adults	139	BCF of x417 in whole fish in 100 days	2
Cypriniform freshwater fish, *Labeo rohita*			
Juveniles and adults	20,000	No deaths in 96 h	60
Juveniles	65,000	LC50 (96 h); liver glycogen reduced; BCF of x22 in whole fish	60
Adults	77,000	LC50 (96 h); survivors had disrupted respiration and decreased liver glycogen	60
Spangled perch, *Leiopotherapon unicolor*, adults, exposed for 2 h	5,000, 10,000, or 20,000	Temporary decrease in ventilation rate at 5 mg/L; significant increase in ventilation rate at 10 and 20 mg/L; bradycardia at 20 mg/L	61
Spot, *Leiostomus xanthurus*	38,000	LC50 (96 h)	62
Bluegill, *Lepomis macrochirus*			
Adults	76-235	Reproduction inhibition	3
Adults	100	Hyperactivity	3
Fry	235	Lethal in 3 days	1,2
Adults, exposed for 7 days, then placed in a lethal NaC1 salinity (1.46%) for 60 h	2,350	Exposed fish all dead in 60 h (8 h for controls); plasma chloride declined in zinc-exposed fish, suggesting that zinc reduces permeability of gills to chloride	63
Adults	5,400	LC50 (96 h) at 20 mg $CaCO_3$/L	3
Adults	40,900	LC50 (96 h) at 360 mg $CaCO_3$/L	3

Marine fish, most species	>1,000	LC50 (96 h)	1
Tidewater silverside, *Menidia peninsulae*	5,600	LC50 (96 h)	62
Striped bass, *Morone saxatilis*			
Larvae	100-119	LC50 (96 h)	1,2
Fry	430-1,180	LC50 (96 h)	1,2
Adults	6,700	LC50 (96 h)	1
Stone loach, *Noemacheilus barbatulus*, adults	1,900-2,000	LC50 (25 days)	3,55
N. barbatulus	3,500	LC50 (96 h)	55
Loach, *Noemacheilus* sp.	25,000	LC50 (96 h)	64
Cutthroat trout, *Oncorhynchus clarki*	61-600	LC50 (96 h)	1,65
O. clarki	360	None dead in 14 days	66
O. clarki	670	LC50 (14 days)	3,66
Coho salmon, *Oncorhynchus kisutch*			
Water hardness <50 mg $CaCO_3$/L	280	LC50 (96 h)	3
Juveniles	500-10,700	Decreased white blood cell count in 24h	3
0.5-0.9 g BW	820-1,810	LC50 (96 h)	67
Rainbow trout, *Oncorhynchus mykiss*			
Immatures	5.6	Avoidance, 10- to 20-rain tests	1,2
Larvae and alevins	10	LC54 (28 days)	3
Immatures	47	94% avoidance, 40-rain tests	2
Early life stages	70-140	LC50 (25 days)	3
Juveniles	81	Hyperglycemia in 24 h	3
Fry	90-93	LC50 (96 h)	1,2
Life cycle	140-547	MATC [b]	1,2
Weight 0.6 g	169	LC50 (96 h)	67
Juveniles	210-1,120	Increased blood glucose in 7-63 days	3
Parr	240-830	LC50 (96 h) at 30 mg CaCO3/L	1
Juveniles	310	LC20 (14 days)	66
Immatures	352	Hyperglycemia in 9 days	2
Larvae and alevins	400-2,800	LC50 (120 h)	3
Juveniles	410	LC50 (14 days)	66
Juveniles	430	LC59 (96 h) at 26 mg CaCO3/L	3
Juveniles	520	LC50 (96 h) at 47 mg CaCO3/L	3
Fry	689	LC50 (96 h)	2
Juveniles	690	Increased respiration in 24 h	3
Immatures	1,030	LC50 (96 h) value for group acclimatized to 80 µg Zn/L for 28 days (469 µg Zn/L in nonacclimatized group)	68

Adults	1,120	Reduced growth in 85 days	3
Parr	1,190-4,520	LC50 (96 h) at 350 mg CaCO3/L	1
Juveniles	2,960	LC50 (96 h) at 179 mg CaCO3/L	3
Parr	4,700	LC50 (96 h) at 500 mg CaCO3/L	1
Juveniles	4,800-7,200	LC50 (96 h) at 333-504 mg CaCO3/L	3
Fry	10,000	16°/o dead in 90 h versus none dead in group pretreated with 5 mg Zn/L for 96 h	69
Fry	15,000	79% dead in 90 h versus 20% dead in group pretreated with 5 mg Zn/L for 96 h	69
Sockeye salmon, *Oncorhynchus nerka*			
Embryo through smolt	242	No measurable effects in 18-month exposure	1
Immatures	447	LC50 (115 h)	1
Immatures	750	LC50 (96 h)	3
Chinook salmon, *Oncorhynchus tshawytscha*			
Swim up	97	LC50 (96 h)	1
Chronic exposure	270-510	MATC [b]	1,2
Smolts	446	LC50 (96 h)	2
Minnow, *Phoxinus phoxinus*			
Yearlings	50-130	Reduced growth during exposure for 150 days; no deaths	70
Larvae	60	Decreased swimming ability after exposure for 108 days	3
Larvae	80	LC37 (40 days)	3
Adults	130	Reduced growth during 150-day exposure; some deaths	70
Juveniles	160	Decreased swimming ability after 109 days	3
Adults	200	Decreased swimming ability after 100 days	3
Adults	200	Reduced growth during 30-day exposure; some deaths	70
Adults	250	LC50 (150 days)	3
Fathead minnow, *Pimephales promelas*			
Life cycle	78-145	MATC [b]	1,2
Juveniles	125	Reduced growth in 7 days	2
Larvae	152-294	LC84 (8 weeks)	3
Adults	180	65 to 83% reduction in fecundity in 10-month exposure	1,2
Adults	480	Reduced growth in 30 days	3

Embryo-larvae	500-1,400	50% developmental malformations in 96h	71
Larvae	600	LC50 (96 h)	3
Adults	600	Preexposure for 14 days increased resistance 28% over controls in 96-h zinc toxicity assays	72
Adults	800	LC50 (30 days)	3
Adults	870	LC50 (96 h) at 20 mg $CaCO_3$/L,	3
Adults	1,800	Exposure for 7 days decreased tolerance 63% in 96-h zinc toxicity assays; tolerance decreased 74% after exposure for 14 days	72
Adults	2,800	LC15 (10 months), no eggs deposited	73
Embryo-larvae	3,600	LC50 (6 days)	71
Adults	4,700-6,100	LC50 (96 h) at 50 mg $CaCO_3$/L	3
Adults	6,400-10,900	LC50 (96 h) at 100 mg $CaCO_3$/L	3
Adults	7,100	LC50 (96 h) at 166 mg $CaCO_3$/L	3
Adults	8,200-21,000	LC50 (96 h) at 200 mg $CaCO_3$/L	3
Adults	33,400	LC50 (96 h) at 360 mg $CaCO_3$/L	3
Guppy, *Poecilia reticulata*			
Age 5 days	128	After 134 days, whole body zinc content of 0.6 mg/kg DW (0.3 mg/kg DW in controls); growth reduced	74
Adults	173	Whole body BCF of x466-965 in 30 days	2
Age 5 days	250	Delayed sexual maturation after 134 days	74
Age 5 days	500	Reproduction inhibited	74
Age 5 days	1,350-1,500	LC50 (96 h)	74
Adult males	4,400-5,700	LC50 (96 h)	74
Adult females	5,600-7,300	LC50 (96 h)	74
Atlantic salmon, *Salmo salar*			
Parr	50	50% avoidance in 4 h	2
Parr	100	Avoidance within 20 rain	3
Immatures, Water hardness	100-500	LC50 (21 days)	1
14 mg $CaCO_3$/L	420	LC50 (96 h)	1
20 mg $CaCO_3$/L	600	LC50 (96 h)	1
Brown trout, *Salmo trutta*			
Yolk-sac fry	4.9	40% with noncalcified vertebrae center; all dead in 18 days at pH 4.5 and soft water	75

Yolk-sac fry	9.8-19.6	60% to 75% dead in 20-30 days; 6% to 21% with abnormal vertebrae in pH 4.5 and soft water	75
Yearlings	<140	LC50 (96 h) at pH 8, 10 mg CaCO3/L	76
Adults	570	LC17 (14 days)	66
Adults	640	LC50 (14 days)	66
Yearlings	3,200	LC50 (96 h) at pH 5, 204 mg CaCO3/L	76
Brook trout, *Salvelinus fontinalis*			
Chronic exposure	534-1,360	MATC [b]	1,2
Adults	630	LC17 (14 days)	
Adults	960	LC50 (14 days)	3,66
Cabezon, *Scorpaenichthys marmoratus*, larvae	192	LC50 (96 h)	2
Dogfish, *Scyliorhinus* sp., exposure for 25 days	15,000	No significant accumulations in kidney and muscle, but elevated levels, as judged by BCF values in gill filament (x1.6), spleen (x1.7), pancreas (x2.7), and liver (x5.2)	77,78
Arctic grayling, *Thymallus arcticus*			
BW 0.2-1.8 g	112-168	LC50 (96 h)	67
Fry	315	LC50 (96 h)	67
Alevins	1,580-2,920	LC50 (96 h)	67
Tilapia, *Tilapia sparrmanii*, adults, exposure for 72 h	98,000	Decreased oxygen consumption, mucous precipitation on gills, histopathology of gill epithelium	79
Bolti, *Tilapia zilli*			
Adults	13,000	LC50 (96 h) at 25° C	52
Adults	21,000	LC50 (96 h) at 21° C; residues in survivors were 38,000 mg/kg DW in gill (70 mg/kg DW in controls); 23,000 mg/kg DW in liver (50 mg/kg DW controls); and 2,000 mg/kg DW in muscle, blood, serum, and liver chemistry (10 mg/kg DW in controls)	58,80
Adults	27,000	LC50 (96 h) at 15.3° C	52
Adults	33,000	LC50 (96 h) at 9.3° C	52
Amphibians			
Marbled salamander, *Ambystoma opacum*, embryos	2,380	50% dead or deformed in 8 days	2
Narrow-mouthed toad, *Gastrophryne*	10	50% dead or deformed in 7 days	2

carolinensis, embryos			
Leapfrog; *Rana dalmutina*, larvae, exposed during formation of gonadal structures	9,000	Toxic effect on larval gonad, especially on germ cells of ovarian structure	S1
Newt, *Triturus cristatus*, adults, held in tank with a zinc-plated base	200 to 3,000 over a 7-day period	Zinc-poisoned newts were lethargic, ate poorly, and has skin darkening before death. Zinc residues were elevated in kidney, brain, liver, and intestine, when compared to controls. The hippocampus region of the brain of poisoned newts contained zinc-rich cells	82
South African clawed frog, *Xenopus laevis*			
Embryos	>1,500	At 96 h, some midgut malformations and pericardial edema	83
Embryos	2,700	50% malformations in 96 h	83
Embryos	3,600	50% developmental malformations in 6 days	71
Embryos	>4,000	Severe edema of the pericardium and eye, gut miscoiling, and head and mouth malformations. At high sub-lethal concentrations, severe skeletal kinking, micropthalamia, and microencephaly	83
Tadpoles, pretreated with 5 mg Zn/L for 96 h	15,000-20,000	At 15 mg/L, none died in pretreated group versus 45% dead in controls at 90 h; at 20 mg/L, 15% died in pretreated group versus 50% in untreated controls	69
Embryos	34,500	LC50 (96 h)	71,83

[a]l. EPA 1980; 2. EPA 1987; 3. Spear 1981; 4. Vymazal 1986; 5. Francis and Harrison 1988; 6. Willis 1988; 7. Yantian 1989; 8. Nelson et al. 1988; 9. Munzinger and Guarducci 1988; 10. Belanger et al. 1986; 11. Farris et al. 1989; 12. Brereton et al. 1973; 13. Hunt and Anderson 1989; 14. Eisler 1980; 15. Mason 1988; 16. Eisler 1977a; 17. Eisler 1977b; 18. Lobel and Marshall 1988; 19. Amiard-Triquet et al. 1986; 20. Redpath and Davenport; 1988; 21. Hietanen et al. 1988b; 22. Hietanen et al. 1988a; 23. Akberali et al. 1985; 24. Conrad 1988; 25. Eisler and Hennekey 1977; 26. Chan 1988a; 27. Ahsanullah et al. 1988; 28. Bagshaw et al. 1986; 29. Ajmalkhan et al. 1986; 30. Paulauskis and Winner 1988; 31. Attar and Maly 1982; 32. Memmert 1987; 33. Khangarot and Ray 1989; 34. Johnson and Jones 1989; 35. Mirenda 1986; 36. Nugegoda and Rainbow 1989c; 37. McLusky and Hagerman 1987; 38. Verriopoulos and Hardouvelis 1988; 39. Verriopoulos and Moraitou-Apostolopoulou 1989; 40. Verriopoulos and Dim as 1988; 41. Lalande and Pinel-Alloul 1986; 42. Hatakeyama 1989; 43. Willis 1989; 44. Fernandez and Jones 1989; 45. Grant, et al. 1989; 46. Nakamura et al. 1989; 47. Voogt et al. 1987; 48. Eisler 1981; 49. Shukla and Pandey 1986b; 50. Shukla and Pandey 1986a; 51. Villegas-Navarro and Villarreal-Trevino 1989; 52. Hilmy et al. 1987c; 53. Somasundaram 1985; 54. Somasundaram et

al. 1985; 55. Solbe and Flook 1975; 56. Sauer and Warabe 1989a; 57. Eisler 1967; 58. Burton and Fisher 1990; 59. Taneja et al. 1958; 60. Bengeri and Patil 1986; 61. Gehrke 1988; 62. Mayer 1987; 63. Heath 1987; 64. Pundir 1989; 65. Mayer and Ellersieck 1986; 66. Nehring and Goettl 1974; 67. Buhl and Hamilton 1990; 68. Anadu et al. 1989; 69. Woodall et al. 1988; 70. Bengtsson 1974; 71. Dawson et al. 1988; 72. Hobson and Birge 1989; 73. Brungs 1969; 74. Pierson 1981; 75. Sayer et al. 1989; 76. Everall et al. 1989b; 77. Floe et al. 1979; 78. Crespo et al. 1979; 79. Grobler et al. 1989; 80. Hilmy et al. 1987c; 81. Gipouloux et al. 1986; 82. Taban et al. 1982; 83. Fort et al. 1989; 84.. NAS 1979; 85. Khangarot and Ray 1988.

[b]MATC = maximum acceptable toxicant concentration. Lower value in each MATC pair indicates highest concentration tested producing no measurable effect on growth, survival, reproduction, and metabolism during chronic exposure; higher value indicates lowest concentration tested producing a measurable effect.

Acute LC50 (96 h) values for freshwater invertebrates were between 32 and 40,930 µg Zn/L; in fish, this range was 66 to 40,900 µg/L (EPA 1987). For marine invertebrates the LC50 (96 h) range was 195 µg/L for embryos of the hard-shelled clam (*Mercenaria mercenaria*) to >320 mg/L for adults of the Baltic clam (*Macoma balthica*). For marine telcosts LC50 (96 h) values were between 191 µg/L for larvae of the cabezon (*Scorpaenichthys marmoratus*) to 38 mg/L for juvenile spot, (*Leiostomus xanthurus*; EPA 1987). Many factors are known to modify the biocidal properties of zinc in aquatic environment. In general, zinc was more toxic to embryos and juveniles than to adult, to starved animals, at elevated temperatures, in the presence of cadmium and mercury, in the absence of chelating agent, at reduced salinities, under conditions of marked oscillations in ambient zinc concentrations, at decreased water hardness and alkalinity, and at low dissolved oxygen concentrations (Skidmore 1964; Weatherley et al. 1980; Spear 1981; EPA 1987; Paulauskis and Winner 1988; Table 6).

Bioconcentration factors (BCF) for zinc accumulation from the medium varied widely between and within species of aquatic organisms. For representative freshwater organisms, BCF values ranged from 107 to 1,130 for insects and from 51 to 432 for fish (EPA 1980). In marine environments, the most effective zinc accumulators included red and brown algae, ostreid and crassostreid oysters, and scallops. The ranges of BCF values for representative marine groups were 370 to 64,000 for algae, 85 to 1,500,000 for crustaceans, 15 to 500 for echinoderms, as much as 4 million for scallop kidneys, and 1,900 to 6,900 for fish (Eisler 1980). Significant zinc accumulations were reported after death in algae and fish, suggesting that residue data from these and other organisms found dead on collection are of limited worth (Eisler 1980). Maximum net daily accumulation rates by various whole marine organisms were 1.3 mg Zn/kg FW for the alga *Ascophyllum nodosum*, 7.7 mg Zn/kg FW for the common mussel *Mytilus edulis*, 19.8 mg Zn/kg FW for the oyster *Crassostrea virginica*, 32 mg Zn/k FW for the killifish *Fundulus heteroclitus*, 32 mg Zn/kg FW for the softshell clam *Mya arenaria*, and 223 mg Zn/kg FW for the sandworm *Nereis diversicolor*; in general, accumulation rates and total accumulations were higher at elevated water temperatures and at higher ambient zinc water concentrations (Eisler 1980).

Algae and Macrophytes

Blue green algae are among the most zinc-resistant aquatic plants (Vymazal 1986). Algae are classified by Vymazal (1986) as very resistant (>10 mg Zn/L), resistant (2-10 mg/L), moderately resistant (0.5-2 mg/L), low resistant (0.1-0.5 mg/L; *Navicula*, *Synedra*), and very low resistant (<0.1 mg Zn/L; *Diatoma*, *Tabellaria*, *Microspora*, *Ulothrix*).

The most sensitive aquatic plant was *Schroederella schroederi*, a diatom; 19 µg Zn/L was sufficient to inhibit growth by 50% in 48 h (EPA 1987). Freshwater aquatic plants are usually absent from areas containing >2.0 mg Zn/L; in hard waters of artificial streams containing 170 mg $CaCO_3$/L, a water concentration of 1.1 mg Zn/L caused a 50% decrease in the number of algal species (Spear 1981). Most freshwater diatom populations decreased in the range of 175-380 µg Zn/L; this sensitivity may be useful as an indicator of zinc contamination (Spear 1981). Zinc and cadmium are strongly synergistic in their toxic action to plants. Any level of cadmium >10 µg/L should be suspected of producing a significant increase in the toxicity of available zinc to freshwater plants (Whitton 1980).

In heavily-contaminated zinc environments (130-6,500 µg Zn/L), zinc-tolerant species are dominant (Spear 1981). Highly-tolerant strains of algae require 1.5-1.65 mg Zn/L for normal growth; at least three species of

some tolerant strains can live in water containing 3 g Zn/L (Vymazal 1986). Highly tolerant mutant strains of *Anacystis nidulans* required 1.5-16.5 mg Zn/L. In France, at least 17 species of freshwater algae seemed to be flourishing at 42.5 mg Zn/L and pH 4.2 (Vymazal 1986). Zinc-tolerant strains of aquatic algae tolerate high zinc concentrations with little bioconcentration. A zinc-tolerant strain of *Euglena gracilis*, for example, tolerates >700 mg Zn/L but contains <500 mg Zn/kg DW whole organism versus 50 mg Zn/L and 5,000 mg/kg DW for nontolerant strains (Fukami et al. 1988a). Another zinc-tolerant strain of *Euglena* had normal growth at 300 mg Zn/L and residues of about 7,000 mg Zn/kg DW versus the population decline of nontolerant strains at 300 mg Zn/L (Fukami et al. 1988b).

Algae are effective accumulators of zinc. Three species of marine algae had a mean BCF of 1,530 in 12 days, 4,680 in 34 days, and 16,600 in 140 days (EPA 1980). Bioconcentration factors for zinc and various species of algae are quite variable and usually range from 76 to 163,750 (Vymazal 1986; EPA 1987). Many species of aquatic plants contain ≥150 mg Zn/kg DW. In one case, algae (*Mougeotia* spp.) from northern England in zinc-contaminated waters contained a spectacular 219 g Zn/kg DW (Vymazal 1986); it is probable that most of the zinc in *Mougeotia* was not biologically incorporated. Algal accumulations of zinc are modified significantly by physiochemical variables. Zinc concentrations in algae were higher under conditions of decreasing light intensity, water pH, DDT levels, copper, cadmium, phosphate, suspended sediments, organic chelators and other complexing agents, calcium, and magnesium and under conditions of increasing water temperature, dissolved oxygen, duration of exposure, and ambient zinc concentrations (Eisler 1980; Whitton 1980; Vymazal 1986).

Unlike algae, submerged aquatic macrophytes play a minor role in cycling of zinc (Lyngby et al. 1982). Rooted aquatic macrophytes may participate in heavy metal cycling in the aquatic environment either as a source or as a sink. But studies with eelgrass (*Zostera marina*) show that zinc exchange between the sediment and the water is insignificant (Lyngby et al. 1982).

Molluscs

Zinc was most toxic to representative molluscs at elevated temperatures (Eisler 1977a; Sprague 1986; Khangarot and Ray 1987), in comparatively soft water or to marine molluscs in low salinity (Sprague 1986; Khangarot and Ray 1987), at earlier developmental stages (Munzinger and Guarducci 1988), at low dissolved oxygen concentrations (Khangarot and Ray 1987), and with increasing exposure to high zinc concentrations (Amiard-Triquet et al. 1986).

High zinc accumulations in molluscs are usually linked to high levels of calcium in tissues, low ambient concentrations of iron or cobalt, exposure to organochlorine or organophosphorus insecticides, low salinity, elevated temperatures, increased particulate loadings in medium, increasing length of exposure to higher doses of zinc, increasing age of the organism, and especially to proximity of heavily carbonized and industrialized areas (Eisler 1980). Radiozinc-65 was rapidly accumulated in southern quahogs (*Mercenaria campechiensis*) during a 10-day period; accumulation in the kidney was linear over time and enhanced at elevated phosphate loadings in the medium (Miller et al. 1985).

Large variations in daily zinc accumulation rates by marine bivalve molluscs are typical. For example, softshell clams (*Mya arenaria*) immersed in 500 µg Zn/L at 16-22° C had daily accumulation rates of 2 mg/kg FW soft parts on day 1 of exposure, 7.7 mg Zn/kg FW soft parts between days 1 and 7, and 3.3 mg Zn/kg FW soft parts between days 7 and 14. At a lower temperature regimen of 0-10° C, immersion in 500 µg/L produced daily accumulation rates of 9.9 mg/kg FW soft parts for the first 42 days, but clams lost zinc at a rate of 0.24 mg/kg daily between days 42 and 112 (Eisler 1981). At 2,500 µg/L and 16-22° C, daily accumulation rates in surviving *Mya* were 32.0 mg Zn/kg FW soft parts on day 1 of exposure and 11.7 between days 1 and 7. Changes in accumulation rates of zinc by *Mya* reflect, at least partially, complex interactions between water temperature, ambient zinc concentrations, duration and season of exposure, and physiological saturation and detoxification mechanisms (Eisler 1977a, 1977b).

The half-time persistence (Tb½) of zinc in whole molluscs is extremely variable and reported to range from 4 days in the common mussel (*Mytilus edulis*) to 650 days in the duck mussel (*Anodonta nutalliana*); intermediate values were 23-40 days in the limpet (*Littorina irroratea*), 76 days in the California mussel (*Mytilus californianus*), and 300 days in the Pacific oyster (NAS 1979). Zinc persistence in selected organs also shows considerable variability and may be significantly different from Tb½ values in the whole animal. For example, the

Tb½ of zinc in the *Mytilus edulis* kidney was estimated at 2 to 3 months (Lobel and Marshall 1988) versus 4 days for whole animal (NAS 1979).

Mytilus edulis has been used extensively as a model for molluscan zinc kinetics. Results of selected studies follow. In mussels, zinc is taken up by the digestive gland, gills, and mantle and rapidly transported by hemolymph to the kidney where it is stored in insoluble granules (Lobel and Marshall 1988). There is a high degree of variability in soft tissues of *M. edulis* that is due entirely to an unusually high degree of variability in zinc of 97 to 7,864 mg/kg DW in the kidney (Lobel 1987). This variability in zinc content of the kidney is due largely to a low molecular weight zinc complex (700-1,300) that showed a high degree of variability and a positive correlation with zinc concentration in the kidney (Lobel and Marshall 1988). But at low ambient concentrations of 50 µg Zn/L, the most sensitive bioindicators of zinc exposure were gills and labial palps (Amiard-Triquet et al. 1986). Food composition had little effect on tissue distribution of radiozinc-65 in mussels as judged by 5-day feeding studies of radiolabeled diatoms (*Thalassiosira pseudonana*), green alga (*Dunaliella tertiolecta*), glass beads, and egg albumin particles (Fisher and Teyssie 1986). Soft part BCF values ranged from 12 to 35 times and was probably due to a rapid desorption of radiozinc from the food particles into the acidic gut, followed by binding to specific ligands or molecules. The Tb½ in mussel soft parts ranged from 42 to 80 days for all food items--including glass beads--and about 20 days in shell (Fisher and Teyssie 1986). Elevated temperatures in the range 10° to 25° C were associated with increased uptake rates of zinc from seawater by mussels (Watkins and Simkiss 1988). If the temperature is oscillated through this range during a 6-h period, there is a further enhancement of zinc uptake. This effect parallels decreases in zinc content of cytosol fractions and increases in granular fractions (Watkins and Simkiss 1988). Mussels were more sensitive to zinc than other tested bivalve molluscs. The pumping rate of mussels completely stopped for as long as 7 h on exposure to 470 to 860 µg Zn/L; however, other tested bivalves showed only a 50% reduction in filtration rates in the range of 750 to 2,000 µg Zn/L (Redpath and Davenport 1988). *Mytilus edulis* accumulates zinc under natural conditions but does not depurate under some conditions (Luten et al. 1986). This conclusion was based on results of a study of mussels that were transferred from a pristine environment in the Netherlands to a polluted estuary for 70 days and then returned for 77 days. At the start, zinc concentration was 106 mg/kg DW soft parts. By day 70, it had risen to 265 mg/kg DW at a linear daily uptake of 0.47 mg/kg. But mussels contained 248 mg/kg DW on day 147, indicating that elimination was negligible (Luten et al. 1986). In another study, zinc depressed sperm motility through respiratory inhibition at 6.5 mg/L, a concentration much higher than that normally found environmentally (Earnshaw et al. 1986). In mussel spermatozoa, zinc caused reductions of bound calcium and phosphorus in both acrosomes and mitochondria, suggesting increased permeability of organelle membranes to both elements (Earnshaw et al. 1986).

Arthropods

Arthropods were the most zinc-sensitive group of tested invertebrates (Table 6). Toxicity was usually greatest to marine crustaceans (Eisler 1981), to larvae (Eisler 1980), at elevated temperatures (Spear 1981; Sprague 1986; McLusky and Hagerman 1987), during extended exposures (EPA 1980, 1987), in soft water (Winner and Gauss 1986; Paulauskis and Winner 1988), under condition of starvation (NAS 1979; Verriopoulos and Moraitou-Apostolopoulou 1989), at salinity extremes above and below the isosmotic point (McLusky and Hagerman 1987), in summer (Eisler 1980), at low concentrations of humic acid (Winner and Gauss 1986; Paulauskis and Winner 1988), in proximity to anthropogenic discharges (Eisler 1980), and at low sediment particulate loadings (Memmert 1987). Acquired zinc tolerance is reported in amphipods collected from zinc-contaminated sewage wastes (Johnson and Jones 1989) and in fiddler crabs (*Uca* spp.) from a metals-contaminated area. *Uca* from zinc-contaminated areas were more resistant to zinc than crabs from pristine areas, as judged by increased survival and lower tissue zinc concentrations (Devi 1987; Devi and Rao 1989a, 1989b). More research into acquired zinc tolerance seems warranted.

Adverse effects of zinc insult to crustaceans include gill histopathology in prawns, *Macrobrachium hendersodyanum* (Patel and Kaliwal 1989); increased tissue total proteins, decreased glycogen, and decreased acid phosphatase activity in crabs, *Portunus pelagicus* (Hilmy et al. 1988); retardation of limb regeneration of fiddler crabs, *Uca pugilator* (Weis 1980); Waiwood et al. 1987). For example, tissue zinc residues in Homarus americanus exposed for 4 days to 25 mg Zn/L were especially high in gills (2,570 mg Zn/kg DW vs. 126 mg Zn/kg DW at start), hepatopancreas (734 mg Zn/kg DW vs. 135 mg Zn/kg DW), and green gland (1,032 mg Zn/kg DW vs. 148 mg Zn/kg DW). After 7 days in uncontaminated media tissue zinc residues remained elevated in gills (675 mg Zn/kg DW), hepatopancreas (603 mg Zn/kg DW), green gland (286 mg Zn/kg DW), and other

tissues (Waiwood et al. 1987). Zinc concentrations in crustacean soft tissues usually are between 50 and 208 mg/kg DW and exceed soft tissue zinc enzymatic requirements by factors of 1.4 to 6.0 (Depledge 1989).

Half-time persistence of zinc is about 17 days in the prawn (*Palaemon elegans*; Nugegoda and Rainbow 1988b) and between 30 and 270 days in five other crustacean species (NAS 1979). Differences in half-time persistence are linked to differences in excretion rates of ionic zinc and complexed zinc. In general, crustaceans excrete ionic zinc first and complexed zinc next; surface-adsorbed zinc is turned over faster than internally-adsorbed zinc; molting accounts for a 33-50% loss of the total body burden in crabs (Eisler 1981).

Crustaceans can accumulate zinc from both water and food (EPA 1987). In uncontaminated waters, the diet is probably the major source of zinc. Absorption from the stomach is efficient and occurs in part through the hepatopancreas. When a large pulse of zinc reaches the blood from the stomach, some is excreted, but much is resorbed and stored in the hepatopancreas in a relatively nonlabile form. Ultimately, stored zinc is also excreted, although removal through the gut is unimportant (Bryan et al. 1986). Zinc absorption is initially at the gill surface, is followed by transport on a saturable carrier in the cell wall, and is most efficient at low dissolved ambient zinc concentrations. Urinary excretion is an important body removal pathway, especially at high dissolved ambient concentrations when it can account for 70-80% of the total zinc excretion (Bryan et al. 1986).

Barnacles (*Elminius modestus*) usually accumulate zinc to high body concentrations without significant excretion. Barnacle detoxification mechanisms of the stored zinc includes production of metabolically inert zinc phosphate granules (Rainbow and White 1989). However, *Elminius modestus* transplanted from an area of high ambient zinc (101 µg/L) to an environment of low ambient zinc (4 µg/L) lost zinc slowly (0.3% body burden daily) during an 11-week period. Whole body zinc burdens declined from 1,554 to 125 mg/kg DW or at about 4.1 mg/kg DW daily (Thomas and Ritz 1986). In the case of *Balanus balanoides*, another barnacle, high BCF values were attributed to inorganic granules that contained as much as 38% zinc and accumulated in tissues surrounding the midgut (Eisler 1980).

Crustaceans--and other groups--can regulate body concentration of zinc against fluctuations in intake, although the ways in which regulation is achieved vary among species (Bryan et al. 1986). Regulation of whole body zinc to a constant level is reported for many crustaceans, including intertidal prawns (*Palaemon* spp.), sublittoral prawns (*Pandalus montagui*), green crabs (*Carcinus maenus*), lobsters (*Homarus gammarus*), amphipods (*Gammarus duebeni*), isopods (*Asellus communis*), and crayfish (*Austropotamobius pallipes*; Devineau and Amiard-Triquet 1985; Bryan et al. 1986; Lewis and McIntosh 1986; Nugegoda and Rainbow 1988b; Johnson and Jones 1989; Rainbow and White 1989). The body zinc concentration at which zinc is regulated in crustaceans usually increases with increasing temperature, salinity, molting frequency, bioavailability of the uncomplexed free metal ions, and chelators in the medium (Nugegoda and Rainbow 1987, 1988a, 1989a, 1989b). Lobsters (*Homarus gammarus*) are able to equilibrate over a 30-day period in seawater containing between 2 and 505 µg/L. In response to a 100-fold rise in seawater concentrations (from 5 to 500 µg/L), zinc levels in whole body, blood, hepatopancreas, excretory organs, and gills almost doubled but changed little in muscle. Zinc concentrations in shells increased about 12 times, largely through adsorption (Bryan et al. 1986). Regulation of zinc in lobster blood is achieved by balancing uptake through the gills against urinary excretion and loss over the body surface including the gills (Bryan et al. 1986). The sublittoral prawn (*Pandalus montagui*) can regulate total body zinc concentration to a constant level (75 mg/kg DW) in dissolved zinc concentrations up to 22 µg/L, beyond which there is net accumulation of body zinc. This threshold of zinc regulation breakdown is lower than that in *Palaemon elegans* (93 µg Zn/L) and *Palaemonetes varians* (190 µg Zn/L) under the same physiochemical conditions (Nugegoda and Rainbow 1987, 1988a, 1988b, 1989a, 1989b, 1989c; Rainbow and White 1989). The authors conclude that regulation of body zinc concentration is most efficient in decapods adapted to the fluctuating environments of littoral habitats, possibly, as a result of changes in permeability of uptake surfaces in combination with improved zinc excretion systems.

Freshwater crayfish (*Orconectes virilis*) are among the more resistant crustaceans (LC50 value of 84 mg Zn/L in 2 weeks) and can easily tolerate the recommended water quality criteria of 50-180 µg/L; nevertheless, some streams in Arkansas and Colorado contain 79-99 mg Zn/L (Mirenda 1986). *Orconectes virilis* exposed to extremely high sublethal ambient zinc concentrations of 63 mg/L for 2 weeks show whole body BCF values of only 2; a similar pattern was observed at other concentrations. In all cases, zinc tended to concentrate in gills and hepatopancreas at the expense of muscle, carapace, and intestine (Mirenda 1986). In freshwater crayfish (*Procambarus acutus acutus*), the major uptake route was the ambient medium and not diet, although retention

time of dietary zinc was greater (Giesy et al. 1980). When dietary zinc was the only zinc source, crayfish rapidly reached a steady state; when water was the only zinc source, crayfish did not reach a steady state (Giesy et al. 1980). Freshwater mysidaceans and their particulate wastes may play an important role in zinc cycling. The freshwater opossum shrimp (*Mysis relicta*) feeding on sediments ingested 2 to 4 times more zinc than mysids feeding on zooplankton. However, sediment-feeding mysids excreted 3 to 5 times more zinc than zooplankton consumers; zinc concentrations were up to 24 times higher in fecal pellets of sediment feeders than in food (Van Duyn-Henderson and Lasenby 1986). In the freshwater crayfish *Austropotamobius pallipes*, fecal excretion is a major zinc removal pathway; a similar case is made for the green crab (*Carcinus maenus*; Bryan et al. 1986).

Marine copepods (*Anomalocera*, *Acartia*, *Temora*) excreted 52% of the ingested zinc in fecal pellets that subsequently leached all zinc to seawater within 24 h (Fisher et al. 1991).

Freshwater insects, including many species of mayflies, damselflies, stoneflies, and caddisflies, are relatively tolerant to zinc, with LC50 values usually >1.33 mg/L--although some species were adversely affected at concentrations between 30 and 37 µg Zn/L (EPA 1987; Table 6). Mayfly (*Epeorus latifolium*) larvae were adversely affected at ambient water concentrations of 30 µg Zn/L but could tolerate dietary loadings of 600 mg Zn/kg DW ration without measurable effects on growth or emergence (Hatakeyama 1989). Chironomid insect populations were reduced or missing immediately downstream from coal mine drainage containing 5-10 mg Zn/L; populations further downstream recovered numerically but in comparison with upstream communities, their diversity was reduced (Wilson 1988).

Annelids

Populations of freshwater oligochaetes and leeches were reduced in numbers of individuals and number of taxa in mine tailing effluents containing 146-213 µg Zn/L or sediments containing >20 g Zn/kg DW (Willis 1985b). Leeches (*Erpobdella octoculata*) experienced a reduction in density and reproductive capacity in streams containing 25 to 310 µg Zn/L from mine wastes and did not avoid these harmful concentrations (Willis 1989).

The highest rate of net zinc absorption reported for any group of invertebrates was 2,230 mg Zn/kg BW daily in sandworms (*Nereis diversicolor*) from sediments with low zinc levels during exposure for 34 days in 250 mg Zn/L. At 10 mg Zn/L, the rate decreased to 55 mg Zn/kg BW daily (Eisler 1981). Zinc uptake in *Nereis* increased with increasing sediment zinc levels, at lower salinities (Eisler 1980), and at elevated temperatures (Fernandez and Jones 1987, 1989). Zinc had no significant effect on burrowing behavior of *Nereis*, even at acutely lethal concentrations (Fernandez and Jones 1987). Sandworms from zinc-contaminated sediments were more resistant to waterborne zinc insult by 10-100 times than sandworms from clean sediments (EPA 1987). Tolerance to zinc in sandworms may be a result of acclimatization or genetic adaption. In either event, the degree of metal tolerance decreases rapidly as the level of zinc contamination declines, suggesting that some zinc-tolerant worms may be competitively inferior to normal individuals in clean environments (Grant et al. 1989). More research on zinc-tolerant populations seems merited.

Unlike other major groups of marine benthic organisms, the polychaete *Neanthes arenaceodentata* has a limited capacity to regulate zinc (Mason et al. 1988). Uptake in *Neanthes* occurs from the free ionic pool of zinc whereas EDTA complexes and EDTA-zinc complexes are largely excluded. Zinc accumulates linearly over time (350 h) and the rate decreases with increasing temperature in the range 4-21° C. Mason et al. (1988) concluded that uptake and accumulation of zinc is passive in *Neanthes* and does not require metabolic energy. Zinc transfer across the plasma membrane is by way of diffusion. Inside the cell, zinc binds to a variety of existing ligands that maintain an inwardly directed diffusion gradient, preventing zinc efflux. Accumulation rats is determined by the number and binding characteristics of the available ligands and their accessibility to zinc. After 50 h of exposure, worms selectively accumulate zinc over cadmium from the medium by a process requiring metabolic energy, and this is attributed to a change in the turnover rate and to the size and nature of the pool of zinc-binding ligands (Mason et al. 1988).

Echinoderms

In echinoderms, zinc concentrations are usually higher in detrital feeders than in carnivores, higher in surface feeders than in sediment feeders, and higher in specimens collected inshore than those collected offshore in deeper waters (Eisler 1980). Sea cucumbers (*Stichopus tremulus*) accumulate radiozinc-65 from

seawater by a factor of 1,400; however, radiozinc accumulation data should be viewed with caution because addition of stable zinc can reduce radiozinc-65 accumulations in echinoderm viscera up to 10-fold (Eisler 1981). Zinc inhibits the formation of the fertilization membrane in sea urchin eggs, possibly by interfering with cortical granule-derived proteases and proteins (Nakamura et al. 1989).

Fish

Several trends are evident (Table 6): (1) freshwater fish are more sensitive to zinc than marine species; (2) embryos and larvae are the most sensitive developmental stages; (3) effects are lethal or sublethal for most species in the range 50-235 µg Zn/L and at 4.9-9.8 µg Zn/L for the brown trout (*Salmo trutta*); and (4) behavioral modifications, such as avoidance, occur at concentrations as low as 5.6 µg Zn/L. Signs of zinc poisoning in fish included hyperactivity followed by sluggishness before death, fish swam at the surface, were lethargic and uncoordinated, showed hemorrhaging at gills and base of fins, shed scales, and had extensive body and gill mucous (Bengeri and Patil 1986). Zinc is most toxic to yearlings of brown trout in soft water at pH 4-6 and pH 8-9; toxicity at alkaline pH is attributed to the formation of ZnOH+, $Zn(OH)_2$, and $ZnCO_3$ in both hard and soft water--suggesting increased entrapment of metal precipitates within mucous and epithelial layers of the gill (Everall et al. 1989a). Acute zinc poisoning in fish is generally attributed to blockade of gas exchange across the gills, causing hypoxia at the tissue level. Tissue hypoxia in fish is a major physiological change before death once the gas exchange process at the gills is no longer sufficient to meet its oxygen requirements (Burton et al. 1972; NAS 1979; Everall et al. 1989a; Grobler et al. 1989). Cardiorespiratory responses to zinc in the spangled perch (*Leiopotherapon unicolor*) are similar to those induced by hypoxia; zinc-poisoned perch had damaged gill epithelia, resulting in impaired gas exchange and lowered oxygen tension in arterial blood (Gehrke 1988). Acute exposures to high lethal concentrations of zinc also caused histopathology of epithelia lining the oral capacity (Eisler and Gardner 1973).

Many factors modify the lethal properties of zinc to fish. Zinc is more toxic under conditions of comparatively low dissolved oxygen concentrations, high sodium concentrations, decreased loadings of organic complexing agents (Spear 1981), and low pH (NAS 1979). In guppies (*Poecilia reticulata*), females were more resistant than males to acute zinc insult; adults of both sexes were more resistant than 5-day-old fry (Pierson 1981). Dominant bluegills (*Lepomis macrochirus*) survived exposure to 32 mg Zn/L longer than submissive fish (NAS 1979). Water temperature is also an important modifier and it is generally agreed that zinc is more toxic at elevated temperatures (NAS 1979; Spear 1981; Hilmy et al. 1987c) when acclimatization temperature is considered. For example, cold-acclimatized (3° C) Atlantic salmon survived longer than warm-acclimatized (19° C) salmon when exposed to lethal concentrations of zinc at their respective acclimatization temperatures. However, at test temperatures lower than their former acclimatization temperatures, salmon were less tolerant of zinc (Hodson and Sprague 1975).

Fish surviving high sublethal concentrations of zinc had significant alterations in blood and serum chemistry, liver enzyme activity (Hilmy et al. 1987b), muscle glycogen, total lipids, phospholipids, cholesterol, RNA, and proteins (Taneja et al. 1988).

Reproductive impairment seems to be one of the more sensitive indicators of zinc stress in freshwater teleosts, and effects are evident in the 50-340 µg Zn/L range (Spear 1981). In some cases, reproduction was almost totally inhibited at zinc concentrations that had no effect on survival, growth, or maturation of these same fish (Brungs 1969). Zinc-induced developmental abnormalities were documented in marine teleosts, but concentrations were grossly elevated. Eggs of the Baltic herring (*Clupea harengus*), for example, exposed to >6 mg Zn/L had an altered rate of development and produced deformed larvae with cellular disruptions in the brain, muscle, and epidermis (Somasundaram 1985; Somasundaram et al. 1985).

Avoidance tests with fathead minnows (*Pimephales promelas*) showed that almost all except males with established territories avoid 284 µg Zn/L when given a choice; avoidance thresholds were 6.4 times higher for established males (Korver and Sprague 1989).

Limited tolerance to zinc was observed in freshwater fish preexposed to sublethal levels of zinc (Spear 1981; Heath 1987; Woodall et al. 1988; Anadu et al. 1989; Hobson and Birge 1989). In one case, rainbow trout acclimatized to 50 µg Zn/L for 21 days were as much as 5 times more tolerant to subsequent zinc exposures than nonacclimatized trout; this was not evident at 100 µg Zn/L; also, acclimatization to zinc produced tolerances to copper and cadmium in trout (Anadu et al. 1989). The mechanisms to account for this

phenomenon are unknown, but several theories are proposed: increased metallothionein synthesis (Woodall et al. 1988), although this is disputed by Hobson and Birge (1989); high mortality during preexposure may have caused the selection of more zinc-tolerant individuals (Spear 1981); and tolerance may be limited to strains capable of increased zinc excretion, although no evidence now exists linking genetic mechanisms to zinc resistance (Spear 1981).

The estimated half-time persistence (Tb½) of zinc in whole mosquitofish (*Gambusia affinis*) was 215 days (Newman and Mitz 1988). The half-time persistence of zinc in whole marine fish ranged from 35 to 75 days in the mummichog (*Fundulus heteroclitus*) to 295-313 days in a flatfish (*Pleuronectes platessa*); Tb½ in mummichogs was shortest at 30° C, longest at 10° C, and intermediate at 20° C (NAS 1979).

Fish can accumulate zinc from both the surrounding medium and from their diet (EPA 1987). The freshwater zebra danio (*Brachydanio rerio*) accumulated zinc from the medium, but there was no additional zinc enrichment from a *Daphnia* diet (Memmert 1987). In marine fish, however, diet was considered the major route of zinc intake and significantly more important than water zinc levels (Eisler 1980).

In freshwater fish, BCF values for whole individuals were between 51 and 500 times (EPA 1987) but are strongly influenced by dose, duration of exposure, water chemistry, and other variables. In mosquitofish, uptake rate from water and zinc elimination rate decreased with increasing age of the fish (Newman and Mitz 1988). In the three-spined stickleback (*Gasterosteus aculeatus*), uptake was greater in hard water than in soft water and greater in larger fish, suggesting a surface adsorption mechanism (Matthiessen and Brafield 1977). In brown trout, however, uptake was lower and excretion greater in hard water of 220 mg $CaCO_3$/L than in soft water of 9 mg $CaCO_3$/L, thereby reducing tissue burdens (Everall et al. 1989a). Starved rainbow trout accumulated zinc more rapidly than fed fish because of an increased contribution of waterborne zinc to total body zinc levels (Handy and Eddy 1990). Rapidly growing chinook salmon (*Oncorhynchus tshawytscha*) fingerlings removed radiozinc-65 from the medium and retained nearly all of it for 63 days after transfer to uncontaminated media. Most of the radiozinc-65 was translocated to vertebral column, head, and visceral mass (Joyner and Eisler 1961). The outer surface of the bone seems to be an ion-exchange medium capable of taking up large quantities of metal ions whether natural or foreign to the system. Metals thus exchanged from serum proteins may be prevented from undergoing further exchange by the overlayering action of growing bone (Joyner and Eisler 1961). Channel catfish (*Ictalurus punctatus*) fingerlings fed diets containing up to 200 mg Zn/kg FW ration for 12 weeks had elevated bone zinc levels (359 mg/kg DW vs. 254 mg/kg DW in controls) and reduced hematocrit, but survival and feed conversion efficiency was the same as by controls (Gatlin et al. 1989). Plasma zinc levels in four species of freshwater fish on diets containing 100-200 mg Zn/kg ration ranged between 9.3 and 15.1 mg Zn/L FW; in rainbow trout, zinc tended to concentrate in the erythrocyte membrane (Bettger et al. 1987).

In marine fish, zinc residues were usually higher in dead than in live or moribund animals, higher in smaller fish, higher in liver and viscera, and higher with decreasing water cadmium levels (Eisler 1980). Uptake from the medium by adult mummichogs was inversely related to zinc concentration in the water (EPA 1987). In mummichogs, zinc accumulates in scales during exposure to 10 mg Zn/L, significantly elevating the zinc to calcium ratio; ratios remained elevated for at least 4 months after transfer to low zinc media, and this phenomenon may have application for environmental monitoring (Sauer and Watabe 1989a).

Scale osteoblasts of zinc-exposed mummichogs showed an increase in the number of lysosome-like structures contained by cytoplasm and suggests that osteoblast lysosomes are involved in zinc accumulation in fish scales by enzymatic degradation of metallothioneins or other metal-binding proteins (Sauer and Watabe 1989). Dietary zinc is not well assimilated in marine flatfish. Turbot (*Scopthalmus maximus*) fed diets containing 100 (control) or 1,000 mg Zn/kg DW for 200 days were not different in renal and hepatic metallothionein levels or in zinc concentrations in the liver, kidney, muscle, skin, or bone; a similar case is made for other marine flatfish (Overnell et al. 1988). However, intraperitoneally injected (2 mg Zn/kg BW) turbots had an 18-fold increase in liver metallothionein constant and a 3-fold increase in liver zinc, confirming the ability of this species to synthesize metallothionein rapidly to a high concentration (Overnell et al. 1988).

Amphibians

Amphibian embryos are more sensitive to zinc than older stages; developmental abnormalities were evident in most species at concentrations >1.5 mg Zn/L (Table 6). Embryos of the narrowmouthed toad (*Gastrophryne carolinensis*) seem to be especially sensitive; adverse effects were reported at 10 µg Zn/L (EPA 1987), but this requires verification. Amphibians and other taxonomic groups were rare or absent in the vicinity of zinc smelters but not in more distant sites (Beyer et al. 1985).

In tests with isolated skin of frogs (*Rana* spp.), Zn^{2+} stimulates sodium transport and inhibits chloride-related tissue conductance; however, the skin of toads is relatively insensitive to zinc (Nagel et al. 1988). In early stages of embryonic development, Zn^{2+} stimulates multiplication of germ cells, but long-term treatment with $ZnSO_4$ has a toxic effect on the larval gonad and especially on the germ cells of the ovarian structure that is developed in frog larvae (Gipouloux et al. 1986).

Birds

Ducks (*Anas* spp.) had reduced survival when fed diets containing 2,500-3,000 mg Zn/kg ration or when force-fed zinc metal shot equivalent to 742 mg Zn/kg BW (Table 7). Domestic chickens (*Gallus* sp.) were more resistant: 8,000 mg Zn/kg ration was fatal to chicks, although higher doses were routinely fed to laying hens to induce molting; 2,000-3,000 mg Zn/kg ration inhibited chick growth; 178 mg Zn/kg feed caused immunosuppression in chicks; and dietary concentrations as low as 100 mg Zn/kg caused pancreas histopathology in chicks under conditions of selenium deficiency (Table 7). Excessive zinc (2,000 mg/kg diet for 21 days) fed to chicks (*Gallus* sp.) caused zinc accumulations in tissues, reduced tissue turnover of zinc, reduced liver turnover of iron, and reduced copper content of the liver and pancreas and iron in the tibia (Stahl et al. 1989b). However, hens were less sensitive and, when fed diets containing 2,000 mg Zn/kg for 44 weeks, produced chicks that had no apparent alteration in tissue zinc, copper, or iron metabolism (Stahl et al. 1990).

Table 7. Effects of zinc on representative birds.

Species, dose, and other variables	Effects	Reference[a]
Mallard, *Anas platyrhynchos*		
Fed diets containing 3,000 mg Zn/kg feed, and higher, for 30 days	At 3,000 mg/kg ration, ducks had leg paralysis and decreased food consumption; at >3,000 mg/kg diet, many deaths occurred	1
Age 7 weeks. Fed diets containing 3,000, 6,000, 9,000, or 12,000 mg Zn/kg dry weight (DW) diet for 60 days; zinc in form of zinc carbonate	Food intake reduced for all groups; the 9,000 and 12,000 mg/kg groups had almost zero intake. High mortality after 30 days in all groups; only 17% of 3,000 mg/kg group alive at day 60. Zinc residues at time of death or at day 60 for the 3,000 mg/kg group were 89 mg/kg fresh weight (FW) in pancreas (1,252 mg/kg FW in controls); 401 mg/kg FW in liver (54 mg/kg FW); 88 mg/kg FW in adrenals (45 mg/kg FW); 413 mg/kg FW in kidney (27 mg/kg FW); 32 mg/kg FW in muscle (14 mg/kg FW); 78 mg/kg FW in testes (17 mg/kg FW); and 71 mg/kg FW in ovary (31 mg/kg FW)	2
Age 1 year. Single oral dose of five number 6 zinc shot in gelatin capsules,	All shot retained in gizzard after 14 days; no adverse effects after 28 days. Residues at	3

equivalent to 0.40 g zinc or 495 mg Zn/kg body weight (BW)	28 days were 217 mg/kg DW in liver, 79 mg/kg DW in kidney, and 126 mg/kg DW in feather	
Drakes, 18 months old, force-fed eight number 6 zinc shot pellets	By day 30 posttreatment, 20% had died. The mean weight loss was 33% in dead birds and 22% in survivors. About 83% of survivors developed signs of zinc poisoning	4
Age 1 year. Single oral dose of ten number 6 zinc shot in gelatin capsules, equivalent to 0.80 g zinc or 990 mg Zn/kg BW	Two to 4 shot voided in first 48 h, but no further loss for 28 days. Residues at 28 days were 211 mg Zn/kg DW in liver (171 mg Zn/kg DW in control birds), 72 mg Zn/kg DW in kidney (61 mg Zn/kg DW), 143 mg Zn/kg DW in feather (128 mg Zn/kg DW)	3
Pekin duck, *Anas platyrhynchos*		
3-day-old male white ducklings fed diet containing 2,500 mg Zn/kg, as $ZnSO_4.H_2O$, for 56 days	Progressive ultrastructural degeneration of pancreatic acinar cells evident as early as day 5	5
Japanese quail, *Coturnix coturnix japonica*		
Intratesticular injection of 3% zinc chloride equivalent to 1 mg Zn/kg testes or 0.02 mg/kg BW	Testicular teratomas produced during a period of testicular growth stimulated by increased photoperiod	6
Hens fed diet containing 15,000 mg Zn/kg ration, as zinc oxide, for 7 days	Significant reduction in body weight, egg production approached zero at day 3, eggshell breaking strength reduced, molting induced	7
14-day-old quail fed diets containing various concentrations of zinc, as zinc phosphide (a rodenticide) for 5 days followed by 3 days of untreated feed	At 600 mg Zn/kg ration, 7% died and all had reduced food intake. At 990 mg Zn/kg diet, 53% died; at 1,634 mg/kg died, 93% died	8
Domestic chicken, *Gallus* sp.		
Developing embryos, 1 day old, with 0.76 mg Zn/yolk at start, supplemented with 0.2, 0.4, or 0.6 mg zinc	Hepatic metallothionein levels increased by factors of 3.9 (0.2 mg), 4.7 (0.4 mg), and 7.1 (0.6 mg)	9
Femurs from 9-day-old chick embryos cultivated for 6 days at 3.26 mg Zn/L	Inhibited calcium accumulations in bone and increased alkaline phosphatase activity of medium	10
As above, 6.5 mg Zn/L	Decrease in calcified tissues	10
Domestic breeding hens fed diets containing 28, 38, 48, 68, 94, or 178 mg Zn/kg ration for up to 9 months	Progeny growth after 3 weeks was not affected by maternal zinc feeding levels. A minimum of 38 mg Zn/kg diet was considered necessary for minimal feather fraying and maximal immune response in chicks. Diets containing 178 mg Zn/kg may be excessive and cause	11

	immunosuppression of young progeny without affecting growth	
Fed 28 (control), 48, 228, or 2,028 mg Zn/kg diets for 12 or 44 weeks. Hens were 56 weeks old at start of short-term study and 24 weeks old at start of long-tern study	Zinc treatments had no effect on overall egg production, feed conversion, feed consumption, hatchability, or progeny growth to age 3 weeks. Zinc was elevated in eggs from hens fed the 2,028 mg/kg diet, but chick performance and tissue zinc content were unaffected by maternal zinc nutritional status	12
Chicks fed diets containing 37 (control), 100, or 2,000 mg Zn/kg feed for 21 days	No accumulations in 100 mg/kg group; zinc excretion rate about x2 controls. No deaths in 2,000 mg/kg group, but growth rate was decreased, anemia evident, tissue copper and iron decreased, and tissue zinc increased	13
Day-old chicks fed selenium-deficient diets plus 100 mg Zn/kg FW, as zinc oxide, purified ration for 9 days	Elevated zinc concentrations in pancreas, and pancreas histopathology	14
Hens fed diets containing 218, 257, 1,762, or 1,861 mg Zn/kg diet for up to 40 weeks	Eggs from hens fed 218 or 257 mg Zn/kg diet contained a maximum of 14 mg/kg FW, equivalent to about 25% more zinc than eggs produced by control hens. Eggs from the two higher-dose diets had a maximum of 19 mg/kg FW or 57-90% more zinc than eggs produced by hens fed a control diet of 26-28 mg Zn/kg.	15
9-day-old chicks fed purified diet containing 500 mg Zn/kg ration for 2 weeks	Plasma alpha-tocopherol reduced 64%; plasma and pancreas zinc concentrations elevated	16
Day-old chicks fed selenium-adequate diet plus 2,000 mg Zn/kg FW, as zinc oxide, nonpurified ration for 9 days	Negligible effects on pancreas zinc concentration and on pancreas exocrine function	14
9-day-old chicks fed nonpurified diet containing 2,000 mg Zn/kg ration for 80 days.	No effect on plasma alpha-tocopherol or plasma and pancreas zinc content	16
Chicks fed diets containing 2,000 or 3,000 mg Zn/kg ration for 30 clays	Slight reduction in growth at 2,000 mg/kg; significant growth reduction at 3,000 mg/kg	1
Day-old chicks fed diets containing up to 4,000 mg Zn/kg ration for 4 weeks	No effect on growth, survival, or feed conversion. Zinc accumulated in tissue metallothioneins, especially in liver and kidney; levels normal after 5 days on zinc-deficient diet	17
Day-old chicks fed diets containing 4,000, 8,000, or 16,000 mg Zn/kg for 5 weeks	All dead at 16,000 mg/kg diet. The 8,000 mg/kg group had 80% mortality; survivors had signifi-cantly reduced growth and feed conversion. At 4,000 mg/kg, no significant effect on growth or	17

	survival; zinc concentrations elevated in kidney, liver, intestinal mucosa, and pancreas--but values normal after 10 days on basal diet	
Age 71 weeks, laying hens. Fed diet containing 10,000 mg Zn/kg feed for 2 days, then 5,000 mg/kg diet for 4 days	Hens started to molt and ceased laying. Feed intake decreased about 90%. Zinc concentrations increased in pancreas 7 times, in liver 6 times, in kidney 3 times, and were elevated in shell gland and yolk. High zinc levels in kidney reflect high zinc excretion rates; high pancreatic zinc (410 mg Zn/kg FW) may suppress the release of insulin by calmodulin inhibition, and could account for the rapid cessation of lay	18
White leghorns and brown layers were fed diets containing 10,000, 20,000, or 30,000 mg Zn/kg feed, as zinc oxide, for up to 3 weeks to induce molting	Cessation of egg laying in all treatments. On resumption of egg production, zinc levels in albumin or eggshell were not affected by the treatment or strain; zinc levels in yolk increased and depended on feed intake rather than dose. No increase in zinc content in eggs laid after egg production resumed, regardless of dose or duration of zinc treatment	19
White leghorn laying pullets and hens fed diet containing 20,000 mg Zn/kg feed for 5 days	Reduced body weight on day 5, and significantly lowered egg production for 4 weeks. Eggs collected 14-28 days after the 5-day study period had reduced fertility and hatchability. Normal growth, egg production, fertility, and hatch-ability during weeks 4-12 posttreatment	20
Laying hens fed diet containing 20,000 mg Zn/kg, as zinc oxide, for 4 days followed by 18 days on basal (35 mg Zn/kg) diet	At day 4, liver zinc concentrations increased 10 times, kidney 3 times, egg yolk 3 times, and pancreas 25 times; liver and kidney values returned to normal by day 22, but pancreas concentration (1,673 mg/kg DW) remained elevated when compared to controls (88 mg/kg DW). At day 10, reduced weight of ovary and oviduct	21
Turkey, *Meleagris gallopavo*		
Zinc concentration of sperm storage medium increased from 25 to 90 mg/L.	Fertilizing ability of stored sperm significantly reduced	22

[a] 1. NAS 1979; 2. Gasaway and Buss 1972; 3. French et al. 1987; 4. Grandy et al. 1968; 5. Kazacos and Van Vleet 1989; 6, Guthrie 1971; 7. Hussein et al. 1988; 8. Hill and Camardese 1986; 9. Fleet and McCormick 1988; 10. Kaji et al, 1988; 11. Stahl et al. 1989a; 12. Stahl et al. 1990; 13. Stahl et al. 1989b; 14. Lu and Combs 1988a; 15. Stahl et al. 1988; 16. Lu and Combs 1988b; 17. Oh et al 1979; 18. Veheyen et al. 1990; 19. Decuypere et al. 1988; 20. Palafox and Ho-A 1988; 21. Williams et al. 1989;p 22. Blesbois and Mauger 1989.

Zinc-poisoned mallards (*Anas platyrhynchos*) force fed zinc shot pellets developed ataxia, paresis, and total loss of muscular control of legs, including the ability to swim (Wobeser 1981). The muscular, weakness associated with zinc intoxication would probably make ducks highly susceptible to predation and argues against the use of zinc shot as a substitute for lead shot (Grandy et al. 1968). Mallards fed 3,000 mg Zn/kg DW ration for 60 days had diarrhea after 15 days; leg paralysis in 20 days; high mortality after 30 days; and zinc residues that were 14 times higher in pancreas than in controls, 7 times higher in liver, 15 times higher in kidney; and 2 to 4 times higher in the adrenals, muscle, testes, and ovary at day 60 (Gasaway and Buss 1972).

In Australia, almost all aviary birds are held in cages of galvanized wire mesh, resulting in sporadic cases of "new wire disease" caused by the ingestion of galvanized metal. In one case, peachfaced lovebirds (*Agapornis roseicollis*) died within 5 weeks of placement in a newly erected wire cage; dead birds had elevated liver zinc concentrations of 75-156 mg/kg DW versus normal values of 21-33 mg/kg DW (Reece et al. 1986). Zinc poisoning in a captive Nicobar pigeon (*Caloenas nicobarica*) was attributed to plated zinc metal fragments found in the gizzard--presumably ingested from the galvanized cage bars. In addition to elevated tissue zinc concentrations, this pigeon had a swollen liver and kidneys and extensive kidney histopathology (Zee et al. 1985). A zinc-poisoned blue and gold macaw (*Ara ararauna*) showed weakness, ataxia, extreme thirst, diarrhea, cyanosis, and a plasma zinc concentration of 15.5 mg/L after ingesting galvanized hardware cloth that was 24% zinc by weight and 0.2% lead. The bird was treated with 35 mg/kg BW calcium versanate intramuscularly and 30 mg thiamine hydrochloride per kilogram of BW; recovery following chelation therapy took 2 months, at which time plasma zinc was 0.6-0.8 mg/L versus 1.3-2.0 mg/L for normal birds (Morris et al. 1986). New galvanized wire used in aviary construction should weather for 1 to 2 months and then be scrubbed with a mild acidic solution such as vinegar and rinsed; flakes of galvanized metal--which contain up to 2.4 g Zn/kg--should be removed before birds are put in cages (Reece et al. 1986).

Zinc toxicosis was diagnosed in a gray-headed chachalaca (*Ortalis cinereiceps*) after it ingested a copper-plated zinc penny; necropsy showed pancreas histopathology and severe gizzard erosion; liver contained 1,910 mg Zn/kg FW (Droual et al. 1991).

Large amounts of zinc are crucial for new feather growth. Zinc deficiency during this period results in stunted, frayed, easily-broken feathers. Studies with the giant Canada goose (*Branta canadensis maxima*) showed that zinc was released from the pectoralis muscle during molt-induced atrophy and used for growth of feathers and leg muscles during this period (Rosser and George 1986).

Zinc phosphide--a rodenticide--is relatively toxic in comparison with elemental zinc or zinc oxide; most of the biocidal action is attributed to the phosphide fraction. Acute oral LD50 values for zinc phosphide were between 16 and 47 mg/kg BW in the ring-necked pheasant (*Phasianus colchicus*), golden eagle (*Aquila chrysaetos*), mallard, and horned lark (*Eremophila alpestris*; Hudson et al. 1984). Signs of zinc phosphide poisoning include excessive drinking, regurgitation, muscular incoordination, appetite loss, sluggishness, rapid breathing, and eyelid droop. Signs appeared as soon as 15 min after dosing, and death usually occurred between 2 and 21 h; remission took up to 1 month (Hudson et al. 1984).

High dietary levels of zinc are frequently fed to poultry to force molting and reduce egg deposition (Decuypere et al. 1988; Hussein et al. 1988). Extremely high dietary levels of 20 g Zn/kg ration have been used as a commercial management technique to force the molting of laying hens .and the subsequent improvement of long-term egg production that molting produces (Lu and Combs 1988a). Laying hens given high zinc diets increased their zinc uptakes 5-40 times in a dose-dependent pattern despite the decreased food intake associated with high zinc dietary levels. Zinc preferentially accumulated in chicken kidney, liver, pancreas, and gizzard; significant increases in egg zinc occurred at dietary levels of 10 and 20 g Zn/kg (Verheyen et al. 1990). Unlike adults, high dietary levels of zinc adversely affected pancreatic exocrine function in the chick; effects were exacerbated under conditions of selenium deficiency and feeding of purified diets (Lu and Combs 1988a). Impaired enteric absorption and transport of vitamin E as a consequence of zinc-induced pancreatic insufficiency is a major cause of reduced tissue concentrations of alpha-tocopherol produced in chicks by excess dietary zinc; these effects were magnified by diets low in corn, soybean meals, and other materials known to chelate zinc and thus reduce its biological availability (Lu and Combs 1988b). Excess dietary zinc causes pancreatic damage in the chick, including reduced activities of major digestive enzymes, elevated plasma amylase activities, reduced digestibility of starch, and reduced vitamin A activity; these changes were associated directly with elevated tissue zinc concentrations, especially in the pancreas (Lu et al. 1990).

Mammals

Livestock and small laboratory animals are comparatively resistant to zinc, as judged by their tolerance for extended periods to dietary loadings >100 times the minimum recommended daily zinc requirement (Table 8). Nevertheless, excessive zinc intake through inhalation or oral exposure can have drastic effects on survival, metabolism, and well being. Sensitive species mammals were affected at 90-300 mg Zn/kg diet, >300 mg Zn/L drinking water, > 90 mg/kg BW daily, > 350 mg Zn/kg BW as a single oral dose, and > 0.8 mg Zn/m^3 air (Table 8).

Zinc is relatively nontoxic in mammals. A wide margin of safety exists between normal intakes and those producing deleterious effects. In most cases, dietary levels up to 100 times the daily requirement for extended periods show no discernable effects (NAS 1979; Wentink et al. 1985; Goyer 1986; Leonard and Gerber 1989). The possibility of oral zinc intoxification in adult humans is unusually low, as judged by the low (40%) bioavailability of zinc from the gastrointestinal tract and the high tolerances to zinc reported in domestic livestock and small laboratory animals (Llobet et al. 1988a, 1988b). Humans ingesting up to 12 g of elemental zinc, equivalent to 33 mg/kg BW for a 60-kg adult, during a 2-day period show no evidence of hematologic, hepatic, or renal toxicity (Goyer 1986).

Excessive zinc intake adversely affects survival of all tested mammals --including humans--and produces a wide variety of neurological, hematological, immunological, hepatic renal, cardiovascular, developmental, and genotoxic effects (PHS 1989). The most sensitive species of mammals showed adverse effects at dietary levels of 80-90 mg Zn/kg in humans, 300 mg Zn/kg ration in domestic cats, and 500 mg Zn/kg feed in rats; drinking water concentrations of 300 mg/L in domestic mice and 800 mg Zn/L in laboratory white rats; daily whole body intakes >90 mg Zn/kg in horses; acute oral LD50 doses of 350-800 mg Zn/kg BW in rats; intraperitoneal injections of 13 mg Zn/kg BW in mice; and 0.8 mg Zn/m^3 air in guinea pigs (Table 8).

Metal fume fever is commonly encountered by industrial workers exposed to zinc fumes and is characterized by pulmonary irritation, fever, chills, and gastroenteritis (Saxena et al. 1989b). Attacks begin 4-8 h after exposure and recovery, in 24-48 h. The pathogenesis of metal fume fever is unknown but may be associated with endogenous pyrogens released by cell lysis (Goyer 1986). Rabbits, rats, and cats exposed to zinc oxide fumes for 3.5 h at concentrations of 110-600 mg/m^3 reacted with a transient fall in body temperature followed by leucocytosis; heavily-exposed animals had signs of bronchopneumonia (Elinder 1986). The current atmospheric threshold limit value for zinc is 5 mg/m^3; however, results of studies with guinea pigs suggest that the current threshold limit value for zinc oxide should be lowered (Lam et al. 1985; Table 8).

Excessive zinc uptake is associated with lameness, unthrifty appearance, and osteochondrosis in foals and pigs, nephrosis in ferrets, and pancreatic fibrosis in sheep (Gunson et al. 1982). Zinc-poisoned mammals are usually characterized by a decreased growth rate, subcutaneous hematomas, ulcerative gastritis, hemorrhagic enteritis, lesions of major limb joints, renal lesions, elevated serum and tissue zinc concentrations, acute diarrhea, copper deficiency, impaired reproduction, and decreased activity of cardiac and hepatic cytochrome oxidase (Saxena et al. 1989b). In severe cases, histopathological changes in the liver and especially in the pancreas, and degenerative changes in the kidney and gastrointestinal tract are evident and are followed by life-threatening hemolytic anemia (Straube et al. 1980; Allen et al. 1983; Robinette 1990). The pancreas is the key to the diagnosis of zinc toxicity and in estimation of the period of exposure; in sheep, it takes about 4 weeks of continued ingestion of toxic amounts of zinc before the pancreas is affected (Allen et al. 1983). More research into the role of the pancreas in zinc toxicokinetics is needed.

Zinc is important to the normal functioning of the central nervous system. At low concentrations, zinc protects mammalian brain neurons by blocking N-methyl-D-aspartate receptor-mediated toxicity. At high concentrations, zinc is a potent, rapidly acting neurotoxicant in the mammalian brain, as judged by zinc-induced neuronal injury of in vitro mature cortical cell cultures (Choi et al. 1988). Increased brain levels of zinc are associated with Pick's disease in certain strains of rodents with inherited epileptic seizures. Intravenous injection of zinc in rats with genetically inherited epilepsy produces seizures; a similar response occurs with intracranial injection of zinc in rabbits with inherited audiogenic seizures (Choi et al. 1988).

Table 8. Effects of zinc on representative mammals.

Organism, route of administration, dose, and other variables	Effects	Reference[a]
Cows, cattle, *Bos* spp.		
Dairy cows fed control diet (310 mg Zn/kg dry weight [DW] feed) or control diet supplemented with 1,000 or 2,000 mg Zn/kg DW ration (as $ZnSO_4 \cdot H_2O$)	The 1,000 mg/kg supplement has no adverse effects on milk production, feed intake, body weight, general health, or reproduction; there was a moderate increase in zinc content of plasma and milk. Cows fed the 2,000 mg Zn/kg diet, however, had decreased milk yield and feed intake after several weeks; calf weights were lower; adverse effects reversed when excess zinc was removed from diet	1
Calves fed diets containing 600 mg Zn/kg for 21 days	Appeared normal, although zinc levels were elevated in pancreas, liver, and kidney	2
Lactating dairy cows fed diets containing 700 or 1,000 mg Zn/kg feed for 6 weeks	No change in general health or milk production; no increase in milk zinc content	2
Lactating cows fed diets containing up to 1,386 mg Zn/kg feed for 5 weeks	No significant change in food intake, weight gain, milk production or in zinc concentrations in plasma (1.15-1.3 mg/kg fresh weight [FW]) or milk (3.7-4.3 mg/kg FW)	3
Calves and young female cattle fed roughage harvested in vicinity of a factory galvanizing steel tubes, and containing 3,000-7,300 mg Zn/kg DW roughage	Signs of chronic zinc poisoning evident after 12-14 months. Signs included reduced appetite, emaciation, submandibular edema, diarrhea, moderate anemia, elevated serum zinc (4.3-6.0 mg/L versus normal 1.8-2.1 mg/L), liver zinc (420-1,600 mg/kg DW versus normal 72-248 mg/kg DW), kidney zinc (910-1,680 mg/kg DW versus normal 40-114 mg/kg DW), and low serum calcium and magnesium	4

Dog, *Canis familiaris*		
Fed diets containing up to 1,000 mg Zn/kg ration for up to 1 year	No measurable signs of damage	2
Pomeranian, 2.2 kg, 4 months old, ingested four copper-clad zinc pennies	Hemolytic anemia, vomiting, salivation, serum zinc dropped from 29 mg/L to 4.4 mg/L 15 days after coins were surgically removed (normal dog serum zinc values range between 0.6 and 2.0 mg/L)	5
Zinc-poisoned oral route, (lethal) dose unspecified	Tissue zinc concentrations (in mg/L or mg/kg FW) were 32 in serum, 16-32 in plasma, 20-25 in urine, 369 in liver, and 295 in kidney. Normal values were 0.7-1.1 in serum, 0.6-1.0 in plasma, 1.3-2.0 in urine, 17-32 in liver, and 9-23 in kidney	6 ..
Died from ingestion of 34 copper-clad zinc pennies	Elevated zinc levels in serum, liver, and kidney; jaundice, anoxeria, anemia, vomitation, dark red urine	7
Guinea pig, *Cavia* sp.		
Inhalation of 0.8 mg Zn/m^3 for 1 h	Difficulty in breathing	8
Inhalation of 4 mg Zn/m^3, 3 h daily for 6 days	Temporary lung damage	8
Inhalation of 5 mg Zn/m^3, as ultrafine zinc oxide, 3 h daily for 6 days	Decrease in lung capacity, alveolar volume, and diffusing capacity for carbon monoxide; values remained depressed for at least 72 h after last exposure. Persistent inflammation of proximal portion of alveolar ducts and adjacent alveoli	9, 10
Horse, *Equus caballus*		
Weanling foals, age 3 months, fed diets containing 7.7 mg Cu/kg plus 29, 250, 1,000, or 2,000 mg Zn/kg ration for 15 weeks. At start, serum zinc level was 0.6 mg/L and serum copper level 1.4 mg/L	Foals fed 29 or 250 mg Zn/kg diets had normal serum copper and zinc concentrations. Those fed 1,000 or 2,000 mg kg diet became hypocupremic in 5 to 6 weeks and developed lameness owing to cartilaginous disease similar to osteochondritis dessicous. Foals fed high zinc diets became lame when serum copper fell to 0.3 mg/L for >1 week; at end of study, arthritic foals had <0.2 mg Cu/L serum. Serum zinc	11

	concentrations rose to >2 mg/L within 2 weeks at 1,000 or 2,000 mg Zn/kg diet; liver zinc was <333 mg/kg DW at diets of 250 mg Zn/kg, 2,728-3,511 mg/kg DW at 1,000 mg Zn/kg diet, and 4,364-4,524 mg/kg DW at the highest dietary loading of 2,000 Zn/kg in 15 weeks	
Adults, vicinity of lead-zinc smelter, ingesting >90 mg Zn/kg body weight (BW) daily	Decreased growth, lameness, bone deformities, death.	2
Cat, *Felis domesticus*		
Fed diet containing 300 mg Zn/kg ration for 16 weeks	Weight loss and pancreas histopathology	2
Fed diets containing >600 mg Zn/kg ration	Diets rejected	2
Fed diet containing 9,000 mg Zn/kg ration for 3-53 weeks	Pancreas histopathology	8
Human, *Homo sapiens*		
Dietary route		
80 mg/kg ration for 6 weeks	Digestive problems	8
90 mg Zn/kg ration for 5 weeks	Decreased serum cholesterol levels	8
153 mg Zn/kg ration for 6 weeks	Altered immune system	8
<150 mg zinc daily	No effect on male plasma cholesterol; females have decreased cholesterol	12
160 mg zinc daily	Increased plasma cholesterol level in both sexes; increased risk of heart disease in males	12
Inhalation route, 600 mg Zn/m^3 for 10 min	Metal fume fever, that is, difficulty in breathing, flu-like symptoms	8
Oral route		
15-year-old girl who consumed 220 mg zinc sulfate twice daily "for some time"	Acute gastrointestinal bleeding ulcers	13
Boy who consumed 12 g of elemental zinc	Headache and lethargy	13
Single oral dose of 45 g zinc as $ZnS0_4$ (normal is 15-20 mg daily)	Death, preceded by dehydration, electrolyte imbalance, abdominal pain, nausea, vomiting, dizziness, muscular incoordination, and acute renal failure	14
Domestic mouse, *Mus* sp.		
Dietary route		
68, 682, or 6,820 mg Zn/kg ration for 13 weeks (fed as 300, 3,000, or	No observed effects at 682 mg/kg diet (and lower), equivalent to 104-109 mg Zn/kg BW daily. At	15

30,000 mg $ZnSO_4 \cdot 7H_2O$/kg ration)	6,820 mg Zn/kg ration, however, adverse effects were documented on survival, growth, food and water intake, and blood chemistry; lesions noted in pancreas, stomach, intestine, spleen, and kidney	
500 mg/kg for 3 months	Anemia	8
30,000 mg/kg ration for 13 weeks	Some deaths, liver and kidney histopathology	8
Drinking water, 300 mg/L, for 5-14 months	Pancreas histopathology	8
Intraperitoneal injection, four injections over 9-day period totaling 13 mg Zn/kg BW	Toxic. Severe weight loss and some deaths	23
European ferret, *Mustela putorius furo*		
Fed basal diet (27 mg Zn/kg feed) or basal diet plus 500, 1,500, or 3,000 mg Zn/kg ration for up to 197 days; four animals per group	Ferrets fed 500 mg/kg all survived with no significant histopathology; zinc concentrations were 148 mg/kg DW in liver (115 mg/kg DW in controls) and 383 mg/kg DW in kidney (180 mg/kg DW). At 1,500 mg/kg, all four ferrets were in extremis or dead by day 21. At death, liver zinc was 859 mg/kg DW and kidney zinc 1,000 mg/kg DW; ferrets had 40-50% loss in body weight; food intake had decreased 80%; and erythrocyte number, hemoglobin, and hematocrit had significantly decreased. Ferrets fed the 3,000 mg/kg diet died between days 9 and 13, lost up to 40% of initial BW, and food intake decreased 77%; postmortem examination showed blood in intestine, orange-colored liver, and kidney histopathology. Elevated zinc content in liver of 1,273 mg/kg DW and in kidney of 1,138 mg/kg DW	8,16,17
Rabbit, *Oryctolagus* sp.		
Single oral dose of 65 mg Zn/kg BW, as $ZnSO_4$	Half-time persistence of 713 min	8
Intravenous injection of 0.325 mg Zn/kg BW, as $ZnSO_4$	Half-time persistence of 268 min	8
Intraperitoneal injection of 3.4 mg zinc daily	Associated with lowered plasma cholesterol levels	12
Domestic sheep, *Ovis aries*		
Domestic ewe, age 5 years, found moribund, suspected zinc poisoning	Elevated zinc levels were 650 mg/kg DW in liver (144 mg/kg DW in controls) and 760 mg/kg DW in kidney (84 mg/kg DW); muscle residues same	18

	as controls, that is, 154 mg/kg DW (158 mg/kg DW); generalized jaundice; liver degeneration and blockage of bile ducts	
Found dead, zinc-poisoned naturally	Zinc concentrations were 463 mg/kg DW in liver (165 mg/kg DW in controls), 274 mg/kg DW in kidney (150 mg/kg DW), and 752 mg/kg DW in pancreas (88 mg/kg DW)	19
Zinc-poisoned experimentally, oral route	Zinc concentrations were 1,125-1,671 mg/kg DW in liver, 2,130-2,442 mg/kg DW in kidney, 1,440-1,932 mg/kg DW in pancreas, and 4,900 mg/kg DW in feces (158 mg/kg DW feces in controls)	19
Lambs fed diets containing 1,000 mg Zn/kg	Food intake reduced; approaching toxic level	2,7
Laboratory white rat, *Rattus* sp.		
Dietary route		
Adult males given 500 mg Zn/kg ration, as $ZnSO_4$, for 6 weeks	After 3 weeks, spermatogenesis was arrested at the primary spermatocyte stage. After 4 weeks, food consumption declined, forelimb lameness, and swelling in cervical lymph nodes. At 6 weeks, testes showed enlarged lumen and abnormal germinal epithelium	20
682 mg Zn/kg ration, as $ZnSO_4.7H_2O$, for 13 weeks	No observable effect level, equivalent to 53-55 mg Zn/kg BW daily	15
2,000 mg Zn/kg ration, chronic exposure	Tolerated	2
4,000-5,000 Zn/kg ration for 18 days	Fetotoxic dose, poor reproduction	2,8
5,000-10,000 mg Zn/kg ration	Reduced growth, anemia, poor reproduction, disrupted liver catalase and cytochrome oxidase activity, copper deficiency	14
6,820 mg Zn/kg ration for 13 weeks	Retarded growth, low food intake, abnormal blood chemistry, regressive changes in pancreas	15
Drinking water route		
Doses equivalent to 0, 160, 320, and 640 mg Zn/kg BW daily for 3 months	No significant effect of any dose on organ weight, hematocrit, hemoglobin, glucose, and enzyme activity. Effects noted only at 640 mg kg BW daily: some deaths, less drinking water ingested, decreased volume of urine, significant increase in urea, and	21

	decrease in creatinine. Tissue residues were significantly elevated over controls in high dose group at 3 months: 60 mg Zn/kg FW in liver (20 mg Zn/kg FW in controls), 38 mg Zn/kg FW in kidney (16 mg Zn/kg FW), 330 mg Zn/kg FW in bone (92 mg Zn/kg FW), 21 mg Zn/kg FW in blood (3 mg Zn/kg FW), and 36 mg Zn/kg FW in spleen (16 mg Zn/kg FW). Residues were the same as controls in brain, lung, and muscle	
800 mg Zn/L for 30 days	Liver alterations	8
Intragastric administration		
180 g adults given single dose of 500 mg, equivalent to 2,777 mg/kg BW	Serum zinc reached a maximum of 3.5 mg Zn/L after 60 min and returned to normal (1.6 mg/L) within 24 h	22
165 g adults given 500 mg daily for up to 30 days, equivalent to 3,030 mg Zn/kg BW daily	Serum zinc after 7, 14, or 80 days was 1.9, 2.2, and 2.1 mg/L, respectively; 10 days after last dose, serum zinc was normal	22
Single oral dose, 350-800 mg Zn/kg BW	Acute oral LD50	2,21
Domestic pig, *Sus* sp.		
Weanlings fed diet containing 1,000 mg Zn/kg feed for 30 days	Decreased growth rate and food intake, arthritis, lameness, and inflammation of the gastrointestinal tract	13

[a] 1. Miller et al. 1989; 2. NAS 1979; 3. Gaynor et al. 1988; 4. Wentink et al. 1985; 5. Latimer et al. 1989; 6. Robinette 1990; 7. Ogden et al. 1988; 8. PHS 1989; 9. Lain et al. 1985; 10. Goyer 1986; 11. Bridges 1990; 12. Sammon and Roberts 1988; 13. Elinder 1986; 14. Prasad 1979; 15. Malta et al. 1981; 16. Straube et al. 1980; 17. Reece et al. 1986; 18. Schlosberg 1976; 19. Allen et al. 1983; 20. Saxena et al. 1989b; 21. Llobet et al. 1988a; 22. Castellano et al. 1988; 23. Kreppel et al. 1988.

Zinc fed to adult male rats at 500 mg/kg diet for 3 weeks or longer harms the testes and other male accessory organs; effects are a direct result of zinc cytotoxicity from transfer across the blood-testes barrier (Saxena et al. 1989a). Elevated dietary zinc also depresses bone calcium levels and increases fecal calcium loss in rats (Greger 1989). Increases in serum zinc levels of rats after acute zinc overload is due mainly to increases in the zinc bound to the albumin fraction and secondarily to that bound to the globulin fraction (Castellano et al. 1988). Albumin may play a new physiological role by fitting its binding capacity to serum zinc levels, essentially binding all excess zinc that arrives in the blood (Castellano et al. 1988).

Zinc toxicosis has been observed in humans and livestock after ingestion of acidic foods or drink prepared and stored in galvanized containers (Latimer et al. 1989). Symptoms occur within 24 h and include nausea, vomiting, diarrhea and abdominal cramps. The emetic dose for zinc in humans was estimated at 225-450 mg (3.2-6.4 mg Zn/kg BW), equivalent to 1-2 g zinc sulfate (Elinder 1986). Zinc poisoning in dogs is well documented as a result of ingestion of galvanized metal objects, calamine lotion, skin and sunblock preparations containing zinc oxide, staples, nails, fertilizers, some paints, products containing zinc undecylenate, metallic hardware items with a high zinc content, nuts on certain types of animal transport cages, and pennies (Latimer et al. 1989; Robinette 1990). The propensity of some individuals to throw pennies (U.S. coinage) into animal cages while visiting zoos and animal parks should be considered a potential source of zinc poisoning in captive animals. Pennies minted before 1982 contain 95% copper and 5% zinc; however, copper-clad pennies minted after 1981 contain 97.6% zinc and 2.4% copper (Ogden et al. 1988).

Humans given zinc supplements should be aware of possible complications attendant to their use (Fosmire 1990). Low intakes of 100-300 mg zinc daily in excess of the recommended dietary allowance of 15 mg zinc daily may produce induced copper deficiency, impaired immune function, and disrupted blood lipid profiles. Patients treated with zinc supplements (150 mg daily) to control sickle cell anemia and nonresponsive celiac disease developed a severe copper deficiency in 13 to 23 months; normal copper status was restored by cessation of zinc supplements and increased dietary copper (Fosmire 1990).

Because of false positives, zinc may confound interpretation of the paralytic shellfish poisoning mouse bioassay, one of the routine tests used to measure shellfish safety for human consumption. For example, mice injected intraperitoneally with extracts of healthy oyster tissues showed extreme weakness, a drop in body temperature, cyanosis, and some deaths (McCulloch et al. 1989). The threshold for a toxic paralytic shellfish poisoning response corresponds to a drained tissue zinc level >900 mg/kg FW, and this overlaps the zinc concentration range of 230-1,650 mg/kg FW (1,900-9,400 mg/kg DW) recorded in healthy oyster soft tissues (McCulloch et al. 1989).

Recommendations

For growing agricultural crops: (1) sewage sludge may be applied to soils if total zinc content does not exceed 150 to 560 kg/ surface hectare (Table 9); (2) a maximum permissible extractable soil zinc concentration of 23 mg/kg DW is recommended, according to Soviet agronomists (Beyer 1990); and (3) seedlings of oak (*Quercus* spp.) and red maple (*Acer rubrum*) will eventually die in culture medium containing >100 mg Zn/kg (Buchauer 1971), although total zinc concentrations for global crop production routinely exceed 100 mg/kg DW soil (Table 9). Research is needed in standardized methodology for measurement of bioavailable (i.e., extractable) soil zinc and on its relation to other soil measurements such as total zinc and depth of cultivation in the case of surface application.

Table 9. Proposed zinc criteria for the protection of natural resources and human health.

Resource, criterion, and other variables	Effective zinc concentration	Reference[a]
Crop plants		
Sewage sludge applied to agricultural soils		
Europe, acceptable	150-<300 kg/ha at pH 6.0-7.0	1
Florida		
Maximum permissible	205 kg/ha	1
Unacceptable	>10,000 mg/kg dry weight (DW)	1
Oregon[b], Wisconsin[b], acceptable	250 - < 1,000 kg/ha	1
Vermont[b], acceptable	280 - < 1,120 kg/ha	1
Maryland[b], Massachusetts[b], acceptable	280-<560 kg/hg	1
Minnesota[b], Missouri[b], acceptable	280-<1,120 kg/ha	1
Illinois, maximum	560 kg/ha	1
Soils		
Soviet Union, maximum permissible	23 mg/kg DW, extractable by ammonium acetate buffer at pH 4.8	1
Alberta, Canada, for growing livestock forage	<100 mg/kg DW	1
Quebec, Canada		

Background	200 mg/kg DW	1
Marginal	500 mg/kg DW	1
Unacceptable	>3,000 mg/kg DW	1
Netherlands		
Background	200 mg/kg DW	1
Marginal	500 mg/kg DW	1
Unacceptable	>3,000 mg/kg DW	1
Ontario, Canada, acceptable	<220 mg/kg DW	1
Germany, acceptable	<300 mg/kg DW	2
New Jersey, goal	<350 mg/kg BW	1
New York, acceptable		
Agricultural soils	168-<250 kg/ha DW	1
Forest soils	<560 kg/ha DW	1
Terrestrial Invertebrates		
Earthworms		
High accumulations, but otherwise safe	97 mg/kg DW soil	3
Adverse effects	>400 mg/kg DW soil	3
Slugs, diet, adverse effects	>300 mg/kg DW	4
Freshwater aquatic life		
Water		
Total recoverable zinc		
60 mg $CaCO_3$/L	47 µg/L, 24 h average; not to exceed 180 µg/L at any time	5
100 mg $CaCO_3$/L	47 µg/L, 24 h average; not to exceed 320 µg/L at any time	5
200 mg $CaCO_3$/L	47 µg/L, 24 h average; not to exceed 570 µg/L at any time	5
Acid-soluble zinc[c]	4-day average concentration not to exceed the numerical value e ((0.8473 [ln] hardness)+ 0.7614) more than once every 3 years on average; 1-h concentration not to exceed e((0.8473 [ln] hardness)+ 0.8604) more than once every 3 years on average. See below for examples	6
50 mg $CaCO_3$/L	4-day average not to exceed 59 µg/L; 1-h average not to exceed 65 µg/L	6
100 mg $CaCO_3$/L	4-day average not to exceed 110 µg/L; 1-h average not to exceed 120 µg/L	6
200 mg $CaCO_3$/L	4-day average not to exceed 190 µg/L; 1-h average not to exceed 210 µg/L	6
Adverse effects, most sensitive species		
Brown trout, *Salmo trutta*, embryos and fry	4.9-19.6 µg/L	7

Daphnid, *Daphnia magna*	5-14 µg/L	6
Rainbow trout, *Oncorhynchus mykiss*	5.6-10 µg/L	5,6,8
Narrow-mouthed toad, *Gastrophryne carolinensis*, embryos	10 µg/L	6
Daphnid, *Daphnia galeata mendotae*	15-30 µg/L	6
Freshwater sponge, *Ephydatia fluviatilis*	26 µg/L	9
Mayfly, *Epeorus latifolium*	30 µg/L	10
Midge, *Tanytarsus dissimilis*	37 µg/L	5,6
Atlantic salmon, *Salmo salar*	50 µg/L	6
Cladoceran, *Ceriodaphnia reticulata*	51 µg/L	6
Flagfish, *Jordanella floridae*	51 µg/L	6
Diet		
Channel catfish, *Ictalurus punctatus*		
Minimum	20 mg/kg DW	11
Recommended	150-200 mg/kg DW	11
Rainbow trout, *Oncorhynchus mykiss*		
Minimum	10-30 mg/kg DW; 15-30 mg/kg fresh weight (FW)	12,13
Adequate	90 mg/kg FW	13
Sediments		
Great Lakes		
Safe	<90 mg/kg DW	1
Marginal	90-200 mg/kg DW	1
Unacceptable	>200 mg/kg DW	1
Wisconsin and Ontario, for Great Lakes sediments dredged from harbors and for disposal in water	<100 mg/kg DW	1
Marine aquatic life		
Seawater		
Total recoverable zinc	58 µg/L, 24-h average; not to exceed 170 µg/L at any time	5
Acid-soluble zinc[c] (20)	4-day average concentration does not exceed 86 µg/L more than once every 3 years on average; 1-h average concentration does not exceed 95 µg/L more than once every 3 years on average	6
No adverse effect, most species		
Algae	<1,400 µg/L	14
Molluscs	<54 µg/L	15
Crustaceans	<230 µg/L	15
Adverse effects, most sensitive species		
Brown algae, *Fucus serratus*	8.8-9.5 µg/L	6

Copepod, *Tisbe holothuriae*	10 µg/L	16
Pacific oyster, *Crassostrea gigas*, larvae	10-20 µg/L	6
Alga, *Rhizosolenia* spp.	15-25 µg,/L	8
Diatom, *Schroederella schroederi*	19 µg/L	6
Diatom, *Skeletonema costatum*	19.6 µg/L	17
Dinoflagellate, *Glenodinium halli*	20 µg/L	6
Purple sea urchin, *Strongylocentrotus purpuratus,* embryos	23 µg/L	6
Sand dollar, *Dendraster excentricus*	28 µg/L	6
Atlantic herring, *Clupea harengus*, embryos	50 µg,/L	6
Mud crab, *Rithropanopeus harrisii,* larvae	50 µg/L	5
Diet, fish, adequate	90 mg/kg FW	13
Tissue residues, minimum theoretical requirement for whole molluscs and crustaceans	34.5 mg/kg DW	18
Birds		
Mallard, *Anas platyrhynchos*		
Zinc-poisoned		
Diet	2,500-3,000 mg/kg DW ration	19,20,21
Single oral dose	0.64; , 517-742 mg/kg body weight (BW)	22
Birds, various, tissue concentrations		
Normal		
Liver	21-33 mg/kg DW	23
Plasma	1.3-2.0 µg/L	24
Zinc-poisoned		
Liver	75-156 mg/kg DW	23
Plasma	15.5 mg/L	24
Japanese quail, *Coturnix coturnix japonica*, safe level	25-30 mg/kg DW diet	25
Chicken, *Gallus* sp.		
Recommended daily intake	>31 mg	26
Diet		
Adverse effects, zinc deficiency	<38 mg/kg DW ration	27,28,29
Adequate	93-120 mg/kg DW ration	28,29
Excessive	>178 mg/kg DW ration	27
Toxic	>2,000 mg/kg DW ration	20,30,31
Mammals		
Cattle, *Bos* spp.		
Diet		
Soluble zinc, recommended level		

Calves	>8 mg/kg DW	20
Adults		
Beef cattle	10-30 mg/kg DW	20
Dairy cattle	40 mg/kg DW	20
Total zinc		
Marginal	25 mg/kg DW	32
Recommended	45-60 mg/kg DW	32,33
Maximum tolerated		
Calves	500 mg/kg DW	35
Adults	1,000 mg/kg DW	34,35
Toxic	>900-2,000 mg/kg DW	34,35
Tissue residues		
Liver		
Zinc-deficient	<10 mg/kg DW	32
Suboptimal	10-30 mg/kg DW	32
Optimal	30-120 mg/kg DW	32
Excessive	>120 mg/kg DW	32
Lethal	>500 mg/kg DW	34
Plasma		
Zinc-deficient	<0.66 mg/L	33
Normal	1.02 mg/L	33
Elevated	1.5 mg/L	33
Serum, zinc-deficient	<0.6 mg/L	36
Recommended daily intake		
Calves		
5 months old	3 g (25-35 mg/kg BW)	34
14-18 months old	16 g (50-80 mg/kg BW)	34
Cows	55 g (110-140 mg/kg BW)	34
Dog, *Canis familiaris*, tissue concentrations, normal versus zinc-poisoned		
Serum	0.7-1.1 versus 33 mg/L	37
Plasma	0.6-1.0 versus 16-32 mg/L	37
Urine	1.3-2.0 versus 20-25 mg/L	37
Liver	17-32 versus 369 mg/kg FW	37
Kidney	9-23 versus 295 mg/kg FW	37
Guinea pig, *Cavia* spp.		
Air, adverse effects	0.8-4.0 mg Zn/m^3	38
Diet		
Deficient	3 mg/kg DW plus 1 mg/L drinking water	39
Adequate	3 mg/kg DW plus 15 mg/L drinking water	40
Normal	20 mg/kg DW	41

Adequate	100 mg/kg FW	39
High	200 mg/kg DW	41
Tissue concentrations, zinc deficient versus normal		
Serum	0.5 versus 1.6-2.0 mg/L	39
Liver	9.4 versus 15-17 mg/kg FW	39
Testes	9.5 versus 19-27 mg/kg FW	39
Kidney	10 versus 16-20 mg/kg FW	39
Domestic goat, *Capra* sp., diet		
Soluble zinc, recommended		
Adults	>4 mg/kg DW	20
Kids	>7 mg/kg DW	20
Total zinc		
Deficient	<15 mg/kg DW	42
Recommended	80 mg/kg DW	42
Bank vole, *Clethrionomys glareolus*, diet, recommended	30 mg/kg DW	43
Horse, *Equus caballus*		
Diet		
No adverse effects	250 mg/kg DW	44
Adverse effects	1,000 mg/kg DW	44
Daily intake, adverse effects	>90 mg/kg BW	20
Domestic cat, *Felis domesticus*, diet, adverse effects	300 mg/kg DW	20
Humans, *Homo sapiens*		
Air		
Safe levels		
Zinc chloride, fumes	<1 mg/m^3	20,38
Zinc oxide, fumes	<5 mg/m^3	28,38,45,46
Zinc and zinc oxides	5-10 mg/m^3	38
Zinc oxide, total dust	10 mg/m^3	38
Zinc oxide, fume and dust, ceiling limit	15 mg/m^3	38
Adverse effects, zinc oxides	600 mg/m^3 for 10 min	38
Daily intake		
Recommended dietary intake, assuming availability of 20%		
Children		
To age 1 year	3-6 mg	48
1-10 years	8-10 mg	48
No age specified	10 mg	2,20,26,47,49,50
Males		

Age 11-17	14-15 mg	48
Age 18+	11-15 mg	48
No age specified	15 mg	2,20,26, 47,49,50
Females		
Age 10-13	13-15 mg	48
Age 14+	11-15 mg	48
No age specified	12 mg	48
Pregnant	15-20 mg	48
Lactating	25-27 mg	47,48
Maximum safe total, adults		
Not zinc deficient	0.3-1.0 mg/kg BW	2
Zinc deficient	1 mg Zn/kg BW, oral administration	48
Adverse effects level	>160 mg (>2.3 mg/kg BW)	51
Diet		
Seafoods, safe level, Australia,	<40 mg/kg FW	14
Adverse effects		
Gastrointestinal disorders	>80 mg/kg DW diet for 6 weeks	38
Severe copper deficiency	150 mg zinc daily for 13-23 months	49
Vomiting	Single dose of 225-450 mg zinc or 1-2 g of $ZnSO_4$	49
Drinking water		
Safe level	5 mg/L	2,20,38
Adverse effects, acute GI distress	>280 mg/L	20
Intravenous injection, adverse effects	23 mg/kg BW daily	52
Soils, Canada, nonhazardous to human health		
Ontario, residential, parkland, commercial, industrial	<800 mg/kg DW	1
Alberta, noncrop uses	<700 mg/kg DW	1
Tissue residues		
Serum		
Normal	0.5-1.29 mg/L	38
No toxic effects	1.92 mg/L	38
Plasma		
Zinc-deficient	0.4-0.6 mg/L	45
Normal	0.7-1.1 mg/L	48
GI disturbances	1.51 mg/L	38
Rhesus monkey, *Macaca mulatta,* diet		
Deficient	4 mg/kg DW	52
Adequate	100 mg/kg DW	53
Mouse, *Mus* spp.		

Diet		
Zinc-deficient	<5 mg/kg DW	54
Zinc-adequate	36.5 mg/kg DW	54
Tolerated	100 mg/kg DW	54
Tolerated	682 mg/kg DW for 13 weeks (107 mg/kg BW)	55
Harmful	500 mg/kg DW for 3 months	38
Harmful	6,820 mg/kg DW	55
Fatal	30,000 mg/kg DW for 13 weeks	38
Drinking water, adverse effects	300 mg/L	38
Tissue residues		
Blood		
Deficient	0.7 mg/L	56
Normal	1.0-1.1 mg/L	56
Liver		
Deficient	12 mg/kg FW	56
Normal	17-19 mg/kg FW	56
European ferret, *Mustela putorius furo*, diet		
Tolerated	500 mg/kg DW	57
Fatal	1,500 mg/kg DW	38
Mink, *Mustela vison*, diet		
Zinc-deficient	4.1 mg/kg FW	58
Adequate	35-45 mg/kg FW; 100-150 mg/kg DW	58
Domestic sheep, *Ovis aries*		
Diet		
Soluble zinc, adequate		
Adults	>4 mg/kg DW	20
Lambs	>7 mg/kg DW	20
Total zinc		
Adults, adequate	33 mg/kg DW	59,60
Lambs		
Adequate	124-130 mg/kg DW	59
Harmful	>1,000 mg/kg DW	20,61,62
Recommended daily intake	>18 mg	26
Tissue residues		
Feces		
Normal	158 mg/kg DW	61
Zinc-poisoned	4,900 mg/kg DW	61
Kidney		
Normal	84-150 mg/kg DW	61,63
Elevated	>180 mg/kg DW	61
Zinc-poisoned	274-760 mg/kg DW	61,63
Liver		

Normal	144-165 mg/kg DW	61,63
Elevated	>250 mg/kg DW	61
Zinc-poisoned	463-650 mg/kg DW	61,63
Pancreas		
Normal	88 mg/kg DW	61
Zinc-poisoned	752 mg/kg DW	61
Laboratory white rat, *Rattus* sp.		
Diet		
Soluble zinc, recommended	15 mg/kgDW	20
Total zinc		
Zinc-deficient	<12 mg/kg DW	47
Adequate	76 mg/kg DW	64
Adverse effects	>500 mg/kg DW	52
Fetotoxic	>4,000 mg/kg DW	20,38
Daily intake		
Tolerated	320 mg/kg BW	65
Harmful	640 mg/kg BW	65
Single oral dose, harmful	>350 mg/kg BW	20,65
Domestic pig, *Sus* sp.		
Diet		
Soluble zinc, safe levels		
Normal	14-20 mg/kg DW	20
Cassava-rice-bran	>40 mg/kg DW	20
Soy base	50 mg/kg DW	20
Total zinc, harmful	1,000 mg/kg DW	47
Recommended daily intake	>20 mg	26

[a] 1. Beyer, 1990; 2. Leonard and Gerber 1989; 3. Beyer et al. 1987; 4. Marigomez et al. 1986; 5. EPA 1980; 6. EPA 1987; 7. Sayer et al. 1989; 8. Spear 1981; 9. Francis and Harrison 1988; 10. Hatakeyama 1989; 11. Gatlin et al. 1989; 12. Bettger et al. 1987; 13. Spry et al. 1988; 14. Eisler 1981; 15. Sprague 1986; 16. Verrioposulos and Hardouvelis 1988; 17. Vymazal 1986; 18. White and Rainbow 1985; 19. Kazacos and Van Vleet 1989; 20. NAS 1979; 21. Gasaway and Buss 1972; 22. Grandy et al. 1968; 23. Reece et al. 1986; 24. Morris et al. 1986; 25. Harland et al. 1975; 26. Ellen et al. 1989; 27. Stahl et al. 1989a; 28. Blamberg et al. 1960; 29. Westmoreland and Hoekstra 1969; 30. Stahl et al. 1990; 31. Oh et al. 1979; 32. Binnerts 1989; 3,3. Ramachandra and Prasad 1989; 34. Wentink et al. 1985; 35. Miller et al. 1989; 36. Damir et al. 1988; 37. Robinette 1990; 38. PHS 1989; 39. Gupta et al.. 1988; 40. Apgar and Everett 1988; 41. Scelsi et al. 1989; 42. Chhabra and Arora 1989; 43. Wlostowski et al. 1988; 44. Bridges 1990; 45. Goyer 1986; 46. Lain et al. 1985; 47. Elinder 1986; 48. Casoy and Hambidge 1980; 49. Fosmire 1990; 50. Sternlieb 1988; 51. Sammon and Roberts 1988; 52. Saxena et al. 1989b; 53. Golub et al. 1988; 54. Mackay-Sire and Dreosti 1989; 55. Malta et al. 1981; 56. Tone et al. 1988; 57. Straube et al. 1980; 58. Mejborn 1989; 59. Vergnes et al. 1990; 60. Khandaker and Telfer 1990; 61. Allen et al. 1983; 62. Ogden et al. 1988; 63. Schlosberg 1976; 64. Ferreira et al. 1989; 65. Llobet et al. 1988a.

[b] Higher values permissible for soils with higher cation exchange capacity (Beyer 1990).

[c] Zinc that passes through a 0.45 µm membrane filter after acidification to pH 1.5-2.0 with nitric acid (EPA 1987).

[d] Higher concentrations recommended to compensate for reduced bioavailability caused by excess calcium and phytate in diet (Gatlin et al. 1989).

Data on zinc hazards to terrestrial invertebrates are limited; however, sensitive species are adversely affected at dietary concentrations >300 mg Zn/kg or at soil concentrations >400 mg/kg (Table 9).

Water quality criteria protection of aquatic life should include both total recoverable zinc and acid-soluble zinc (EPA 1980, 1987). For example, if total recoverable zinc is substantially above the proposed criteria and acid-soluble zinc is below the limit, there is cause for concern (EPA 1987). To protect about 95% of freshwater animal genera, EPA recommends water concentrations that average <47 µg total recoverable zinc per liter, not to exceed 180 µg/L at any time in soft water (i.e., <50 mg $CaCO_3$/L), or a mean concentration of 59 µg acid soluable zinc per liter, not to exceed 65 µg/L at any time in soft water (Table 9). These criteria are unsatisfactory because lower ambient zinc concentrations between 5 and 51 µg/L clearly have significant negative effects on growth, survival, and reproduction of important species of freshwater fish, amphibians, insects, sponges, and crustaceans (Table 9). Some downward modification seems necessary in the current proposed zinc criteria for freshwater aquatic life protection.

To protect important species of marine animals, EPA recommends that total recoverable zinc in seawater should average <58 µg/L and never exceed 170 µg/L; for acid-soluble zinc, these values are <86 and 95 µg/L (Table 9). As was the case for freshwater biota, there is a growing body of evidence (Table 9) demonstrating that many species of marine plants, crustaceans, molluscs, echinoderms, and fish are adversely affected at ambient zinc concentrations between 9 and 50 µg/L or significantly below the current proposed criteria for marine life protection.

Effects of zinc deficiency were produced experimentally in freshwater sponges at <0.65 µg Zn/L (Francis and Harrison 1988), in rainbow trout fed diets containing <15 mg Zn/kg FW (Spry et al. 1988), in certain species of marine algae at <0.7 µg Zn/L (Vymazal 1986), and in certain species of marine invertebrates at <6.5 µg Zn/L (Clapper et al. 1985a, 1985b) or <34 mg Zn/kg DW whole organism (White and Rainbow 1985). Zinc deficiency in natural aquatic ecosystems has not yet been credibly documented.

In aquatic environments, Spear (1981) spotlights three research needs: (1) development of analytical procedures for measurement of individual dissolved zinc species, notably the aquo ion and zinc chloride, and for nondissolved species that occur in natural waters; (2) separation of natural from anthropogenic influences of sediment-water interactions on flux rates with an emphasis on anoxic conditions, the role of microorganisms, and the stability of organozinc complexes; and (3) establishment of toxicity thresholds for aquatic organisms based on bioaccumulation and survival to determine the critical dose and the critical dose rate with an emphasis on aquatic communities inhabiting locales where zinc is deposited in sediments. These research needs are still valid.

Bird diets should contain 25-38 mg Zn/kg DW feed to prevent zinc deficiency effects, 93-120 mg Zn/kg DW feed for adequate to optimal growth, <178 mg Zn/kg DW feed to prevent marginal sublethal effects, and <2,000 mg Zn/kg DW feed to prevent the death of chicks and ducklings (Table 9). Extremely high dietary levels of 20 g Zn/kg ration are fed routinely to laying hens by poultry managers to force molting and to improve long-term egg preduction (Lu and Combs 1988a); in these cases, zinc preferentially accumulates in the kidney, liver, pancreas, and eggs (Verheyen et al. 1990). Much additional work now seems warranted on the role of zinc in avian nutrition and on the significance of tissue concentrations as an indicator of zinc stress.

The normal daily intake for all human age groups ranges between 8 and 14 mg (Casey and Hambidge 1980), but pregnant women require an additional 350-375 mg zinc during their pregnancy (Jameson 1980). Zinc used therapeutically in humans at >160 mg daily may have deleterious effects on copper status (Samman and Roberts 1988). Lower levels--close to the recommended daily allowance of 15 mg--are reported to interfere with iron metabolism and with high density lipoprotein cholesterol concentrations (Fosmire 1990) but this requires verification. The proposed air quality criterion for human health protection is 5 mg Zn/m^3, although this is demonstrably harmful to guinea pigs (Table 9). It is not yet known whether guinea pigs are more sensitive than humans to atmospheric zinc or if some downward modification is needed in the current zinc air quality criterion for protection of human health and presumably wildlife.

Single oral doses >350 mg Zn/kg BW were fatal to rats, although doses of 320 mg/kg BW were tolerated (Table 9), suggesting a rapid breakdown in ability to regulate zinc in a relatively narrow critical threshold range. More research into zinc regulation of massive doses seems needed.

Data that link zinc concentrations in tissues with environmental zinc perturbations in mammals are rare. For example, elevated zinc concentrations were >120 mg Zn/kg DW tissue in cattle liver, >180 mg Zn/kg DW tissue in sheep kidney, and >250 mg Zn/kg DW tissue in sheep liver (Table 9). The significance of zinc residues in animal tissues is unclear. No international regulations or guidelines applicable to zinc are available (PHS 1989). No U.S. Food and Drug Administration action level or other maximum acceptable concentration exists for zinc, and therefore no Final Residue Value can be calculated (EPA 1987). This seems to be a research need of high priority.

Eating seafoods that contain high concentrations of zinc does not seem to present a threat to human health. However, oysters from Tasmania allegedly caused nausea and vomiting in some people who ate them; these oysters contained about 20 g Zn/kg FW soft parts or about 500 times more than the Australian food regulation of 40 mg/kg FW (Eisler 1981).

In mammals, large differences are evident between and within species in resistance to zinc poisoning and in sensitivity to zinc nutritional needs (Table 9). Adverse effects of excess dietary zinc occurred in sensitive species at 80 mg Zn/kg DW (in humans) and 300 mg Zn/kg DW (in cats); other tested species were significantly more resistant. Daily intake rates considered harmful over long periods ranged from about 2.3 mg/kg BW in humans to >90 mg/kg BW in horses. Dietary loadings that optimally prevented zinc deficiency were 30 mg Zn/kg DW diet for bank voles, 33 mg Zn/kg DW diet for adult sheep (124-130 mg Zn/kg DW diet for lambs), 37 mg Zn/kg DW diet for mice, 45-60 mg Zn/kg DW diet for cattle, 76 mg Zn/kg DW diet for rats, 80 mg Zn/kg DW diet for goats, 100 mg Zn/kg DW diet for monkeys, and 150 mg Zn/kg DW diet for minks; recommended daily intake ranged from about 0.2 mg/kg BW in humans to 110-140 mg/kg BW in cattle (Table 9). More research with animals of various age and nutrient status is needed to determine the interaction effects of zinc with proteins, calcium, chloride, and other trace elements and on the long-term consequences of nutrient interactions (Gregor 1989).

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