

AMBIENT WATER QUALITY CRITERIA FOR
ZINC

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FOREWORD

Section 304 (a)(1) of the Clean Water Act of 1977 (P.L. 95-217), requires the Administrator of the Environmental Protection Agency to publish criteria for water quality accurately reflecting the latest scientific knowledge on the kind and extent of all identifiable effects on health and welfare which may be expected from the presence of pollutants in any body of water, including ground water. Proposed water quality criteria for the 65 toxic pollutants listed under section 307 (a)(1) of the Clean Water Act were developed and a notice of their availability was published for public comment on March 15, 1979 (44 FR 15926), July 25, 1979 (44 FR 43660), and October 1, 1979 (44 FR 56628). This document is a revision of those proposed criteria based upon a consideration of comments received from other Federal Agencies, State agencies, special interest groups, and individual scientists. The criteria contained in this document replace any previously published EPA criteria for the 65 pollutants. This criterion document is also published in satisfaction of paragraph 11 of the Settlement Agreement in Natural Resources Defense Council, et. al. vs. Train, 8 ERC 2120 (D.D.C. 1976), modified, 12 ERC 1833 (D.D.C. 1979).

The term "water quality criteria" is used in two sections of the Clean Water Act, section 304 (a)(1) and section 303 (c)(2). The term has a different program impact in each section. In section 304, the term represents a non-regulatory, scientific assessment of ecological effects. The criteria presented in this publication are such scientific assessments. Such water quality criteria associated with specific stream uses when adopted as State water quality standards under section 303 become enforceable maximum acceptable levels of a pollutant in ambient waters. The water quality criteria adopted in the State water quality standards could have the same numerical limits as the criteria developed under section 304. However, in many situations States may want to adjust water quality criteria developed under section 304 to reflect local environmental conditions and human exposure patterns before incorporation into water quality standards. It is not until their adoption as part of the State water quality standards that the criteria become regulatory.

Guidelines to assist the States in the modification of criteria presented in this document, in the development of water quality standards, and in other water-related programs of this Agency, are being developed by EPA.

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CRITERIA DOCUMENT

ZINC

CRITERIA

Aquatic Life

For total recoverable zinc the criterion to protect freshwater aquatic life as derived using the Guidelines is 47 $\mu\text{g/l}$ as a 24-hour average and the concentration (in $\mu\text{g/l}$) should not exceed the numerical value given by $e^{(0.83[\ln(\text{hardness})]+1.95)}$ at any time. For example, at hardnesses of 50, 100, and 200 mg/l as CaCO_3 the concentration of total recoverable zinc should not exceed 180, 320, and 570 $\mu\text{g/l}$ at any time.

For total recoverable zinc the criterion to protect saltwater aquatic life as derived using the Guidelines is 58 $\mu\text{g/l}$ as a 24-hour average and the concentration should not exceed 170 $\mu\text{g/l}$ at any time.

Human Health

Sufficient data are not available for zinc to derive a level which would protect against the potential toxicity of this compound. Using available organoleptic data, for controlling undesirable taste and odor quality of ambient water, the estimated level is 5 mg/l . It should be recognized that organoleptic data as a basis for establishing a water quality criteria have limitations and have no demonstrated relationship to potential adverse human health effects.

INTRODUCTION

Zinc is a bluish-white metal which dissolves readily in strong acids. Its principal uses include electroplating and the production of alloys. Zinc is never found free in nature, but occurs as the sulfide, oxide, or carbonate (Lange, 1956).

Zinc has an atomic number of 30 and its atomic weight is 65.38 (Weast, 1977). The chemistry of zinc is similar to that of cadmium, which is directly below it in the periodic table (Cotton and Wilkinson, 1972). In aqueous solution, zinc always has a valence of +2, and it exhibits amphoteric properties, dissolving in acids to form hydrated Zn(II) cations and in strong bases to form zincate anions [probably $\text{Zn}(\text{OH})_4^{2-}$]. Compounds of zinc with the common ligands of surface waters are soluble in neutral and acidic solutions, so that zinc is readily transported in most natural waters and is one of the most mobile of the heavy metals. The geochemistry of zinc in surface water has been extensively reviewed by Hem (1972). Since the divalent zinc ion does substitute to some extent for magnesium in the silicate minerals of igneous rocks, weathering of this zinc-containing bedrock gives rise to Zn^{+2} in solution whereupon the hydrated cation remains dominant to pH values of about 9. Zinc forms complexes with a variety of organic and inorganic ligands, but these compounds are sufficiently soluble to prevent their becoming a limiting factor for the solubility of the small concentrations of zinc found in most aquatic environments. Adsorption on clay minerals, hydrous oxides, and organic matter is a more probable limiting mechanism.

Most of the zinc introduced into the aquatic environment is partitioned into the sediments by sorption onto hydrous iron and manganese oxides, clay

minerals, and organic materials. Precipitation of the sulfide is an important control on the mobility of zinc in reducing environments, and precipitation of the hydroxide, carbonate, or basic sulfate can occur where zinc is present in high concentrations. Formation of complexes with organic and inorganic ligands can increase the solubility of zinc and probably increases the tendency for zinc to be adsorbed.

Sorption of zinc by hydrous metal oxides, clay minerals, and organic materials is probably the dominant fate of zinc in the aquatic environment. The tendency of zinc to be sorbed is affected not only by the nature and concentration of the sorbent but by pH and salinity as well. In a study of heavy metal adsorption by two oxides and two soils, zinc was completely removed from solution when pH exceeded 7; below pH 6, little or no zinc was adsorbed. Addition of inorganic complexing ligands enhanced the affinity for adsorption (Huang, et al. 1977).

Helz, et al. (1975) found that zinc is desorbed from sediments as salinity increases. This phenomenon, which is exhibited by many of the other metals as well, is apparently due to displacement of the adsorbed zinc ions by alkali and alkaline earth cations which are abundant in brackish and saline waters. In summary, sorption is the dominant fate process affecting zinc, and it results in enrichment of suspended and bed sediments relative to the water column. Variables affecting the mobility of zinc include the concentration and composition of suspended and bed sediments, dissolved and particulate iron and manganese concentrations, pH, salinity, concentration of complexing ligands, and the concentration of zinc.

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Aquatic Life Toxicology*

INTRODUCTION

Acute toxicity tests have been conducted with thirty species of freshwater animals, and the median toxicity values range from 90 to 58,100 $\mu\text{g/l}$. Chronic tests with six species have resulted in chronic values from 47 to 852 $\mu\text{g/l}$. With nine different plant species the results ranged from 30 to 67,700 $\mu\text{g/l}$.

The zinc data base for saltwater organisms includes the results of acute toxicity tests with twenty-one species of invertebrates and three species of fishes. Zinc was acutely toxic to the mummichog at 83,000 $\mu\text{g/l}$ and at 166 $\mu\text{g/l}$ to the hard-shelled clam. A chronic value of 166 $\mu\text{g/l}$ is available from a life-cycle test with a mysid shrimp, and residue data are reported for five species of algae and nine species of invertebrates. Decreased growth of various plants was reported at concentrations ranging from 50 to 25,000 $\mu\text{g/l}$.

Zinc is a common trace constituent of natural waters and is a required trace element in the metabolism of most organisms. The uptake of zinc from the environment, either via ingestion or absorption, must exceed some minimum rate in order for an organism to function properly. Whether any waters are deficient in zinc content from the standpoint of the existing biota is not clear, but the question is probably moot with regard to water quality criteria for zinc.

Above some theoretical minimum concentration of zinc in water, there exists a range of zinc concentrations which is readily tolerated through

*The reader is referred to the Guidelines for Deriving Water Quality Criteria for the Protection of Aquatic Life and Its Uses in order to better understand the following discussion and recommendation. The following tables contain the appropriate data that were found in the literature, and at the bottom of each table are calculations for deriving various measures of toxicity as described in the Guidelines.

each organism's capacity to regulate the uptake, internal distribution, and excretion of zinc. This range undoubtedly varies among individuals, species, and larger phylogenetic groups. In addition, this ability, and hence the tolerated range, probably varies with the range of zinc concentrations to which the various populations have been historically exposed and acclimated. Thus, biological variability in zinc tolerance should be expected to occur based on phylogenetic differences and historic exposure patterns, both short-term and geologic in scale.

Compounding the problem of defining biologically safe zinc concentrations is the occurrence of many different forms of zinc in surface waters. Zinc can occur in both suspended and dissolved forms. Dissolved zinc may occur as the free (hydrated) zinc ion or as dissolved complexes and compounds with varying degrees of stability and toxicity. Some forms of suspended (undissolved) zinc may be readily dissolved following minor changes in water chemistry. Other suspended zinc may be reversibly sorbed onto suspended solids or, conversely, almost irreversibly included in suspended mineral particles.

Paramount to the question of zinc toxicity are the physical and chemical state of the zinc, the toxicity of each form of the zinc, and the degree of interconversion to be expected among the various forms. All zinc forms are presumably nontoxic unless they can be sorbed or bound by biological materials. Conversely, all zinc forms are potentially toxic if they can be sorbed or bound by biological tissues. Most likely, zinc will not be sorbed or bound unless it is dissolved, but some solution of the zinc may reasonably be expected to occur in the alimentary canal following ingestion of particulates containing undissolved zinc. Thus, the toxicity of undissolved zinc to any organism probably depends on feeding habits, with the result

that plants and most fish would presumably be relatively unaffected by suspended zinc, but many invertebrates could be adversely affected by ingestion of sufficient quantities of particulates containing zinc.

A complex array of data concerning an assortment of toxic responses to zinc is one result of the physical, chemical, and biological variability described. However, by evaluating the toxicity on a species-by-species basis and by considering several water chemistry parameters, the information can be simplified.

The toxicity of zinc, as well as other heavy metals, is reported to be influenced by a number of chemical factors including calcium, magnesium, hardness, pH, and ionic strength. These factors appear to affect the toxicity of zinc either by influencing the proportion of available zinc or by inhibiting the sorption or binding of available zinc by biological tissues. In freshwater, zinc appears to be less toxic at high hardness levels for a variety of reasons, such as:

- 1) The ions contributing to hardness, primarily calcium and magnesium, are divalent and compete with zinc, which is also divalent for sites of uptake and binding in biological tissues;
- 2) Harder waters have higher ionic strengths due to the greater quantity of charged ions (primarily mono- and divalent cations and anions) in solution, and these ions electrostatically inhibit the ability of other ions (including zinc) to approach the absorption or binding sites of the organisms. Basically, zinc ions have lower activity in harder waters; and
- 3) Generally, harder waters have higher alkalinites and higher pH values. Insoluble, and possibly soluble, zinc carbonate and hydroxide compounds can form which are not sorbed by many organisms. Changes in

hardness, pH, and alkalinity will cause corresponding changes in the toxicity of the zinc in the water.

Hardness, on the other hand, may have scant relationship to the amount of zinc sorbed to or included in particulate material or bound to organic chemicals. Nevertheless, hardness appears to be the single best chemical parameter to reflect the variation in zinc toxicity induced by differences in general water chemistry.

However, water quality criteria for freshwater developed with hardness as the sole physical-chemical variable may be lower than ambient total zinc levels in some surface waters of the United States. This may result, in part, from the current inability to correlate quantitatively the effects on zinc toxicity of physical-chemical factors other than hardness and those factors such as ionic strength, pH, and alkalinity which are qualitatively related to hardness. Alternatively, where zinc levels exceed criteria, the zinc may be harming the biota, or the biota may have evolved as a zinc-resistant population. The actual situation must be evaluated based on the biological, chemical, and physical factors just discussed.

Of the analytical measurements currently available, a water quality criterion for zinc is probably best stated in terms of total recoverable zinc, because of the variety of forms of zinc that can exist in bodies of water and the various chemical and toxicological properties of these forms. The forms of zinc that are commonly found in bodies of water and are not measured by the total recoverable procedure, such as the zinc that is a part of minerals, clays, and sand, probably are forms that are less toxic to aquatic life and probably will not be converted to the more toxic forms very readily under natural conditions. On the other hand, forms of zinc that are commonly found in bodies of water and are measured by the total recoverable pro-

cedure, such as the free ion, and the hydroxide, carbonate, and sulfate salts, probably are forms that are more toxic to aquatic life or can be converted to the more toxic forms under natural conditions.

Because the criteria are derived on the basis of tests conducted on soluble inorganic salts of zinc, the total zinc and total recoverable zinc concentrations in the tests would probably be about the same and a variety of analytical procedures would produce about the same results. Except as noted, all concentrations reported herein are expected to be essentially equivalent to total recoverable zinc concentrations. All concentrations are expressed as zinc, not as the compound.

EFFECTS

Acute Toxicity

Zinc produces acute toxicity to freshwater organisms over a range of concentrations from 90 to 58,100 $\mu\text{g/l}$ (Table 1). The range of acute median effect concentrations is similar for freshwater fish and invertebrates, with ranges of 90 to 40,900 and 100 to 58,100 $\mu\text{g/l}$, respectively. A portion of this range is due to hardness related factors, and the remainder is due to species differences and other biological and physical-chemical factors.

Within the larger data sets for individual fish species, especially those for rainbow trout and fathead minnow, the lower LC_{50} values at a given hardness were obtained using younger, smaller fish. Also, acute toxicity tests conducted by Cairns, et al. (1978) with both Daphnia magna and Daphnia pulex at 5, 10, 15, 20 and 25°C (Tables 1 and 6) showed that acute toxicity increased as temperature increased. The value at 20°C in Table 1 was used in the calculation of the species mean acute intercept because acute and chronic tests with daphnids are usually conducted at this temperature.

An exponential equation was used to describe the observed relationship of the acute toxicity of zinc to hardness in freshwater. Least squares regression of the natural logarithms of the acute values on the natural logarithms of hardness was used to calculate slopes for 10 species (Table 1). Five of the slopes were significant, two were not significant, and the other three could not be tested because only two values were available. The arithmetic mean (0.83) of the five significant slopes was used with the geometric mean toxicity value and hardness for each species to obtain a logarithmic intercept for each of the 29 freshwater species for which acute values are available for zinc. The species mean acute intercept, calculated as the exponential of the logarithmic intercept, was used to compare the relative acute sensitivities (Table 3).

Interestingly, all tests with 10 of the 12 species reported to be more resistant than bluegill (Table 3) were tested in several series of experiments reported by Rehwoldt, et al. (1971, 1972, 1973) conducted in Hudson River water. Whether the water reduced the toxicity of the zinc or whether the species tested were really more resistant cannot be determined. Many of the invertebrates tested by Rehwoldt and his co-workers are known to be generally resistant to heavy metals, so species resistance is a likely explanation. One species tested by Rehwoldt, et al. (1971, 1972), the striped bass, was rather sensitive to zinc, but other investigators obtained acute values which were quite a bit lower for this species.

A freshwater Final Species Acute Intercept of 7.02 $\mu\text{g/l}$ was obtained for zinc using the species mean acute intercepts listed in Table 3 and the calculation procedures described in the Guidelines. Thus the Final Acute Equation is $e^{(0.83[\ln(\text{hardness})] + 1.95)}$.

Acute toxicity data for zinc are available for 21 species of saltwater invertebrates (Table 1) and represent more than two orders of magnitude dif-

ference in sensitivity. Larval molluscs were the most sensitive invertebrates with acute values for an oyster of 310 $\mu\text{g/l}$ and for the hard shelled clam of 166 $\mu\text{g/l}$. Acute values for adult molluscs ranged from 2,500 for the blue mussel to 7,700 for the soft-shelled clam. Zinc was acutely toxic to saltwater polychaetes over the range from 900 $\mu\text{g/l}$ for Neanthes arenaceodentata to 55,000 for Nereis diversicolor. The decapod crustaceans had 96-hour LC₅₀ values of 175 and 1,000 $\mu\text{g/l}$ for the lobster and crab, respectively. The reported acute values for copepods ranged from 290 $\mu\text{g/l}$ for Acartia tonsa to 4,090 $\mu\text{g/l}$ for Eurytemora affinis. Results from tests with two mysid shrimp showed similar values; 498 $\mu\text{g/l}$ for Mysidopsis bahia and 591 $\mu\text{g/l}$ for Mysidopsis bigelowi.

The data base for saltwater fishes contains nine values for three species of fish and three taxonomic families (Table 1). The acute values range from 2,730 for larval Atlantic silversides to 83,000 for larval mummichog. Saltwater fish were generally more resistant to acute zinc poisoning than saltwater invertebrates, although there were cases of individual overlap.

The saltwater Final Acute Value for zinc, derived from the Species Mean Acute Values listed in Table 3 using the calculations procedures described in the Guidelines, is 173 $\mu\text{g/l}$.

Chronic Toxicity

Chronic toxicity tests have been conducted with six species of freshwater organisms (Table 2). Chronic values for five species of fish ranged from 47 $\mu\text{g/l}$ for flagfish (Jordanella floridae) to 852 $\mu\text{g/l}$ for brook trout (Salvelinus fontinalis). No tests of the chronic toxicity of zinc to fish have been conducted in hard water. Four chronic toxicity tests are reported for Daphnia magna, with chronic values ranging from 47 to 136 $\mu\text{g/l}$. Surprisingly, the chronic toxicity of zinc to this daphnid appears to increase

with increasing hardness, a phenomenon which may be attributable to ingestion of precipitated zinc by Daphnia magna in hard water tests.

The 10-month chronic toxicity test conducted by Brungs (1969) provided evidence that the chronic value for the fathead minnow in hard water would be below 180 $\mu\text{g/l}$ (Table 6). In light of the 106 $\mu\text{g/l}$ chronic value obtained by Benoit and Holcombe (1978) in soft water, this strongly suggests that the chronic toxicity of zinc to fish may also be relatively unaffected by hardness. Thus, the available toxicity data indicate that hardness effects are much less dramatic for the chronic toxicity of zinc than for acute toxicity, and that the slope of the hardness-toxicity regression may be near zero or even negative for some species.

The only chronic data reported (Table 2) for a saltwater species exposed to zinc are those for the mysid shrimp, Mysidopsis bahia (U.S. EPA, 1980). In this flow-through life cycle test the number of spawns recorded at 231 $\mu\text{g/l}$ was significantly ($p<0.05$) fewer than at 120 $\mu\text{g/l}$, but the number of spawns at 59 and 120 $\mu\text{g/l}$ were not statistically significantly different from those in the control. Brood size was significantly ($p<0.05$) reduced at 231 $\mu\text{g/l}$ but not at lower concentrations. Based upon reproductive data, the lower and upper chronic endpoints were 120 and 231 $\mu\text{g/l}$, respectively, which results in a chronic value of 166 $\mu\text{g/l}$ (Table 3).

The acute-chronic ratios derived from the nine chronic tests with zinc in freshwater show a rather wide range (Table 2). Some of the range is due to the differing acute values for different life stages of the same species. Additional variation is due to differences in water quality, but in soft water the values range from less than 1 to 32. It appears that 3 would be a reasonable estimate of an acute-chronic ratio for zinc in freshwater, and this agrees with the only value available in saltwater.

An acute-chronic ratio of 3 used with the freshwater Final Acute Equation (Table 3) would result in a freshwater Final Chronic Equation of $e^{(0.83[\ln(\text{hardness})] + 0.85)}$. However, this would result in a Final Chronic Value of 55 $\mu\text{g/l}$ at a hardness of 45 and in higher values at higher hardnesses. Because 47 $\mu\text{g/l}$ is the chronic value for both a sensitive invertebrate in hard water and a medium sensitive fish in soft water, it would appear reasonable to set the freshwater Final Chronic Value at 47 $\mu\text{g/l}$ for all hardnesses. This is also supported by the data suggesting that increasing hardness does not decrease the chronic toxicity of zinc like it decreases the acute toxicity of zinc.

The saltwater Final Acute Value of 173 $\mu\text{g/l}$ divided by an acute-chronic ratio of 3 results in a saltwater Final Chronic Value of 57.7 $\mu\text{g/l}$ (Table 3).

Plant Effects

Results of zinc toxicity tests with nine species of freshwater plants are listed in Table 4. Zinc concentrations from 30 to 21,600 $\mu\text{g/l}$ have been shown to reduce the growth of various plant species. Algae appear to be more sensitive to zinc than macrophytes, with Selenastrum capricornutum the most sensitive of the tested algal species. Selenastrum sensitivity to zinc is greater in softer waters (Greene, et al. 1975), but the range of hardness values tested was limited (4 to 15 mg/l as CaCO_3). The significance of short-term growth inhibition in algae has not been established; however, the existence of growth inhibition at low zinc levels should be considered of potential ecological importance.

Data for the toxic effects of zinc to 13 species of saltwater plants are also listed in Table 4. The growth of kelp was inhibited at 100 $\mu\text{g/l}$ for Laminaria digitata (Bryan, 1969) and 250 $\mu\text{g/l}$ for Laminaria hyperborea.

(Hopkins and Kain, 1971). The giant kelp, Macrocystis pyrifera, was relatively insensitive to the effects of zinc on photosynthesis with an EC₅₀ of 10,000 µg/l.

Microalgae had a wide range of sensitivities to zinc. Growth inhibition was reported at 25,000 µg/l for the diatom, Phaeodactylum tricornutum (Jensen, et al. 1974), for Skeletonema costatum and Thalassiosira pseudonana at 200 µg/l (Braek, et al. 1976), at 100 µg/l for Gymnodinium splendens and Thalassiosira rotula (Kayser, 1977), and at 50 µg/l for Schroederella schroederi (Kayser, 1977).

No freshwater or saltwater Final Plant Value is possible because zinc concentrations were not measured in any of the toxicity tests with plants.

Residues

Table 5 contains bioconcentration factors for zinc determined with two freshwater fish species and two freshwater invertebrate species. The factors for fish were 51 and 432, and those for invertebrate species were 107 and 1,130.

Bioconcentration factors also have been determined for three species of macroalgae and six species of saltwater invertebrates (Table 5), but no data are available for saltwater fishes. The accumulation of zinc by macroalgae varied from a high of 16,600 times ambient for Fucus serratus (Young, 1975) to 1,530 times above ambient for Enteromorpha prolifera (Munda, 1979). Among invertebrate species bioconcentration factors ranged from 20 for the polychaete Nereis diversicolor (Bryan and Hummerstone, 1973) to 16,700 for the oyster Crassostrea virginica (Shuster and Pringle, 1969).

Bioconcentration factors varied considerably among the different species of bivalve molluscs; 43 was obtained with the soft-shell clam (Eisler, 1976b), 500 with the mussel (Pentreath, 1973) and 16,700 with the oyster (Shuster and Pringle, 1969). Bryan (1966) reported zinc accumulation in

crab muscle resulting in a bioconcentration factor of 8,800. Because of the variation between species and phyla it is difficult to identify specific trends between bioconcentration and phylogenetic position. Bioconcentration factors for invertebrate species are generally 500 or less except for the crab and oyster.

A Final Residue Value cannot be calculated for either fresh or saltwater because no maximum permissible tissue concentration is available.

Miscellaneous

Table 6 contains other data concerning the effects of zinc on freshwater organisms. Sprague (1968) found that rainbow trout would avoid a zinc concentration of 5.6 $\mu\text{g/l}$ in a laboratory behavior test in water with a hardness of 14 mg/l as CaCO_3 , but the ecological significance of laboratory avoidance behavior is not known. Sprague (1964b) and Sprague, et al. (1965) emphasized that laboratory avoidance thresholds are probably low estimates, because territorial or migrational motivations would be expected to cause higher thresholds for avoidance in nature. In addition, acclimation to zinc could substantially alter avoidance behavior.

Significant avoidance behavior probably will not occur in nature at zinc concentrations below those required for acute and chronic protection for the most sensitive freshwater organisms. Acclimation and territorial and migratory urges would probably counteract mild aversion to waters containing low zinc concentrations. However, the possibility that a sensitive, nonacclimated species would at least temporarily avoid a body of water with an apparently acceptable zinc concentration cannot be ruled out on the basis of existing data.

Anderson, et al. (1980) reported an average LC_{50} value of 37 $\mu\text{g/l}$ (range 26 to 54 $\mu\text{g/l}$) for the chironomid Tanytarsus dissimilis following 10-day exposure of the embryonic, hatching, and molting stages. Growth of

surviving larvae was not significantly affected. Because of the duration and nature of the test and the short life-span of the chironomid tested, this test should probably be considered equivalent to the early life stage test with fish. The sensitivity of this species is further support for a freshwater Final Chronic Value of 47 $\mu\text{g/l}$.

The data for saltwater aquatic life in Tables 1 and 6 indicate that zinc causes increasing cumulative mortality with increasing time of exposure past 96 hours. Eisler and Hennekey (1977) reported a significant increase in zinc mortality to the mummichog, sandworm, soft-shell clam, and mudsnail when exposures were extended from 4 to 7 days. Benijts-Claus and Benijts (1975) reported delayed development of crab larvae after 16 days exposure at 50 $\mu\text{g/l}$, indicating cumulative mortality to macrocrustaceans.

Data in Table 6 also indicate a relationship between salinity and acute toxicity. Herbert and Wakeford (1964) reported a decrease in the sensitivity of Atlantic salmon (smolt) and yearling rainbow trout to the acute toxicity of zinc at salinities of 3, 7, and 14 g/kg but an increase in sensitivity at 26 g/kg salinity. The relationship therefore was not linear over the range of salinities tested. Jones (1975) reported a linear increase in mortality with decreasing salinities for both the marine isopod, Idotea baltica and the euryhaline isopod, Jaera albifrons.

Molluscan larvae as a taxa are generally more sensitive than the invertebrates to zinc toxicity. Brereton, et al. (1973) reported that reduced development and inhibition of substrate attachment occurred at 125 $\mu\text{g/l}$ with the Pacific oyster, and Nelson (1972) reported that abnormal shell development occurred at 70 $\mu\text{g/l}$.

Summary

Zinc is an essential trace element which can be toxic at higher concentrations. The acute toxicity of zinc to aquatic organisms is affected by

hardness, but the chronic toxicity apparently is not. The range of acute values for freshwater organisms is from 90 to 38,100 $\mu\text{g/l}$ and is similar for fish and invertebrates. Results from chronic toxicity tests indicate a range of chronic values from 47 to 852 $\mu\text{g/l}$. Of the nine reported chronic toxicity tests, five are tests with fish in soft water and four are tests with Daphnia magna at hardnesses from 45 to 211 mg/l as CaCO_3 . Chronic zinc toxicity is relatively unaffected by hardness, with zinc possibly becoming more toxic in harder waters. Data from two tests with fathead minnows confirm the apparent inability of hardness to reduce the chronic toxicity of zinc. A chronic value of 47 $\mu\text{g/l}$ was obtained with both a sensitive invertebrate (Daphnia magna) in hard water and a medium sensitive fish (flagfish) in soft water. In addition, a 10-day LC_{50} of 37 $\mu\text{g/l}$ was obtained with a midge.

Although most plants appear to be insensitive to zinc, some values with one species were below 47 $\mu\text{g/l}$, but other values for the same species were much higher. Data on bioconcentration indicates that concentrations of zinc which do not harm sensitive freshwater organisms will not harm consumers of aquatic organisms. The possibility of avoidance of zinc at low concentrations is suggested by laboratory behavior tests with fish, but the quantitative extrapolation of these results to field situation is apparently not justified.

The saltwater acute values for zinc and fishes ranged from 2,730 $\mu\text{g/l}$ for larval Atlantic silversides to 83,000 for larval mummichog. Acute values for the invertebrate species ranged from 166 for clam larvae to 55,000 for adult polychaetes. The one chronic study conducted with the mysid shrimp produced a chronic value of 166 $\mu\text{g/l}$ resulting in an acutechronic ratio of 3.0. Plant studies with macroalgae reported growth inhibition at 100 $\mu\text{g/l}$ for Laminaria digitata. Microalgae had a wide range of sensitivi-

ties to zinc with the lowest value being 50 $\mu\text{g/l}$ for Schroederella schroederi. Bioconcentration factors were generally less than 500 for the commercially important species of invertebrates except for the factors of 16,700 and 8,800 for an oyster and crab, respectively. Zinc mortality is cumulative for exposures beyond four days. The effect of salinity on zinc toxicity appears to be non-linear with fishes and linear with invertebrate species.

CRITERIA

For total recoverable zinc the criterion to protect freshwater aquatic life as derived using the Guidelines is 47 $\mu\text{g/l}$ as a 24-hour average, and the concentration (in $\mu\text{g/l}$) should not exceed the numerical value given by $e^{(0.83[\ln(\text{hardness})]+1.95)}$ at any time. For example, at hardnesses of 50, 100, and 200 mg/l as CaCO_3 the concentration of total recoverable zinc should not exceed 180, 320, and 570 $\mu\text{g/l}$ at any time.

For total recoverable zinc the criterion to protect saltwater aquatic life as derived using the Guidelines is 58 $\mu\text{g/l}$ as a 24-hour average, and the concentration should not exceed 170 $\mu\text{g/l}$ at any time.

Table 1. Acute values for zinc

<u>Species</u>	<u>Method*</u>	<u>Chemical</u>	<u>FRESHWATER SPECIES</u>		<u>Species Mean Acute Value[#] (µg/l)</u>	<u>Reference</u>
			<u>Hardness (mg/l as CaCO₃)</u>	<u>LC50/EC50** (µg/l)</u>		
Worm, <u>Nais</u> sp.	S, M	-	50	18,400	-	Rehwoldt, et al. 1973
Snail, <u>Physa heterostropha</u>	S, U	Zinc chloride	43	900***	-	Cairns & Scheler, 1958
Snail, <u>Physa heterostropha</u>	S, U	Zinc chloride	41	600***	-	Cairns & Scheler, 1958
Snail, <u>Physa heterostropha</u>	S, U	Zinc chloride	165	3,300***	-	Cairns & Scheler, 1958
Snail, <u>Physa heterostropha</u>	S, U	Zinc chloride	178	4,400***	-	Cairns & Scheler, 1958
Cladoceran, <u>Daphnia magna</u>	S, M	Zinc chloride	45	100	-	Blesinger & Christensen, 1972
Cladoceran, <u>Daphnia magna</u>	S, M	Zinc sulfate	45	280	-	Cairns, et al. 1978
Cladoceran, <u>Daphnia magna</u>	S, M	Zinc chloride	54	334	-	Chapman, et al. Manuscript
Cladoceran, <u>Daphnia magna</u>	S, M	Zinc chloride	105	525	-	Chapman, et al. Manuscript
Cladoceran, <u>Daphnia magna</u>	S, M	Zinc chloride	196	655	-	Chapman, et al. Manuscript
Cladoceran, <u>Daphnia pulex</u>	S, M	Zinc sulfate	45	500	-	Cairns, et al. 1978
Scud, <u>Gammarus</u> sp.	S, M	-	50	8,100	-	Rehwoldt, et al. 1973
Damsel fly, Unidentified sp.	S, M	-	50	26,200	-	Rehwoldt, et al. 1973
Midge, <u>Chironomus</u> sp.	S, M	-	50	18,200	-	Rehwoldt, et al. 1973

Table 1. (Continued)

<u>Species</u>	<u>Method*</u>	<u>Chemical</u>	<u>Hardness (mg/l as CaCO₃)</u>	<u>Species Mean Acute Values** (µg/l)</u>		<u>Reference</u>
				<u>LC50/EC50# (µg/l)</u>	<u>LC50/EC50# (µg/l)</u>	
Caddisfly, Unidentified sp.	S, M	-	50	58, 100	-	Rehwoldt, et al. 1973
Rotifer, <i>Philodia acuticornis</i>	S, U	Zinc chloride	25	1,500	-	Bulkema, et al. 1974
Rotifer, <i>Philodia acuticornis</i>	S, U	Zinc sulfate	25	1,200	-	Bulkema, et al. 1974
American eel, <i>Anguilla rostrata</i>	S, m	Zinc nitrate	53	14,600	-	Rehwoldt, et al. 1971
American eel, <i>Anguilla rostrata</i>	S, M	-	55	14,500	-	Rehwoldt, et al. 1972
Coho salmon, <i>Oncorhynchus kisutch</i>	FT, M	Zinc chloride	25	905	-	Chapman & Stevens, 1978
Coho salmon, <i>Oncorhynchus kisutch</i>	FT, M	Zinc chloride	94	4,600	-	Lorz & McPherson, 1976
Sockeye salmon <i>Oncorhynchus nerka</i>	FT, M	Zinc chloride	22	749	-	Chapman, 1978a
Chinook salmon (smolt), <i>Oncorhynchus tshawytscha</i>	FT, M	Zinc chloride	24	97	-	Chapman, 1978b
Chinook salmon (parr), <i>Oncorhynchus tshawytscha</i>	FT, M	Zinc chloride	22	463	-	Chapman, 1978b
Chinook salmon (smolt), <i>Oncorhynchus tshawytscha</i>	FT, M	Zinc chloride	24	701	-	Chapman, 1978b
Cutthroat trout, <i>Salmo clarki</i>	R, M	Zinc sulfate	-	90	-	Rabe & Sappington, 1970
Rainbow trout (alevin), <i>Salmo gairdneri</i>	FT, M	Zinc chloride	22	815	-	Chapman, 1978b
Rainbow trout (smolt) <i>Salmo gairdneri</i>	FT, M	Zinc chloride	22	93	-	Chapman, 1978b

Table 1. (Continued)

<u>Species</u>	<u>Method*</u>	<u>Chemical</u>	<u>Hardness (mg/l as CaCO₃)</u>	<u>LC50/EC50** (µg/l)</u>	<u>Species Mean Acute Value** (µg/l)</u>	<u>Reference</u>
Rainbow trout (parr), <i>Salmo gairdneri</i>	FT, M	Zinc chloride	24	136	-	Chapman, 1978b
Rainbow trout, <i>Salmo gairdneri</i>	FT, M	Zinc chloride	83	1,760	-	Chapman & Stevens, 1978
Rainbow trout, <i>Salmo gairdneri</i>	R, U	Zinc phosphate	20	90	-	Garton, 1972
Rainbow trout, <i>Salmo gairdneri</i>	FT, M	Zinc sulfate	350	4,520	-	Goettl, et al. 1972
Rainbow trout, <i>Salmo gairdneri</i>	FT, M	Zinc sulfate	350	1,190	-	Goettl, et al. 1972
Rainbow trout, <i>Salmo gairdneri</i>	FT, M	Zinc sulfate	30	560	-	Goettl, et al. 1972
Rainbow trout, <i>Salmo gairdneri</i>	FT, M	Zinc sulfate	30	240	-	Goettl, et al. 1972
Rainbow trout, <i>Salmo gairdneri</i>	FT, M	Zinc sulfate	30	810	-	Goettl, et al. 1972
Rainbow trout, <i>Salmo gairdneri</i>	FT, M	Zinc sulfate	30	410	-	Goettl, et al. 1972
Rainbow trout, <i>Salmo gairdneri</i>	FT, M	Zinc sulfate	30	830	-	Goettl, et al. 1972
Rainbow trout, <i>Salmo gairdneri</i>	FT, M	Zinc sulfate	47	370	-	Holcombe & Andrew, 1978
Rainbow trout, <i>Salmo gairdneri</i>	FT, M	Zinc sulfate	47	517	-	Holcombe & Andrew, 1978
Rainbow trout, <i>Salmo gairdneri</i>	FT, M	Zinc sulfate	44	756	-	Holcombe & Andrew, 1978
Rainbow trout, <i>Salmo gairdneri</i>	FT, M	Zinc sulfate	178	2,510	-	Holcombe & Andrew, 1978

Table 1. (Continued)

<u>Species</u>	<u>Method*</u>	<u>Chemical</u>	<u>Hardness (mg/l as CaCO₃)</u>	<u>LC50/EC50** (µg/l)</u>	<u>Species Mean Acute Values*</u> (µg/l)	<u>Reference</u>
Rainbow trout, <u>Salmo gairdneri</u>	FT, M	Zinc sulfate	179	2,960	-	Holcombe & Andrew, 1978
Rainbow trout, <u>Salmo gairdneri</u>	FT, M	Zinc sulfate	170	1,910	-	Holcombe & Andrew, 1978
Rainbow trout, <u>Salmo gairdneri</u>	R, U	Zinc sulfate	5	280	-	McLeay, 1976
Rainbow trout, <u>Salmo gairdneri</u>	FT, M	Zinc sulfate	333	7,210	-	Sinley, et al., 1974
Rainbow trout, <u>Salmo gairdneri</u>	FT, M	Zinc sulfate	26	430	-	Sinley, et al., 1974
Rainbow trout, <u>Salmo gairdneri</u>	FT, M	Zinc sulfate	500	4,700	-	Solbe, 1974
Atlantic salmon, <u>Salmo salar</u>	FT, M	Zinc sulfate	14	740	-	Carson & Carson, 1972
Atlantic salmon, <u>Salmo salar</u>	FT, M	Zinc sulfate	20	600	-	Sprague, 1964a
Atlantic salmon, <u>Salmo salar</u>	FT, M	-	14	420	-	Sprague & Ramsey, 1965
Brook trout, <u>Salvelinus fontinalis</u>	FT, M	Zinc sulfate	47	1,550	-	Holcombe & Andrew, 1978
Brook trout, <u>Salvelinus fontinalis</u>	FT, M	Zinc sulfate	47	2,120	-	Holcombe & Andrew, 1978
Brook trout, <u>Salvelinus fontinalis</u>	FT, M	Zinc sulfate	44	2,420	-	Holcombe & Andrew, 1978
Brook trout, <u>Salvelinus fontinalis</u>	FT, M	Zinc sulfate	178	6,140	-	Holcombe & Andrew, 1978
Brook trout, <u>Salvelinus fontinalis</u>	FT, M	Zinc sulfate	179	6,980	-	Holcombe & Andrew, 1978

Table 1. (Continued)

<u>Species</u>	<u>Method*</u>	<u>Chemical</u>	<u>Hardness (mg/l as CaCO₃)</u>	<u>LC50/EC50#</u> <u>(µg/l)</u>	<u>Species Mean Acute Value##</u> <u>(µg/l)</u>	<u>Reference</u>	
Brook trout, <i>Salvelinus fontinalis</i>	FT, M	Zinc sulfate	170	4,980	-	Holcombe & Andrew, 1978	
Longfin dace, <i>Agosia chrysogaster</i>	R, M	Zinc sulfate	217	790	-	Lewis, 1978	
Goldfish, <i>Carassius auratus</i>	S, U	Zinc sulfate	20	6,440	-	Pickerling & Henderson, 1966	
Goldfish, <i>Carassius auratus</i>	S, U	Zinc sulfate	45	7,500	-	Cairns, et al., 1969	
Carp, <i>Cyprinus carpio</i>	S, M	Zinc nitrate	53	7,800	-	Rahwoldt, et al., 1971	
Carp, <i>Cyprinus carpio</i>	S, M	-	55	7,800	-	Rahwoldt, et al., 1972	
Golden shiner, <i>Notemigonus crysoleucus</i>	S, U	Zinc sulfate	45	6,000	-	Cairns, et al., 1969	
Fathead minnow, <i>Pimephales promelas</i>	FT, M	Zinc sulfate	46	600	-	Benolt & Holcombe, 1978	
Fathead minnow, <i>Pimephales promelas</i>	FT, M	Zinc sulfate	200	2,610	-	Broderius & Smith, 1979	
Fathead minnow, <i>Pimephales promelas</i>	FT, M	Zinc sulfate	203	8,400	-	Brungs, 1969	
Fathead minnow, <i>Pimephales promelas</i>	FT, M	Zinc sulfate	203	10,000	-	Brungs, 1969	
Fathead minnow, <i>Pimephales promelas</i>	S, U	Zinc sulfate	203	12,000	-	Brungs, 1969	
Fathead minnow, <i>Pimephales promelas</i>	S, U	-	Zinc sulfate	203	13,000	-	Brungs, 1969
Fathead minnow, <i>Pimephales promelas</i>	S, M	-	Zinc sulfate	45	3,100	-	Judy & Davies, 1979

Table 1. (Cont Inued)

<u>Species</u>	<u>Method*</u>	<u>Chemical</u>	<u>Hardness (mg/l as CaCO₃)</u>	<u>LC50/EC50** (µg/l)</u>	<u>Species Mean Acute Values# (µg/l)</u>	<u>Reference</u>
Fathead minnow, <i>Pimephales promelas</i>	FT, M	Zinc sulfate	50	12,500	-	Mount, 1966
Fathead minnow, <i>Pimephales promelas</i>	FT, M	Zinc sulfate	50	13,800	-	Mount, 1966
Fathead minnow, <i>Pimephales promelas</i>	FT, M	Zinc sulfate	100	18,500	-	Mount, 1966
Fathead minnow, <i>Pimephales promelas</i>	FT, M	Zinc sulfate	100	25,000	-	Mount, 1966
Fathead minnow, <i>Pimephales promelas</i>	FT, M	Zinc sulfate	200	29,000	-	Mount, 1966
Fathead minnow, <i>Pimephales promelas</i>	FT, M	Zinc sulfate	200	35,500	-	Mount, 1966
Fathead minnow, <i>Pimephales promelas</i>	FT, M	Zinc sulfate	50	13,700	-	Mount, 1966
Fathead minnow, <i>Pimephales promelas</i>	FT, M	Zinc sulfate	50	6,200	-	Mount, 1966
Fathead minnow, <i>Pimephales promelas</i>	FT, M	Zinc sulfate	100	12,500	-	Mount, 1966
Fathead minnow, <i>Pimephales promelas</i>	FT, M	Zinc sulfate	100	12,500	-	Mount, 1966
Fathead minnow, <i>Pimephales promelas</i>	FT, M	Zinc sulfate	200	19,000	-	Mount, 1966
Fathead minnow, <i>Pimephales promelas</i>	FT, M	Zinc sulfate	200	13,600	-	Mount, 1966
Fathead minnow, <i>Pimephales promelas</i>	FT, M	Zinc sulfate	50	4,700	-	Mount, 1966
Fathead minnow, <i>Pimephales promelas</i>	FT, M	Zinc sulfate	50	5,100	?	Mount, 1966

Table 1. (Continued)

<u>Species</u>	<u>Method*</u>	<u>Chemical</u>	<u>Hardness (mg/l as CaCO₃)</u>	<u>LC50/EC50** (µg/l)</u>	<u>Species Mean Acute Value†† (µg/l)</u>	<u>Reference</u>
Fathead minnow, <i>Pimephales promelas</i>	FT, M	Zinc sulfate	100	8,100	-	Mount, 1966
Fathead minnow, <i>Pimephales promelas</i>	FT, M	Zinc sulfate	100	9,900	-	Mount, 1966
Fathead minnow, <i>Pimephales promelas</i>	FT, M	Zinc sulfate	200	8,200	-	Mount, 1966
Fathead minnow, <i>Pimephales promelas</i>	FT, M	Zinc sulfate	200	15,500	-	Mount, 1966
Fathead minnow, <i>Pimephales promelas</i>	S, U	Zinc sulfate	20	960	-	Pickering & Henderson, 1966
Fathead minnow, <i>Pimephales promelas</i>	S, U	Zinc sulfate	20	780	-	Pickering & Henderson, 1966
Fathead minnow, <i>Pimephales promelas</i>	S, U	Zinc sulfate	360	33,400	-	Pickering & Henderson, 1966
Fathead minnow, <i>Pimephales promelas</i>	S, U	-	20	2,550	-	Pickering & Henderson, 1966
Fathead minnow, <i>Pimephales promelas</i>	S, U	-	20	2,350	-	Pickering & Henderson, 1966
Fathead minnow (try), <i>Pimephales promelas</i>	FT, M	Zinc sulfate	186	870	-	Pickering and Vigor, 1965
Fathead minnow, <i>Pimephales promelas</i>	S, U	Zinc sulfate	166	7,630	-	Rachlin & Perlmuter, 1968
Banded killifish, <i>Fundulus diaphanus</i>	S, M	-	55	19,200	-	Rehwoldt, et al. 1972
Banded killifish, <i>Fundulus diaphanus</i>	S, M	-	53	19,100	-	Rehwoldt, et al. 1971

Table 1. (Continued)

<u>Species</u>	<u>Method*</u>	<u>Chemical</u>	<u>Hardness (mg/l as CaCO₃)</u>	<u>LC50/EC50** (µg/l)</u>	<u>Species Mean Acute Values (µg/l)</u>	<u>Reference</u>
<u>Flagfish, <i>Jordanella floridae</i></u>	FT, M	Zinc sulfate	44	1,500	-	Sopher, 1976
<u>Guppy, <i>Poecilia reticulata</i></u>	S, U	Zinc sulfate	45	30,000	-	Cairns, et al. 1969
<u>Guppy, <i>Poecilia reticulata</i></u>	S, U	Zinc sulfate	20	1,270	-	Pickerling & Henderson, 1966
<u>Southern platyfish, <i>Xiphophorus maculatus</i></u>	S, U	Zinc sulfate	166	12,000	-	Rachlin & Perlmutter, 1968
<u>White perch, <i>Morone americana</i></u>	S, M	Zinc nitrate	53	14,300	-	Rehwoldt, et al. 1971
<u>White perch, <i>Morone americana</i></u>	S, M	-	55	14,400	-	Rehwoldt, et al. 1972
<u>Striped bass, <i>Morone saxatilis</i></u>	S, M	-	55	6,800	-	Rehwoldt, et al. 1972
<u>Striped bass, <i>Morone saxatilis</i></u>	S, M	Zinc nitrate	53	6,700	-	Rehwoldt, et al. 1971
<u>Striped bass (fry), <i>Morone saxatilis</i></u>	S, M	-	137	1,180	-	O'Rear, 1972
<u>Striped bass (larvae), <i>Morone saxatilis</i></u>	S, U	Zinc chloride	38	100	-	Hughes, 1973
<u>Pumpkinseed, <i>Lepomis gibbosus</i></u>	S, M	Zinc nitrate	53	20,000	-	Rehwoldt, et al. 1971
<u>Pumpkinseed, <i>Lepomis gibbosus</i></u>	S, M	-	55	20,100	-	Rehwoldt, et al. 1972
<u>Bluegill, <i>Lepomis macrochirus</i></u>	FT, M	Zinc sulfate	46	9,900	-	Cairns, et al. 1971
<u>Bluegill, <i>Lepomis macrochirus</i></u>	FT, M	Zinc sulfate	46	12,100	-	Cairns, et al. 1971

Table 1. (Continued)

<u>Species</u>	<u>Method*</u>	<u>Chemical</u>	<u>Hardness (mg/l as CaCO₃)</u>	<u>LC50/EC50** (µg/l)</u>	<u>Species Mean Acute Value*** (µg/l)</u>	<u>Reference</u>
Bluugill, <u>Lepomis macrochirus</u>	S, U	Zinc chloride	52	7,450	-	Calirns & Scheler, 1959
Bluugill, <u>Lepomis macrochirus</u>	S, U	Zinc chloride	52	7,200	-	Calirns and Scheler, 1959
Bluugill, <u>Lepomis macrochirus</u>	S, U	Zinc chloride	52	6,910	-	Calirns & Scheler, 1959
Bluugill, <u>Lepomis macrochirus</u>	S, U	Zinc sulfate	20	5,460	-	Pickerling & Henderson, 1966
Bluugill, <u>Lepomis macrochirus</u>	S, U	Zinc sulfate	20	4,850	-	Pickerling & Henderson, 1966
Bluugill, <u>Lepomis macrochirus</u>	S, U	Zinc sulfate	20	5,820	-	Pickerling & Henderson, 1966
Bluugill, <u>Lepomis macrochirus</u>	S, U	Zinc chloride	20	5,370	-	Pickerling & Henderson, 1966
Bluugill, <u>Lepomis macrochirus</u>	S, U	Zinc sulfate	360	40,900	-	Pickerling & Henderson, 1966
Bluugill, <u>Lepomis macrochirus</u>	S, U	-	20	6,440	-	Pickerling & Henderson, 1966
Bluugill, <u>Lepomis macrochirus</u>	S, U	Zinc chloride	45	3,840	-	Calirns & Scheler, 1957a
Bluugill, <u>Lepomis macrochirus</u>	S, M	Zinc chloride	45	3,750***	-	Calirns & Scheler, 1957b
Bluugill, <u>Lepomis macrochirus</u>	S, M	Zinc chloride	45	3,430***	-	Calirns & Scheler, 1957b
Bluugill, <u>Lepomis macrochirus</u>	S, M	Zinc chloride	174	12,390***	-	Calirns & Scheler, 1957b

Table 1. (Continued)

<u>Species</u>	<u>Method*</u>	<u>Chemical</u>	<u>Hardness (mg/l as CaCO_3)</u>	<u>LC50/EC50** ($\mu\text{g/l}$)</u>	<u>Species Mean Acute Values** ($\mu\text{g/l}$)</u>	<u>Reference</u>
<u>Bluegill, <i>Lepomis macrochirus</i></u>	S,M	Zinc chloride	174	12,120***	-	Cairns & Schaefer, 1957b
<u>SALTWATER SPECIES</u>						
<u>Polychaete (adult), <i>Capitella capitata</i></u>	S, U	Zinc sulfate	-	3,500	-	Reish, et al. 1976
<u>Polychaete (larvae), <i>Capitella capitata</i></u>	S, U	Zinc sulfate	-	1,700	2,440	Reish, et al. 1976
<u>Polychaete (adult), <i>Neanthes arenaceodentata</i></u>	S, U	Zinc sulfate	-	1,800	-	Reish, et al. 1976
<u>Polychaete (juvenile), <i>Neanthes arenaceodentata</i></u>	S, U	Zinc sulfate	-	900	1,270	Reish, et al. 1976
<u>Polychaete (adult), <i>Nereis diversicolor</i></u>	S, U	Zinc sulfate	-	55,000	-	Bryan & Hummerstone, 1973
<u>Polychaete (adult), <i>Nereis diversicolor</i></u>	S, U	Zinc sulfate	-	11,000	24,600	Bryan & Hummerstone, 1973
<u>Sandworm (adult), <i>Nereis virens</i></u>	S, U	Zinc chloride	-	8,100	8,100	Eisler & Hennekey, 1977
<u>Oyster, <i>Crassostrea virginica</i></u>	S, U	Zinc chloride	-	310	310	Calabrese, et al. 1973
<u>Hard shelled clam, <i>Meretrix mercenaria</i></u>	S, U	Zinc chloride	-	166	166	Calabrese & Nelson, 1974
<u>Soft shelled clam, <i>Mya arenaria</i></u>	S, U	Zinc chloride	-	5,200	-	Eisler, 1977a
<u>Soft shelled clam, <i>Mya arenaria</i></u>	S, U	Zinc chloride	-	7,700	6,330	Eisler & Hennekey, 1977

Table 1. (Continued)

<u>Species</u>	<u>Method*</u>	<u>Chemical</u>	<u>Hardness (mg/l as CaCO_3)</u>	<u>LC50/EC50 ($\mu\text{g/l}$) **</u>	<u>Species Mean Acute Value*** ($\mu\text{g/l}$)</u>	<u>Reference</u>
				<u>LC50/EC50 ($\mu\text{g/l}$) **</u>	<u>Species Mean Acute Value*** ($\mu\text{g/l}$)</u>	
Mussel, <i>Mytilus edulis planulatus</i>	F, M	Zinc chloride	-	4,300	-	Ahsanullah, 1976
Mussel, <i>Mytilus edulis planulatus</i>	F, M	Zinc chloride	-	3,600	-	Ahsanullah, 1976
Mussel, <i>Mytilus edulis planulatus</i>	S, M	Zinc chloride	-	2,500	3,380	Ahsanullah, 1976
Mud snail (adult), <i>Nassarius obsoletus</i>	S, U	Zinc chloride	-	50,000	50,000	Eisler & Hennekey, 1977
Copepod (adult), <i>Acartia clausi</i>	S, U	Zinc chloride	-	950	950	U.S. EPA, 1980
Copepod (adult), <i>Acartia tonsa</i>	S, U	Zinc chloride	-	290	290	U.S. EPA, 1980
Copepod (adult), <i>Eurytemora affinis</i>	S, U	Zinc chloride	-	4,090	4,090	U.S. EPA, 1980
Copepod (adult), <i>Nitocra spinipes</i>	S, U	Zinc chloride	-	1,450	1,450	Bengtsson, 1978
Copepod (adult), <i>Pseudodiaptomus coronatus</i>	S, U	Zinc chloride	-	1,783	1,780	U.S. EPA, 1980
Copepod (adult), <i>Tigriopus japonicus</i>	S, U	Zinc chloride	-	2,160	2,160	U.S. EPA, 1980
Mysid shrimp, <i>Mysidopsis bahia</i>	S, M	Zinc chloride	-	498	498	U.S. EPA, 1980
Mysid shrimp, <i>Mysidopsis bigelowi</i>	S, M	Zinc chloride	-	591	591	U.S. EPA, 1980
Lobster (larvae), <i>Homarus americanus</i>	S, U	Zinc chloride	-	575	-	U.S. EPA, 1980
Lobster (larvae), <i>Homarus americanus</i>	S, U	Zinc chloride	-	375	-	U.S. EPA, 1980

Table 1. (Continued)

<u>Species</u>	<u>Method*</u>	<u>Chemical</u>	<u>Hardness (mg/l as CaCO₃)</u>	<u>LC50/EC50 (µg/l) **</u>	<u>Species Mean Acute Value** (µg/l)</u>	<u>Reference</u>
Lobster (larvae), <u>Homarus americanus</u>	S, U	Zinc chloride	-	282	-	U.S. EPA, 1980
Lobster (larvae), <u>Homarus americanus</u>	S, U	Zinc chloride	-	175	321	U.S. EPA, 1980
Crab (larvae), <u>Carcinus maenas</u>	S, U	Zinc sulfate	-	1,000	1,000	Conner, 1972
Hermit crab (adult), <u>Pagurus longicarpus</u>	S, U	Zinc chloride	-	400	400	Eisler & Hennetkey, 1977
Starfish (adult), <u>Asterias forbesi</u>	S, U	Zinc chloride	-	39,000	39,000	Eisler & Hennetkey, 1977
Mummichog (adult), <u>Fundulus heteroclitus</u>	S, U	Zinc chloride	-	60,000	-	Eisler & Hennetkey, 1977
Mummichog (larvae), <u>Fundulus heteroclitus</u>	S, U	Zinc chloride	-	83,000	70,600	U.S. EPA, 1980
Atlantic silverside (larvae), <u>Menidia menidia</u>	S, U	Zinc chloride	-	4,960	-	U.S. EPA, 1980
Atlantic silverside (larvae), <u>Menidia menidia</u>	S, U	Zinc chloride	-	4,170	-	U.S. EPA, 1980
Atlantic silverside (larvae), <u>Menidia menidia</u>	S, U	Zinc chloride	-	3,700	-	U.S. EPA, 1980
Atlantic silverside (larvae), <u>Menidia menidia</u>	S, U	Zinc chloride	-	3,060	-	U.S. EPA, 1980
Atlantic silverside (larvae), <u>Menidia menidia</u>	S, U	Zinc chloride	-	2,730	3,640	U.S. EPA, 1980
Winter flounder (larvae), <u>Pseudopleuronectes americanus</u>	S, U	Zinc chloride	-	18,200	-	U.S. EPA, 1980

Table 1. (Continued)

<u>Species</u>	<u>Method*</u>	<u>Chemical</u>	<u>Hardness (mg/l as CaCO₃)</u>	<u>LC50/EC50 (µg/l) **</u>	<u>Species Mean Acute Values (µg/l)</u>	<u>Reference</u>
Winter flounder (larvae), <i>Pseudopleuronectes americanus</i>	S, U	Zinc chloride	-	4,920	9,460	U.S. EPA, 1980

* S = static; FT = flow-through; R = renewal; M = measured; U = unmeasured

** Results are expressed as zinc, not as the compound.

***Calculated by logit analysis of the authors' data.

<u>Species</u>	<u>N</u>	<u>Slope</u>	<u>Intercept</u>	<u>R</u>	<u>Significance</u>
Snail, <i>Physa heterostropha</i>	4	1.18	2.19	0.99	*
Cladoceran, <i>Daphnia magna</i>	5	0.90	1.87	0.80	N.S.
Coho salmon, <i>Oncorhynchus kisutch</i>	2	1.23	2.86	-	-
Rainbow trout, <i>Salmo gairdneri</i>	22	0.85	3.18	0.83	**
Brook trout, <i>Salvelinus fontinalis</i>	6	0.82	4.48	0.96	**
Goldfish, <i>Carassius auratus</i>	2	0.19	8.20	-	-
Fathead minnow, <i>Pimephales promelas</i>	32	0.78	5.35	0.60	**
Guppy, <i>Poecilia reticulata</i>	2	3.90	-4.53	-	-
Striped bass, <i>Morone saxatilis</i>	4	0.79	4.05	0.22	N.S.

Table 1. (Continued)

<u>Species</u>	<u>N</u>	<u>Slope</u>	<u>Intercept</u>	<u>R</u>	<u>Significance</u>
<u>Bluegill,</u>					
<u>Lepomis macrochirus</u>	16	0.54	6.80	0.76	**

* = significant at $p = 0.05$ ** = significant at $p = 0.01$ Arithmetical mean acute slope = 0.83 ($n = 5$, see text)

Table 2. Chronic values for zinc

<u>Species</u>	<u>Test*</u>	<u>Chemical</u>	<u>Hardness (mg/l as CaCO_3)</u>	<u>Limits** ($\mu\text{g/l}$)</u>	<u>Chronic Value** ($\mu\text{g/l}$)</u>	<u>Reference</u>
<u>FRESHWATER SPECIES</u>						
<u>Cladoceran, <i>Daphnia magna</i></u>	LC	Zinc chloride	45	70-102	85	Blesinger & Christensen, 1972
<u>Cladoceran, <i>Daphnia magna</i></u>	LC	Zinc chloride	52	97-190	136	Chapman, et al. Manuscript†
<u>Cladoceran, <i>Daphnia magna</i></u>	LC	Zinc chloride	104	43-52	47	Chapman, et al. Manuscript†
<u>Cladoceran, <i>Daphnia magna</i></u>	LC	Zinc chloride	211	42-52	47	Chapman, et al. Manuscript†
<u>Ch Inook salmon, <i>Oncorhynchus tshawytscha</i></u>	ELS	Zinc chloride	25	270-510	371	Chapman, Manuscript†
<u>Rainbow trout, <i>Salmo gairdneri</i></u>	ELS	Zinc sulfate	26	140-547	277	Sinley, et al. 1974
<u>Brook trout, <i>Salvelinus fontinalis</i></u>	LC	Zinc sulfate	45	534-1,360	852	Holcombe, et al. 1979
<u>Fathead minnow, <i>Pimephales promelas</i></u>	LC	Zinc sulfate	46	78-145	106	Benolt & Holcombe, 1978
<u>Flagfish, <i>Jordanella floridae</i></u>	LC	Zinc sulfate	44	26-85	47	Spehar, 1976
<u>SALTWATER SPECIES</u>						
<u>Mysid shrimp, <i>Mysidopsis bahia</i></u>	LC	Zinc chloride	-	120-231	166	U.S. EPA, 1980

* LC = life cycle or partial life cycle; ELS = early life stage

** Results are expressed as zinc, not as the chemical.

Table 2. (Continued)

<u>Species</u>	<u>Hardness (mg/l as CaCO₃)</u>	<u>Acute-Chronic Ratio</u>		<u>Chronic Value (μg/l)</u>	<u>Ratio</u>
		<u>Acute Value (μg/l)</u>	<u>Chronic Value (μg/l)</u>		
<u>Cladoceran, <i>Daphnia magna</i></u>	45	100	85	85	1.2
<u>Cladoceran, <i>Daphnia magna</i></u>	52-54	334	136	2.4	
<u>Cladoceran, <i>Daphnia magna</i></u>	104-105	525	47	11	
<u>Cladoceran, <i>Daphnia magna</i></u>	196-211	655	47	14	
<u>Cladoceran, <i>Daphnia magna</i></u>	22-25	97-701	371	0.26-1.89	
<u>Ch Inok salmon, <i>Oncorhynchus tshawytscha</i></u>	26-30	240-830*	217	0.87-3.0	
<u>Brook trout, <i>Salvelinus fontinalis</i></u>	45	2,000	852	2.3	
<u>Fathead minnow, <i>Pimephales promelas</i></u>	46	600	106	5.7	
<u>Flagfish, <i>Jordanella floridae</i></u>	44	1,500	47	32	
<u>Mysid shrimp, <i>Mysidopsis bahia</i></u>	-	498	166	3.0	

* Acute values from Goetti et al. 1972.

Final Acute-Chronic Ratio = 3.0 (see text)

Table 3. Species mean acute intercepts and values and acute-chronic ratios for zinc

Rank*	Species	Species Mean Acute Intercept ($\mu\text{g/l}$)		Species Mean Acute-Chronic Ratio	
		FRESHWATER SPECIES			
29	Caddisfly, Unidentified sp.	2,260	-	-	-
28	Damsel fly, unidentified sp.	1,019	-	-	-
27	Pumpkinseed, <u>Lepomis gibbosus</u>	732	-	-	-
26	Worm, <u>Nais</u> sp.	716	-	-	-
25	Midge, <u>Chironomus</u> sp.	708	-	-	-
24	Banded killifish, <u>Fundulus diaphanus</u>	699	-	-	-
23	American eel, <u>Anguilla rostrata</u>	531	-	-	-
22	White perch, <u>Morone americana</u>	524	-	-	-
21	Goldfish, <u>Carassius auratus</u>	413	-	-	-
20	Guppy, <u>Poecilia reticulata</u>	367	-	-	-
19	Scud, <u>Gammarus</u> sp.	315	-	-	-
18	Bluegill, <u>Lepomis macrochirus</u>	293	-	-	-
17	Carp, <u>Cyprinus carpio</u>	285	-	-	-
16	Golden shiner, <u>Notemigonus crysoleucus</u>	255	-	-	-

Table 3. (Continued)

<u>Rank*</u>	<u>Species</u>	<u>Species Mean Acute Intercept (µg/l)</u>	<u>Species Mean Acute-Chronic Ratio</u>
15	Southern platyfish, <u>Xiphophorus maculatus</u>	172	-
14	Fathead minnow, <u>Pimephales promelas</u>	169	5.7
13	Rotifer, <u>Philodina acuticornis</u>	92.8	-
12	Brook trout, <u>Salvelinus fontinalis</u>	82.6	2.3
11	Coho salmon, <u>Oncorhynchus kisutch</u>	81.4	-
10	Flagfish, <u>Jordanella floridae</u>	64.9	32
9	Atlantic salmon, <u>Salmo salar</u>	57.9	-
8	Sockeye salmon, <u>Oncorhynchus nerka</u>	57.6	-
7	Striped bass, <u>Morone saxatilis</u>	49.3	-
6	Smelt, <u>Physa heterostropha</u>	42.0	-
5	Rainbow trout, <u>Salmo gairdneri</u>	26.2	0.87-3.0
4	Chinook salmon, <u>Oncorhynchus tshawytscha</u>	23.1	0.26-1.89
3	Cladoceran, <u>Daphnia pulex</u>	21.2	-
2	Longfin dace, <u>Agonopsis chrysogaster</u>	9.09	-

Table 3. (Continued)

<u>Rank#</u>	<u>Species</u>	<u>Species Mean Acute Intercept (µg/l)</u>	<u>Species Mean Acute-Chronic Ratio</u>
1	Cladoceran, <u>Daphnia magna</u>	8.89	1.2-14
<u>SALTWATER SPECIES</u>			
24	Mummichog, <u>Fundulus heteroclitus</u>	70,600	-
23	Mud snail, <u>Nassarius obsoletus</u>	50,000	-
22	Starfish, <u>Asterias forbesi</u>	39,000	-
21	Polychaete, <u>Nereis diversicolor</u>	24,600	-
20	Winter flounder, <u>Pseudopleuronectes americanus</u>	9,460	-
19	Sandworm, <u>Nereis virens</u>	8,100	-
18	Soft shelled clam, <u>Mya arenaria</u>	6,330	-
17	Copepod, <u>Eurytemora affinis</u>	4,090	-
16	Atlantic silverside <u>Menidia menidia</u>	3,640	-
15	Mussel, <u>Mytilus edulis planulatus</u>	3,380	-
14	Polychaete, <u>Capitella capitata</u>	2,440	-

Table 3. (continued)

<u>Rank #</u>	<u>Species</u>	<u>Species Mean Acute Value (µg/l)</u>	<u>Species Mean Acute-Chronic Ratio</u>
13	Copepod, <u>Tigriopus japonicus</u>	2,160	-
12	Copepod, <u>Pseudolaptomus coronatus</u>	1,780	-
11	Copepod, <u>Nitocra spinipes</u>	-	-
10	Polychaete, <u>Neanthes arenaceodentata</u>	1,270	-
9	Crab, <u>Carcinus naenias</u>	1,000	-
8	Copepod, <u>Acartia clausi</u>	950	-
7	Mysid shrimp <u>Mysidopsis bigelowi</u>	591	-
6	Mysid shrimp, <u>Mysidopsis bahia</u>	498	3.0
5	Hermit crab, <u>Pagurus longicarpus</u>	400	-
4	Lobster, <u>Homarus americanus</u>	321	-
3	Oyster, <u>Crassostrea virginica</u>	310	-
2	Copepod, <u>Acartia tonsa</u>	290	-
1	Hard shelled clam, <u>Meretrix mercenaria</u>	166	-

* Ranked from least sensitive to most sensitive based on species mean acute intercept or value.

Table 3. (continued)

Freshwater:

$$\text{Final Acute Intercept} = 7.02 \mu\text{g/l}$$

$$\text{Natural logarithm of } 7.02 = 1.95$$

$$\text{Acute slope} = 0.83$$

$$\text{Final Acute Equation} = e^{(0.83(\ln(\text{hardness}))+1.95)}$$

$$\text{Final Acute-Chronic Ratio} = 3.0 \text{ (Table 2)}$$

$$\text{Final Chronic Intercept} = (7.02 \mu\text{g/l})/3.0 = 2.34 \mu\text{g/l}$$

$$\text{Natural logarithm of } 2.34 = 0.85$$

$$\text{Chronic slope} = 0.83$$

$$\text{Final Chronic Equation} = e^{(0.83(\ln(\text{hardness}))+0.85)}$$

$$\text{Final Chronic Value} = 47 \mu\text{g/l} \text{ (see text)}$$

Saltwater:

$$\text{Final Acute Value} = 173 \mu\text{g/l}$$

$$\text{Final Acute-Chronic Ratio} = 3.0 \text{ (Table 2)}$$

$$\text{Final Chronic Value} = (173 \mu\text{g/l})/3.0 = 57.7 \mu\text{g/l}$$

Table 4. Plant values for Zn²⁺

<u>Species</u>	<u>Chemical</u>	<u>Hardness (mg/l as CaCO_3)</u>	<u>Effect</u>	<u>Result^a ($\mu\text{g/l}$)</u>	<u>Reference</u>
<u>FRESHWATER SPECIES</u>					
Alga, <i>Chorella vulgaris</i>	Zinc chloride	-	33-day EC50 cell number	5,100	Rosko & Rachlin, 1977
Alga, <i>Chorella vulgaris</i>	Zinc chloride	-	96-hour EC50 growth	2,400	Rachlin & Farran, 1974
Alga, <i>Selenastrum capricornutum</i>	Zinc chloride	-	7-day LC100	700	Bartlett, et al. 1974
Alga, <i>Selenastrum capricornutum</i>	Zinc chloride	-	7-day EC100 growth	120	Bartlett, et al. 1974
Alga, <i>Selenastrum capricornutum</i>	Zinc chloride	-	7-day Incomplete growth inhibition	30	Bartlett, et al. 1974
Alga, <i>Selenastrum capricornutum</i>	Zinc chloride	-	14-day EC95 growth	40	Greene, et al. 1975
Alga, <i>Selenastrum capricornutum</i>	Zinc chloride	-	14-day EC95 growth	68	Greene, et al. 1975
Alga, <i>Chlamydomonas</i> sp.	Zinc sulfate	-	5-day EC65 mean growth rate	15,000	Cairns, et al. 1978
Alga, <i>Scenedesmus quadrifolius</i>	Zinc sulfate	-	5-day EC25 mean growth rate	20,000	Cairns, et al. 1978
Alga, <i>Cyclotella meneghiniana</i>	Zinc sulfate	-	5-day EC65 mean growth rate	20,000	Cairns, et al. 1978
Diatom, <i>Nitzschia linearis</i>	Zinc chloride	-	12-hr LC50	4,300	Patrick, et al. 1968
Eurasian watermillet, <i>Myriophyllum spicatum</i>	-	-	32-day EC50 root weight	21,600	Stanley, 1974
Eurasian watermillet, <i>Myriophyllum spicatum</i>	-	-	32-day EC50 root length	21,600	Stanley, 1974
Eurasian watermillet, <i>Myriophyllum spicatum</i>	-	-	32-day EC50 shoot length	20,900	Stanley, 1974

Table 4. (continued)

<u>Species</u>	<u>Chemical</u>	<u>Hardness (mg/l as CaCO_3)</u>	<u>Effect</u>	<u>Result* ($\mu\text{g/l}$)</u>	<u>Reference</u>
Duckweed, <i>Lemna minor</i>	Zinc sulfate	-	28-day LC50	67,700	Brown & Rattigan, 1979
Macrophyte, <i>Elodea canadensis</i>	Zinc sulfate	-	28-day LC50	22,500	Brown & Rattigan, 1979
Macrophyte, <i>Elodea canadensis</i>	Zinc sulfate	-	28-day EC50 photosynthesis	8,100	Brown & Rattigan, 1979
<u>SALTWATER SPECIES</u>					
Alga, <i>Amphidinium carteri</i>	Zinc sulfate	-	Growth inhibition	400	Braek, et al. 1976
Alga, <i>Amphidinium carteri</i>	Zinc sulfate	-	Interaction with copper on growth	100	Braek, et al. 1976
Alga, <i>Dunaliella tertiolecta</i>	-	-	Reduction in potassium content	6,500	Overnell, 1975
Kelp, <i>Laminaria hyperborea</i>	Zinc sulfate	-	Growth inhibition	250	Hopkins & Kain, 1971
Kelp, <i>Laminaria digitata</i>	Zinc sulfate	-	Growth inhibition	100	Bryan, 1969
Giant kelp, <i>Macrocystis pyrifera</i>	-	-	50% inhibition of photosynthesis	10,000	Clemdenning & North, 1959
Alga, <i>Phaeodactylum tricornutum</i>	Zinc chloride	-	Growth inhibition	25,000	Jensen, et al. 1974
Alga, <i>Phaeodactylum tricornutum</i>	Zinc sulfate	-	Interaction with copper on growth	1,000	Braek, et al. 1976
Alga, <i>Skeletonema costatum</i>	Zinc sulfate	-	Growth inhibition	200	Braek, et al. 1976
Alga, <i>Skeletonema costatum</i>	Zinc sulfate	-	Interaction with copper on growth	50	Braek, et al. 1976

Table 4. (continued)

<u>Species</u>	<u>Chemical</u>	<u>Hardness (mg/l as CaCO_3)</u>	<u>Effect</u>	<u>Result* ($\mu\text{g/l}$)</u>	<u>Reference</u>
Alga, <i>Thalassiosira pseudonana</i>	Zinc sulfate	-	Growth inhibition	400	Braak, et al., 1976
Alga, <i>Thalassiosira pseudonana</i>	Zinc sulfate	-	Interaction with copper on growth	200	Braak, et al., 1976
Alga, <i>Scrippsiella aerophore</i>	Zinc sulfate	-	Decrease in cell numbers	1,000	Kayser, 1977
Alga, <i>Procentium micans</i>	Zinc sulfate	-	Decrease in cell numbers	500	Kayser, 1977
Alga, <i>Gymnodinium splendens</i>	Zinc sulfate	-	Decrease in cell numbers	100	Kayser, 1977
Alga, <i>Schroederella schroederi</i>	Zinc sulfate	-	Decrease in cell numbers	50	Kayser, 1977
Alga, <i>Thalassiosira rotula</i>	Zinc sulfate	-	Decrease in cell numbers	100	Kayser, 1977

* Results are expressed as zinc, not as the compound.

Table 5. Residues for zinc

<u>Species</u>	<u>Tissue</u>	<u>Chemical</u>	<u>Hardness (mg/l as CaCO₃)</u>	<u>Bioconcentration factor</u>	<u>DURATION (days)</u>	<u>Reference</u>
<u>FRESHWATER SPECIES</u>						
<u>Mayfly, <i>Ephemeraella grandis</i></u>	Whole body	Zinc sulfate	30-70	1,130	14	Nehring, 1976
<u>Stonefly, <i>Pteronarcys californica</i></u>	Whole body	Zinc sulfate	30-70	107	14	Nehring, 1976
<u>Atlantic salmon, <i>Salmo salar</i></u>	Whole body	Zinc sulfate	12-24	51	80	Farmer, et al. 1979
<u>Flagfish, <i>Jordanella floridae</i></u>	Whole body	Zinc sulfate	44	432	100	Spehler, et al. 1978
<u>SALTWATER SPECIES</u>						
<u>Alga, <i>Cladophora</i> sp.</u>	-	Zinc chloride	-	4,680	34	Baudlin, 1974
<u>Alga, <i>Fucus serratus</i></u>	-	Zinc chloride	-	16,600	140	Young, 1975
<u>Alga, <i>Enteromorpha prolifera</i></u>	-	Zinc sulfate	-	1,530	12	Munda, 1979
<u>Polychaete (adult), <i>Nereis diversicolor</i></u>	-	Zinc sulfate	-	20	34	Bryan & Hummertone, 1973
<u>Oyster (adult), <i>Crassostrea virginica</i></u>	-	Zinc chloride	-	16,700	140	Shuster & Pringle, 1969
<u>Gastropod (adult), <i>Littorina obtusata</i></u>	-	Zinc chloride	-	670	50	Young, 1975
<u>Soft-shell clam (adult), <i>Mya arenaria</i></u>	Soft parts	Zinc chloride	-	85	50	Pringle, et al. 1968
<u>Soft-shell clam (adult), <i>Mya arenaria</i></u>	Soft parts	Zinc chloride	-	43	112	Eisler, 1977b
<u>Mussel (adult), <i>Mytilus edulis</i></u>	Soft parts	Zinc chloride	-	225	13	Phillips, 1977

Table 5. (Continued)

<u>Species</u>	<u>Tissue</u>	<u>Chemical</u>	<u>Hardness (mg/l as CaCO_3)</u>	<u>Bioconcentration factor</u>	<u>Duration (days)</u>	<u>Reference</u>
Mussel (adult), <i>Mytilus edulis</i>	Soft parts	Zinc chloride	-	500	21	Pentreath, 1973
Mussel (adult), <i>Mytilus edulis</i>	Soft parts	Zinc chloride	-	282	35	Phillips, 1976
Crab (adult), <i>Carcinus maenas</i>	Muscle	Zinc chloride	-	8,800	22	Bryan, 1966

Table 6. Other data for zinc

<u>Species</u>	<u>Chemical</u>	<u>Hardness (mg/l as CaCO_3)</u>	<u>Duration</u>	<u>Effect</u>	<u>Result* ($\mu\text{g/l}$)</u>	<u>Reference</u>
		<u>FRESHWATER SPECIES</u>				
<u>Algae, <i>Selenastrum capricornutum</i></u>	Zinc phosphate	15	14 days	Growth Inhibition	64	Garton, 1972
<u>Cladoceran, <i>Daphnia magna</i></u>	Zinc chloride	-	64 hrs	LC50	72	Anderson, 1948
<u>Snail, <i>Gonobasis livescens</i></u>	Zinc sulfate	154	48 hrs	LC50	13,500	Cairns, et al. 1976
<u>Snail, <i>Lymnaea emarginata</i></u>	Zinc sulfate	154	48 hrs	LC50	4,150	Cairns, et al. 1976
<u>Snail, <i>Physa integra</i></u>	Zinc sulfate	154	48 hrs	LC50	4,400	Cairns, et al. 1976
<u>Cladoceran, <i>Daphnia magna</i></u>	Zinc sulfate	45	48 hrs	LC50 (5 C)	2,300	Cairns, et al. 1978
<u>Cladoceran, <i>Daphnia magna</i></u>	Zinc sulfate	45	48 hrs	LC50 (10 C)	1,700	Cairns, et al. 1978
<u>Cladoceran, <i>Daphnia magna</i></u>	Zinc sulfate	45	48 hrs	LC50 (15 C)	1,100	Cairns, et al. 1978
<u>Cladoceran, <i>Daphnia magna</i></u>	Zinc sulfate	45	48 hrs	LC50 (25 C)	560	Cairns, et al. 1978
<u>Cladoceran, <i>Daphnia pulex</i></u>	Zinc sulfate	45	48 hrs	LC50 (5 C)	1,600	Cairns, et al. 1978
<u>Cladoceran, <i>Daphnia pulex</i></u>	Zinc sulfate	45	48 hrs	LC50 (10 C)	1,200	Cairns, et al. 1978
<u>Cladoceran, <i>Daphnia pulex</i></u>	Zinc sulfate	45	48 hrs	LC50 (15 C)	940	Cairns, et al. 1978
<u>Mayfly, <i>Ephemeraella grandis</i></u>	Zinc sulfate	30-70	14 days	LC50 (25 C)	280	Cairns, et al. 1978
					>9,200	Nehrling, 1976

Table 6. (Cont'd.)

Species	Chemical	Hardness (mg/l as CaCO_3)	Duration	Effect	Result* ($\mu\text{g/l}$)	Reference
Mayfly, <i>Ephemera</i> <i>subvaria</i>	Zinc sulfate	54	10 days	LC50	16,000	Warnick & Bell, 1969
Stonefly, <i>Acroneuria lycorias</i>	Zinc sulfate	50	14 days	LC50	32,000	Warnick & Bell, 1969
Stonefly, <i>Pteronarcys californica</i>	Zinc sulfate	30-70	14 days	LC50	>13,900	Nehr Ing, 1976
Midge, <i>Tanytarsus dissimilis</i>	Zinc chloride	47	10 days	LC50	37	Anderson, et al. 1980
Caddisfly, <i>Hydropsyche betteni</i>	Zinc sulfate	52	11 days	LC50	32,000	Warnick & Bell, 1969
Coho salmon, <i>Oncorhynchus kisutch</i>	Zinc sulfate	3-10	96 hrs	WBC-T counts depressed at 1/2 96-hr LC50	500	McLeay, 1975
Sockeye salmon, <i>Oncorhynchus nerka</i>	Zinc chloride	20-90	18 mos	None (embryo to smolt)	242	Chapman, 1978a
Sockeye salmon, <i>Oncorhynchus nerka</i>	Zinc chloride	53	115 hrs	LC50	447	Chapman, 1978a
Sockeye salmon, (Zn acclimated) <i>Oncorhynchus nerka</i>	Zinc chloride	53	115 hrs	LC50	>630	Chapman, 1978a
Sockeye salmon, (Zn acclimated) <i>Oncorhynchus nerka</i>	Zinc chloride	22	96 hrs	LC50	1,660	Chapman, 1978a
Cutthroat trout, <i>Salmo clarki</i>	Zinc chloride	34-47	14 days	LC50	670	Nehr Ing & Goettl, 1974
Rainbow trout, <i>Salmo gairdneri</i>	Zinc sulfate	-	5 days	LC50	4,600	Bell, 1967
Rainbow trout, <i>Salmo gairdneri</i>	Zinc sulfate	240	48 hrs	LC50	4,000	Brown & Dalton, 1970

Table 6. (Continued)

<u>Species</u>	<u>Chemical</u>	<u>Hardness (mg/l as CaCO₃)</u>	<u>Duration</u>	<u>Effect</u>	<u>Result* (µg/l)</u>	<u>Reference</u>
Rainbow trout, <u>Salmo gairdneri</u>	Zinc sulfate	40	24 hrs	LC50 (5 C)	2,800	Cairns, et al. 1978
Rainbow trout, <u>Salmo gairdneri</u>	Zinc sulfate	40	24 hrs	LC50 (15 C)	1,560	Cairns, et al. 1978
Rainbow trout, <u>Salmo gairdneri</u>	Zinc sulfate	40	24 hrs	LC50 (30 C)	2,100	Cairns, et al. 1978
Rainbow trout, <u>Salmo gairdneri</u>	Zinc sulfate	14	21 days	LC50	500-1,000	Grande, 1967
Rainbow trout, <u>Salmo gairdneri</u>	Zinc sulfate	320	48 hrs	LC50	3,860	Herbert & Shurben, 1964
Rainbow trout, <u>Salmo gairdneri</u>	Zinc sulfate	320	48 hrs	LC50	2,460	Herbert & Van Dyke, 1964
Rainbow trout, <u>Salmo gairdneri</u>	Zinc sulfate	320	48 hrs	LC50	5,000	Herbert & Wakeford, 1964
Rainbow trout, <u>Salmo gairdneri</u>	Zinc acetate	-	96 hrs	LC50	550	Hale, 1977
Rainbow trout, <u>Salmo gairdneri</u>	Zinc sulfate	15-20	7 days	LC50	560	Lloyd, 1961
Rainbow trout, <u>Salmo gairdneri</u>	Zinc sulfate	320	3 days	LC50	3,500	Lloyd, 1961
Rainbow trout, <u>Salmo gairdneri</u>	-	38-54	14 days	LC50	410	Nehring & Goettl, 1974
Rainbow trout, <u>Salmo alpinus</u>	Zinc sulfate	25	5 days	LC50	135	Sinley, et al. 1974
Rainbow trout, <u>Salmo gairdneri</u>	Zinc sulfate	333	22 mos	LC10	1,055	Sinley, et al. 1974
Rainbow trout, <u>Salmo gairdneri</u>	Zinc sulfate	13-15	20 min	threshold avoidance level	5.6	Sprague, 1968

Table 6. (Continued)

<u>Species</u>	<u>Chemical</u>	<u>Hardness (mg/l as CaCO_3)</u>	<u>Duration</u>	<u>Effect</u>	<u>Result^a ($\mu\text{g/l}$)</u>	<u>Reference</u>
Rainbow trout, <u><i>Salmo gairdneri</i></u>	Zinc sulfate	374	85 days	EC25 growth inhibition	1,120	Watson & McKeown, 1976
Brown trout, <u><i>Salmo trutta</i></u>	Zinc sulfate	14	21 days	LC50	500-1,000	Grande, 1967
Brown trout, <u><i>Salmo trutta</i></u>	-	22-35	14 days	LC50	640	Nehring & Goettl, 1974
Atlantic salmon, <u><i>Salmo salar</i></u>	Zinc sulfate	12-24	21 days	LC50	1,450	Farmer, et al. 1979
Atlantic salmon, <u><i>Salmo salar</i></u>	Zinc sulfate	12-24	21 days	LC50	1,600	Farmer, et al. 1979
Atlantic salmon, <u><i>Salmo salar</i></u>	Zinc sulfate	12-24	21 days	LC50	510	Farmer, et al. 1979
Atlantic salmon, <u><i>Salmo salar</i></u>	Zinc sulfate	12-24	21 days	LC50	1,460	Farmer, et al. 1979
Atlantic salmon, <u><i>Salmo salar</i></u>	Zinc sulfate	12-24	21 days	LC50	340	Farmer, et al. 1979
Atlantic salmon, <u><i>Salmo salar</i></u>	Zinc sulfate	12-24	21 days	LC50	350	Farmer, et al. 1979
Atlantic salmon, <u><i>Salmo salar</i></u>	-	14	21 days	LC50	100-500	Grande, 1967
Atlantic salmon, <u><i>Salmo salar</i></u>	-	14	96-182 hrs	Incidient lethal level	150-1,000	Zitko & Carson, 1977
Brook trout, <u><i>Salvelinus fontinalis</i></u>	-	12-24	14 days	LC50	960	Nehring & Goettl, 1974
Goldfish, <u><i>Carassius auratus</i></u>	Zinc sulfate	40	24 hrs	LC50 (5 C)	103,000	Cairns, et al. 1978
Goldfish, <u><i>Carassius auratus</i></u>	Zinc sulfate	40	24 hrs	LC50 (15 C)	40,000	Cairns, et al. 1978
Goldfish, <u><i>Carassius auratus</i></u>	Zinc sulfate	40	24 hrs	LC50 (30 C)	24,000	Cairns, et al. 1978

Table 6. (Continued)

<u>Species</u>	<u>Chemical</u>	<u>Hardness (mg/l as CaCO_3)</u>	<u>Duration</u>	<u>Effect</u>	<u>Result* ($\mu\text{g/l}$)</u>	<u>Reference</u>
Golden shiner, <u>Notemigonus crysoleucus</u>	Zinc sulfate	40	24 hrs	LC50 (5 C)	11,400	Cairns, et al. 1976
Golden shiner, <u>Notemigonus crysoleucus</u>	Zinc sulfate	40	24 hrs	LC50 (15 C)	7,760	Cairns, et al. 1978
Golden shiner, <u>Notemigonus crysoleucus</u>	Zinc sulfate	40	24 hrs	LC50 (50 C)	8,330	Cairns, et al. 1978
Fathead minnow, <u>Pimephales promelas</u>	Zinc sulfate	203	10 mos	EC65 fecundity	180	Brunsga, 1969
Fathead minnow, <u>Pimephales promelas</u>	Zinc acetate	20	96 h	LC50	880	Pickering & Henderson, 1966
Guppy, <u>Poecilia reticulatus</u>	Zinc sulfate	80	90 days	EC60 growth	1,150	Crandall & Goodnight, 1962
Striped bass (embryo), <u>Morone saxatilis</u>	-	137	96 hrs	LC50 (5 C)	1,850	O'Rear, 1972
Bluegill, <u>Lepomis macrochirus</u>	Zinc sulfate	40	24 hrs	LC50 (5 C)	23,000	Cairns, et al. 1978
Bluegill, <u>Lepomis macrochirus</u>	Zinc sulfate	40	24 hrs	LC50 (15 C)	19,100	Cairns, et al. 1978
Bluegill, <u>Lepomis macrochirus</u>	Zinc sulfate	40	24 hrs	LC50 (30 C)	8,850	Cairns, et al. 1978
Bluegill, <u>Lepomis macrochirus</u>	Zinc chloride	45	96 hrs	LC50 periodic low DO	2,350	Cairns & Scheler, 1957a
Bluegill, <u>Lepomis macrochirus</u>	Zinc sulfate	370	20 days	LC50 (DO 1.7)	7,200	Pickering, 1968
Bluegill, <u>Lepomis macrochirus</u>	Zinc sulfate	370	20 days	LC50 (DO 1.9)	7,500	Pickering, 1968
Bluegill, <u>Lepomis macrochirus</u>	Zinc sulfate	370	20 days	LC50 (DO 3.2)	10,700	Pickering, 1968

Table 6. (Continued)

<u>Species</u>	<u>Chemical</u>	<u>Hardness (mg/l as CaCO_3)</u>	<u>Duration</u>	<u>Effect</u>	<u>Result* ($\mu\text{g/l}$)</u>	<u>Reference</u>
<u>Bluegill, <i>Lepomis macrochirus</i></u>	Zinc sulfate	370	20 days	LC50 (DO 3.2)	10,500	Pickerling, 1968
<u>Bluegill, <i>Lepomis macrochirus</i></u>	Zinc sulfate	370	20 days	LC50 (DO 5.4)	12,000	Pickerling, 1968
<u>Bluegill, <i>Lepomis macrochirus</i></u>	Zinc sulfate	370	20 days	LC50 (DO 5.3)	10,700	Pickerling, 1968
<u>Bluegill, <i>Lepomis macrochirus</i></u>	Zinc sulfate	-	1-24 hrs	Increased cough response	3,000	Sparks, et al. 1972a
<u>Bluegill, <i>Lepomis macrochirus</i></u>	Zinc sulfate	51	3 days	Lethal to fry	235	Sparks, et al. 1972b
<u>Bluegill, <i>Lepomis macrochirus</i></u>	Zinc phosphate	46	96 hrs	No death	32,000	Cairns, et al. 1971
<u>SALTWATER SPECIES</u>						
<u>Marine Isopod, <i>Idotea baltica</i></u>	Zinc sulfate	-	96 hrs	40% mortality (35 g/kg sal.)	10,000	Jones, 1975
<u>Marine Isopod, <i>Idotea baltica</i></u>	Zinc sulfate	-	78 hrs	60% mortality (28 g/kg sal.)	10,000	Jones, 1975
<u>Marine Isopod, <i>Idotea baltica</i></u>	Zinc sulfate	-	72 hrs	75% mortality (21 g/kg sal.)	10,000	Jones, 1975
<u>Marine Isopod, <i>Idotea baltica</i></u>	Zinc sulfate	-	48 hrs	100% mortality (14 g/kg sal.)	10,000	Jones, 1975
<u>Marine Isopod, <i>Jaera albifrons</i></u>	Zinc sulfate	-	72 hrs	10% mortality (35 g/kg sal.)	10,000	Jones, 1975
<u>Marine Isopod, <i>Jaera albifrons</i></u>	Zinc sulfate	-	67 hrs	30% mortality (3 g/kg sal.)	10,000	Jones, 1975
<u>Marine Isopod, <i>Jaera albifrons</i></u>	Zinc sulfate	-	52 hrs	80% mortality (0.4 g/kg sal.)	10,000	Jones, 1975

Table 6. (Continued)

<u>Species</u>	<u>Chemical</u>	<u>Hardness (mg/l as CaCO₃)</u>	<u>Duration</u>	<u>Effect</u>	<u>Result* (µg/l)</u>	<u>Reference</u>
Hermit crab (adult), <i>Pagurus longicarpus</i>	Zinc chloride	-	168 hrs	LC50	200	Eisler & Hennekey, 1977
Crab (larvae), <i>Rhithropanopeus harrisi</i>	Zinc chloride	-	16 days	Delayed development	50	BenJits-Claus & BenJits, 1975
Mummichog (adult), <i>Fundulus heteroclitus</i>	Zinc chloride	-	96 hrs	LC28	60,000	Eisler & Gardner, 1973
Mummichog (adult), <i>Fundulus heteroclitus</i>	Zinc chloride	-	24 hrs	Histological damage	60,000	Eisler & Gardner, 1973
Mummichog (adult), <i>Fundulus heteroclitus</i>	Zinc chloride	-	168 hrs	LC0	10,000	Eisler & Hennekey, 1977
Mummichog (adult), <i>Fundulus heteroclitus</i>	Zinc chloride	-	168 hrs	LC50	52,000	Eisler & Hennekey, 1977
Mummichog (adult), <i>Fundulus heteroclitus</i>	Zinc chloride	-	168 hrs	LC100	120,000	Eisler & Hennekey, 1977
Mummichog (adult), <i>Fundulus heteroclitus</i>	Zinc chloride	-	14 days	Increase in liver ALA-D enzyme activity	10,000	Jackl m, 1973
Mummichog (adult), <i>Fundulus heteroclitus</i>	Zinc chloride	-	48 hrs	LC100	157,000	Eisler, 1967
Mummichog (adult), <i>Fundulus heteroclitus</i>	Zinc chloride	-	192 hrs	LC0	43,000	Eisler, 1967
Mummichog (adult), <i>Fundulus heteroclitus</i>	Zinc chloride	-	192 hrs	LC50	66,000	Eisler, 1967
Atlantic salmon (smolt), <i>Salmo salar</i>	Zinc sulfate	-	48 hrs	50% survival (3 g/kg sal.)	6,000	Herbert & Wakeford, 1964
Atlantic salmon (smolt), <i>Salmo salar</i>	Zinc sulfate	-	48 hrs	50% survival (7 g/kg sal.)	15,000	Herbert & Wakeford, 1964
Atlantic salmon (smolt), <i>Salmo salar</i>	Zinc sulfate	-	48 hrs	50% survival (14 g/kg sal.)	35,000	Herbert & Wakeford, 1964

Table 6. (Continued)

<u>Species</u>	<u>Chemical</u>	<u>Hardness (mg/l as CaCO_3)</u>	<u>Duration</u>	<u>Effect</u>	<u>Result* ($\mu\text{g/l}$)</u>	<u>Reference</u>
Atlantic salmon (smolt), <i>Salmo salar</i>	Zinc sulfate	-	48 hrs	50% survival (26 g/kg sal.)	28,000	Herbart & Wakeford, 1964
Rainbow trout (yearling), <i>Salmo gairdneri</i>	Zinc sulfate	-	48 hrs	50% survival (3 g/kg sal.)	15,000	Herbart & Wakeford, 1964
Rainbow trout (yearling), <i>Salmo gairdneri</i>	Zinc sulfate	-	48 hrs	50% survival (7 g/kg sal.)	25,000	Herbart & Wakeford, 1964
Rainbow trout (yearling), <i>Salmo gairdneri</i>	Zinc sulfate	-	48 hrs	50% survival (14 g/kg sal.)	85,000	Herbart & Wakeford, 1964
Rainbow trout (yearling), <i>Salmo gairdneri</i>	Zinc sulfate	-	48 hrs	50% survival (26 g/kg sal.)	35,000	Herbart & Wakeford, 1964
Protozoan, <i>Cristigera</i> sp.	Zinc sulfate	-	4-5 hrs	Reduced growth	125	Gray, 1974
Protozoan, <i>Cristigera</i> sp.	Zinc sulfate	-	-	Growth reduction	125	Gray & Ventilla, 1973
Polychaete, <i>Ctenodrilus serratus</i>	Zinc sulfate	-	21 days	Reduced survival	10,000	Reish & Carr, 1978
Sandworm (adult), <i>Nereis virens</i>	Zinc sulfate	-	168 hrs	LC50	2,600	Eisler & Hennekey, 1977
Polychaete, <i>Ophryotrocha dialema</i>	Zinc sulfate	-	21 days	Reduced survival	1,750	Reish & Carr, 1978
Polychaete, <i>Ophryotrocha labronica</i>	Zinc sulfate	-	13 hrs	LC50	1,000	Brown & Ahsanullah, 1971
Hard-shell clam (larva), <i>Mercenaria mercenaria</i>	Zinc chloride	-	10 days	LC50	195	Calabrese, et al. 1977
Hard-shell clam (larva), <i>Mercenaria mercenaria</i>	Zinc chloride	-	12 days	LC95	341	Calabrese, et al. 1977
Soft-shell clam (adult), <i>Mya arenaria</i>	Zinc chloride	-	168 hrs	LC50 (20 C)	3,100	Eisler & Hennekey, 1977

Table 6. (Continued)

<u>Species</u>	<u>Chemical</u>	<u>Hardness (mg/l as CaCO₃)</u>	<u>Duration</u>	<u>Effect</u>	<u>Result* (µg/l)</u>	<u>Reference</u>
Soft-shell clam (adult), <i>Mya arenaria</i>	Zinc chloride	-	168 hrs	LC50 (22 C)	1,550	Eisler, 1977a
Mud snail (adult), <i>Nassarius obsoletus</i>	Zinc chloride	-	72 hrs	Decreased oxygen consumption	200	MacInnes & Thurberg, 1973
Mud snail (adult), <i>Nassarius obsoletus</i>	Zinc chloride	-	168 hrs	LC50	7,400	Eisler & Hennekey, 1977
Hard-shell clam (embryo), <i>Meretrix mercenaria</i>	Zinc chloride	-	42-48 hrs	LC100	279	Calabrese & Nelson, 1974
Hard-shell clam (larva), <i>Meretrix mercenaria</i>	Zinc chloride	-	12 days	LC5	50	Calabrese, et al., 1977
Oyster (larva), <i>Crassostrea gigas</i>	Zinc sulfate	-	5 days	Substrate attachment inhibition	125	Boyden, et al. 1975
Oyster (larva), <i>Crassostrea gigas</i>	Zinc sulfate	-	48 hrs	Reduced development	125	Brereton, et al. 1973
Oyster (larva), <i>Crassostrea gigas</i>	Zinc sulfate	-	6 days	Growth inhibition	125	Brereton, et al. 1973
Oyster (larva), <i>Crassostrea gigas</i>	Zinc chloride	-	48 hrs	Abnormal shell development	70	Nelson, 1972
Oyster (larva), <i>Crassostrea gigas</i>	Zinc chloride	-	48 hrs	LC0	75	Calabrese, et al. 1973
Oyster (larva), <i>Crassostrea virginica</i>	Zinc chloride	-	48 hrs	LC100	500	Calabrese, et al. 1973
Sea urchin (spermatozoa), <i>Arbacia punctulata</i>	Zinc chloride	-	4 mins	Decreased motility	1,635	Young & Nelson, 1974
Sea urchin (embryo), <i>Arbacia punctulata</i>	-	-	15 hrs	Abnormal development	1,250	Waterman, 1937
Starfish (adult), <i>Asterias forbesi</i>	Zinc chloride	-	168 hrs	LC50	2,300	Eisler & Hennekey, 1977

Table 6. (Continued)

<u>Species</u>	<u>Chemical</u>	<u>Hardness (mg/l as CaCO_3)</u>	<u>Duration</u>	<u>Effect</u>	<u>Result*</u> ($\mu\text{g/l}$)	<u>Reference</u>
Starfish (adult), <i>Asterias forbesi</i>	Zinc chloride	-	24 hrs	Equilibrium loss	2,700	Galtsoff & Loosanoff, 1939

* Results are expressed as zinc, not as the compound.

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Mammalian Toxicology and Human Health Effects

INTRODUCTION

More than 100 years ago it was shown that zinc was essential for the growth of Aspergillus niger. It was then shown that it was an essential metal for plant life. In the 1930's, the essentiality of zinc for the growth of rats was shown. Zinc has for a long time been regarded as an essential element for human beings but not until the 1960's was it shown that zinc deficiency could cause a certain syndrome and that therapy with zinc salts could alleviate or even cure the symptoms of zinc deficiency. During the recent past some other disease states including congenital diseases have been related to zinc. Zinc therapy has attracted the interest of clinicians. The evergrowing interest in the metabolism of zinc and the relationship between zinc and certain diseases has, during the last decades, been reflected in a large number of reviews and books (Brewer and Prasad, 1977; Halsted, et al. 1974; National Research Council, 1978; Pories, et al. 1974; Prasad, 1966, 1976, 1978; Sandstead, 1973, 1975; Vallee, 1959; Underwood, 1977). The National Research Council (NRC) report contains 1,855 references and gives information not only on metabolism and essentiality of zinc for human beings but also much information on occurrence of zinc, analytical methods, and human health hazards from excessive exposure to zinc. Since this document relies to a large extent on the NRC report, reference will be given to chapters or page numbers in that report whenever it is quoted in this or following sections.

The information given will rely mainly on the previously mentioned references and specific references will only be given when there is information which might add to the understanding of the metabolism and health effects of zinc, especially in humans.

EXPOSURE

Ingestion from Water

The National Research Council (1978) (Chapter 2 pp. 25-28 and Chapter 11 pp. 269-271) summarized available data on zinc in drinking water and concluded that generally the concentrations were well below 5 mg/l. In a study by the U.S. Department of Health, Education and Welfare (U.S. DHEW, 1970) 2,595 water samples were tested and of them eight had zinc concentrations above the 5 mg/l level. The highest concentration found was 13 mg/l. The average zinc concentration was 0.19 mg/l. In water leaving treatment plants, Craun and McCabe (1975) found that all samples contained less than 5 mg/l of zinc, but that in cities with soft acidic water the concentrations increased in the distribution system. Tapwater could thus have concentrations around 5 mg/l. In a study by U.S. EPA (1975) it was found that in 591 water samples all had zinc concentrations below 4 mg/l.

Uncontaminated fresh water generally contains zinc at less than 0.01 mg/l (NRC, 1978). Analysis of filtered surface waters in the U.S. revealed that of 714 samples only 7 had concentrations exceeding 1 mg/l and that 607 (85 percent) had concentrations below 0.1 mg/l (Durum, et al. 1971).

The concentration of zinc in both natural waters and in drinking water is generally low, but may increase due to pollution of water systems or release of zinc from distribution systems and household plumbing, respectively.

In the NRC document the content of zinc in different foodstuffs is listed in detail (Appendix A-1 pp. 313-326). It was noted that meat products contain relatively high concentrations of zinc, whereas fruits and vegetables have relatively low concentrations and contribute little to the daily intake. Zinc concentrations in milk are generally low, but a high intake of milk can make an important contribution to daily intake of zinc.

Additional data are provided by Mahaffey, et al. (1975) who calculated that meats, fish, and poultry on an average contained 24.5 mg/kg of zinc, whereas grains (and cereal products) and potatoes only provided 8 and 6 mg/kg, respectively. These data were obtained from Food and Drug Administration (FDA) market basket studies which are based on the diets of males 15 to 20 years old. In the years 1973 and 1974 it was calculated that the daily intake in this age group was 18 and 18.6 mg/day of zinc, respectively. In this age group was 18 and 18.6 mg/day of zinc, respectively. Gregger (1977) calculated the daily intake of zinc in subjects living in an institution for the aged, with an average age of 75 years, and found that on an average the intake was 18.7 mg/day. In girls 12 to 14 years old, Gregger, et al. (1978) found that the average intake of zinc was 10 mg/day.

In the "recommended dietary allowances" the National Research Council [National Academy of Sciences (NAS), 1974] recommended an average intake of zinc was 10 mg/day.

Ingestion from Food

The concentration of zinc in both natural waters and in drinking water is generally low, but may increase due to pollution of water systems or release of zinc from distribution systems and

tions of several aquatic species (Table 1). Bioccentration factors are available for the edible portion of other freshwater and estuarine fish and shellfish is

that per capita consumption of bivalve molluscs is 0.8 g/day and and estuarine fish and shellfish is 6.5 g/day (Stephan, 1980). was used to estimate that the per capita consumption of freshwater oysters, scallops, and mussels) should be considered a separate similar, except that for some compounds bivalve molluscs (clams, tion factors for the edible portion of most aquatic animals are for a variety of inorganic compounds indicate that biocencentration a range from the consumption of fish and shellfish. Residue data concerning food intake to calculate the amount of zinc which might be cerenting they live. An appropriate BCF can be used with data concerning a chemical in aquatic animals to the concentration in the water of a bioccentration factor (BCF) relates the concentration of

necessarily mean that zinc deficiency will occur.

A bioccentration factor (BCF) relates the concentration of zinc would be about 0.5 mg/kg for the infant and about 0.2 mg/kg zinc in the adult. These recommended doses take individual variations into account. An intake less than the recommended intake does not necessarily mean that zinc deficiency will occur.

mg/day was suggested. Based on body weight the requirement for mg/day was recommended and for children aged 0.5 to 1 year, 5 mg/day was recommended. In infants up to six months old, 3 mg/day was recommended. As a requirement of preadolescent children, intake of 25 mg/day. As a requirement of preadolescent children, women should have an intake of 20 mg/day and lactating women an intake of 15 mg/day, but pregnant

5.7 g/day.

Species	BCF	Reference	Carcinus meenas
Oyster (adult), Crassostrea virginica	16,700	Shuster and Pringle, 1969	Crab, Bryan, 1966
Mya arenaria Soft-shell clam,	85	Pringle, et al., 1968	Mysticus edulis Mussel, Phillips, 1977
Mya arenaria Soft-shell clam,	43	Eisler, 1977	Mysticus edulis Mussel, Penetreath, 1973
Mya arenaria Soft-shell clam,	225	Phillips, 1977	Mysticus edulis Mussel, Phillips, 1976
Mya arenaria Soft-shell clam,	500	Penetreath, 1973	Mysticus edulis Mussel, Phillips, 1976
Mya arenaria Soft-shell clam,	282	Phillips, 1976	Mysticus edulis Mussel, Phillips, 1976
Crab, Carcinus meenas	8,800	Bryan, 1966	

BIOCONCENTRATION FACTORS OF EDIBLE PORTIONS OF AQUATIC ORGANISMS

TABLE I

mental pollution. The U.S. data may be compared to data from 15 and 0.8 $\mu\text{g}/\text{m}^3$, respectively, indicating very severe environment. It is noted that the average lead and cadmium concentrations were 24-hour values ranged from 0.27 to 15.7 $\mu\text{g}/\text{m}^3$. It should be ambient air a yearly mean zinc concentration of 5 $\mu\text{g}/\text{m}^3$. The from a smelter in Kellogg, Idaho, Ragaini, et al. (1977) found in concentrations have been reported near smelters. About 1.5 miles annual averages varied from 0.29 to 0.38 $\mu\text{g}/\text{m}^3$. Much higher concentrations in New York City during the years 1972 to 1975 where the As an example, Lioy, et al. (1978) presented data on zinc concentrations in areas of zinc are, in most areas, fairly constant. The air levels of zinc are, in most areas, quarterly average.

Air Survey Network sites throughout the U.S. zinc concentrations (1979) observed zinc concentrations at approximately 50 National than 1 $\mu\text{g}/\text{m}^3$ (Chapter 3 p. 42-43). In 1975 and 1976, U.S. EPA that zinc concentrations throughout the U.S. generally are less air quality data compiled in the NRC document (1978) show by Americans is calculated to be 47.

portion of all freshwater and estuarine aquatic organisms consumed weighed average bioconcentration factor for zinc and the edible values of 353 and 3.5 are used with the consumption data, the probably about 1 percent of that for bivalve molluscs. If the for copper and cadmium, the mean BCF value for other species is 1979) and 432 (Sperhar, et al. 1978). Based on the available data for the whole body of two species of fish were 51 (Farmer, et al. but the value for the crab seems too high, considering that values the geometric mean of the values for bivalve molluscs is 353,

crities in a heavily industrialized European country, Belgium (Kretzschmar, et al., 1977). During the period May 1972 to April 1975 the average concentrations in 15 locations were from 0.22 to 3.05 $\mu\text{g}/\text{m}^3$. The highest value recorded during 24 hours was 57

The fate of inhaled particles containing zinc will depend on particle size and solubility as well as functional state of the lungs. The quantitative features of the deposition patterns of zinc particles have been reviewed by the Task Group on Lung Dynamics (1966) and the Task Group on Metal Accumulation (1973). There are no quantitative data on the deposition and absorption of zinc compounds, but experiments on human beings by Sturgis, et al. (1927) and Driuker, et al. (1927) indicated that both zinc oxide fumes and zinc oxide powder with very small particle size were deposited and Dr. Driuker, et al. (1927) indicated that both zinc oxide fumes and zinc oxide powder, but experiments on human beings by Sturgis, et al. (1927) no quantitative data on the deposition and absorption of zinc compounds, but experiments on human beings by Sturgis, et al. (1927) and the Task Group on Metal Accumulation (1973). There are no quantitative data on the deposition and absorption of zinc compounds, but experiments on human beings by Sturgis, et al. (1927) and the Task Group on Metal Accumulation (1973).

Absorption

PHARMACOKINETICS

Larger amounts can generally be well tolerated. homeostatic regulation of zinc ensures that such amounts and even total daily intakes of 25 to 30 mg. As discussed later, the i.e., 15 to 20 mg, the additional intake via water will result in mended limits. In people with recommended daily intakes of zinc, dietary intake, but the total intake will still be within recommended daily intake, but the total intake will be within a low later amount might double the intake for people on a low this might result in daily intakes of 2 and 10 mg, respectively. The later amount might result in daily intake of 2 liters of water have been reported. Assuming a daily intake of 2 liters of water levels around 1 mg/l are not uncommon and levels around 5 mg/l The intake via drinking water might be of more significance.

Airborne exposure is insignificant.

Conribute even less than that. Compared to the intake via food, assuming that he inhales 20 m³ of air per day. Smoking would of 5 µg/m³, would have an additional daily intake of 100 µg, adults. An individual inhaling air with an average concentration

The protein content of the diet has been shown to influence the uptake of zinc. In studies done on people with zinc deficiency it has been noted that the effect of zinc therapy is enhanced by a simultaneous administration of protein. It has also been shown that the absorption of zinc will be reduced if the diet contains large amounts of phytate especially in the presence of large amounts of calcium (NRC Chapter 7 pp. 183-187). Since phytofactors are found in cereals, zinc in vegetable diets that

Spenner, et al. (1965) showed in human beings that 65Zn as the chloride was rapidly taken up, with plasma peak values within four hours. It was calculated that about 50 percent was absorbed, but with a wide range (20 to 80 percent). In that study it was not possible to show that the amount of calcium in the diet influenced the uptake of zinc from the gut. There are difficulties in assessing the absorption of zinc, since there is also competition in absorbing the absorption of zinc since there is also competition in absorbing the absorption of zinc.

In the alveoli, that inhaled zinc is absorbed by the lining of increased serum and plasma levels of zinc in exposed workers. It should be pointed out, however, that part of the inhaled material will be transported to the gastrointestinal tract via ciliary activity and some zinc may also be absorbed that way. The absorption of ingested zinc will depend mainly on the zinc status of the organism. The presence or absence of other nutritional constituents may also influence absorption.

which molecular weights lower than metallothionein have been found may bind zinc (Richards and Cousins, 1977). Zinc binding ligands molecular weight, metal-binding protein, in the intestinal mucosa absorption process. There is evidence that metallothionein, a low proteins and low molecular weight compounds may be involved in the control, and data from animal experiments suggest that several The mechanisms for absorption of zinc are homeostatically controlled that there was no difference in absorption.

however, was not statistically significant, and the authors comment and in the control group it was 46 percent. The difference, centing the contraceptive agents, the average absorption was 33 percent measured by neutron activation analysis. Among the women taking between intake and fecal output of the stable isotope which was measured by neutron activation analysis. All women took, 70Zn , and the absorption was determined from the difference, 70Zn , all were of similar age. Zinc was administered as a stable isotope, 70Zn , who did not take contraceptive pills (King, et al. 1978). Women who did not take oral contraceptive agents on the absorption of zinc was studied in 14 women. They were compared with eight of zinc was available and thus meat is a good source of zinc.

The influence of oral contraceptive agents on the absorption of zinc (Sandstedt, et al. 1978). Zinc in animal proteins seems to small. The fiber content of the diet may influence the uptake of found. In this study, the influence of phytate seems to have been repeated after one month and the same average absorption was ranging from 12.2 to 39.1 percent in 11 subjects. The study was absorption of 65Zn added to bread during baking was 25 percent for absorption. Arvidsson, et al. (1978) found that the average include large amounts of unleavened bread may be less available

tion has recently been discussed by Cousins (1979).
 complexes in rat intestine. The role of ligands in zinc absorption
 metallothionein may occur as low molecular weight zinc binding
 (1978) that degradation products of intestinal proteins including
 the ligand or ligands. It has also been shown by Cousins, et al.
 ent no certain conclusions can be drawn regarding the nature of
 Evans and Johnson, 1979). The data are controversial and at pres-
 Johnson, 1977; Schrieker and Forbes, 1978; Lonnnerdal, et al. 1979;
 isolate and identify this ligand (Song and Adham, 1977; Evans and
 rat. During the last years several studies have been performed to
 was similar to the ligand found in pancreatic secretions from the
 thought that the low molecular weight zinc binding ligand in milk
 similar results were reported by Evans and Johnson (1976) who
 usually occurred when infants were weaned from human breast milk.
 for the congenital disease acrodermatitis enteropathica which
 species difference was taken by these authors as an explanation
 mainly associated with low molecular weight fractions. This
 with high molecular weight fractions, whereas in human milk it was
 milk and human milk showed that in cow's milk zinc was associated
 Eckhardt, et al. (1977) reported that gel chromatography of cow's
 human milk, but has not been found in bovine milk. In 1976,
 Of special interest is a zinc binding ligand which occurs in
 tion.
 could bind zinc in the gastrointestinal tract and enhance absorption
 was produced in the pancreas and through the pancreatic secretions
 in animals. Evans, et al. (1975) proposed that such a compound

al. 1976; NRC, 1978).

constitutes the diffusible serum zinc (Giroux, 1975; Giroux, et zinc in the α^2 -macroglobulin. The zinc bound to amino acids here is an exchange of zinc, whereas there is no exchange with few percent to amino acids. In the albumin and the amino acids third of the serum zinc is bound to an α^2 -macroglobulin and a one of the histidine moieties of the albumin molecule. About one mainly bound to albumin and it is thought that the binding is to several zinc metalloenzymes are present. In plasma, zinc is the zinc metalloenzyme carbonic anhydrase and in leukocytes where zinc is found in erythrocytes mainly due to the presence of

Distribution

zinc could be shown, but no abnormal values were found. Anteby, et al. (1978) reported that in women using an intrauterine device containing copper and zinc, a slight rise in serum zinc oxide, there was absorption of zinc. Hällmans showed that in humans treated for burns with gauzes containing sulfate was higher than from zinc oxide. Hällmans (1977) also found that in animals treated with zinc gauzes containing zinc sulfate. At a concentration of 20 percent there were even systemic effects. Hällmans concluded that the absorption from zinc sulfate was a high absorption of zinc from gauzes containing zinc there was a high absorption of zinc from gauzes containing zinc Hällmans (1978a,b) showed that in rats with excisional wounds cant.

and was of a magnitude which might be clinically significant animals and was of a magnitude which might be clinically significant authors the amount of zinc absorbed was higher in zinc-deficient absorbed through intact skin of the rat. According to these Keeen and Hurtley (1977) have shown that zinc salts will be

In the tissues, the highest concentrations of zinc are found in the male reproductive system where the prostate has the highest concentration. Zinc concentrations of zinc also occur in the muscle, bone, liver, kidney, pancreas, and some endocrine glands, especially the thyroid. The largest amounts of zinc are found in the muscles and the bone. Within tissues there may be variation; in the human prostate gland the highest zinc concentrations are found in the lateral prostate and the lowest in the interior and inner prostate. Also significant is the finding that semen has a high zinc content. In most organs there are relatively small variations in zinc levels during a lifetime except that in the newborn, zinc concentrations generally are higher than later in life. It should also be pointed out that the zinc content of the kidney and liver will, to a large degree, depend on the cadmium concentration, and renal zinc concentrations will vary with age (Eliander, 1971).

Of special interest is the relationship between zinc and histidine. It has been shown in human beings that oral administration of histidine will cause decreases in serum zinc and an increase in urinary zinc excretion (Henklin, et al. 1975). This observation has also been made in experiments on rats (Freeman and Taylor, 1977) and dogs (Yunice, et al. 1978). The latter authors also showed that cysteine caused a considerable increase in excretion of zinc. This is thought to be one explanation for the losses of zinc seen in patients given parenteral hyperalimentation, since the fluids given usually contained large amounts of essential amino acids, without sufficient amounts of essential

days in 19 female patients undergoing treatment for rheumatoid and Kennedy, et al. (1978) found that the average half-time was 412 the injected zinc for this group was on an average 247 days. taken for 84 to 190 days. The slow component for the half-time of injections of 65Zn. After the injection, measurements were 151-154). Arvidsson, et al. (1978) gave eight subjects single to about 400 days, depending on the zinc status (NRC Chapter 6 pp. of 65Zn to human beings, the half-time may vary from about 200 zinc status; it has been shown that after oral intake or injection the long-term biological half-time of zinc will depend on the about one-tenth (1/10) of the urinary excretion.

Losses by desquamation would be about 20 to 40 µg, only daily losses by the zinc content of epidermis. They calculated that the variation the zinc content of epidermis. Molin and Wester (1976) determined by neutron activation losses. Molin and Wester (1976) determined by neutrinacti-blood losses. Molin and Wester (1976) determined by neutrinacti-losses of zinc may also occur via the skin and menstrual to the fetus.

Via hair and milk, and in the female there is a placental transfer and Emmett, 1978; Honnadek, et al. 1973). Zinc is also excreted or exercise, much larger quantities may be excreted in sweat (Cohn relatively small but with certain conditions, i.e., extreme heat part of that zinc is reabsorbed. Urinary excretion of zinc is zinc is mainly excreted via the gastrointestinal tract but

Excretion

also found in metallothionein. Zinc is generally an essential component of many enzymes. Zinc is regarding the form in which zinc is stored in different organs, et al. 1978; Piscator and Lind, 1972; Schroeder, et al. 1967).

osteoarthritis. They were given an oral dose of 65Zn. In certain body compartments, e.g., bone, the half-time may be considerably longer (NRC Chapter 6 pp. 149-154).

Metallothionein was briefly discussed in previous reports and books concerning zinc, but during the last several years there has been an enormous increase in the number of papers on this protein. Recently a very comprehensive report on metallothionein has been prepared (Norberg and Kojima, 1979). Mammalian metallothionein is a protein with a molecular weight of 6,000 to 7,000 which is characterized by a very special amino acid composition, a high cysteine content, but lack of aromatic amino acids and histidine. Metallothionein was first discovered in equine renal cortex by Margoshes and Vallee (1957) and has now been shown to occur in most mammalian tissues, and also in lower organisms. Total metal content of metallothionein can reach 6 to 7 g atoms per mole. The metals generally found in metallothionein are zinc, copper, and cadmium. The relative occurrence of these metals will depend on a number of factors. In fetal liver metallothionein, zinc and copper are the major constituents, whereas in animals exposed to cadmium, cadmium will be the dominant metal especially in the renal tissue. A number of factors can induce the synthesis of metallothionein. In addition to administration of the above-men- tioned metals, metallothionein synthesis seems also to be induced by factors that might influence zinc metabolism. Thus, en- duced by factors that might influence zinc metabolism. Thus, environmental stresses of different kinds may induce the synthesis.

With regard to zinc metabolism, it has been shown that paren- teral or dietary administration of zinc will cause an increase of zinc in the body compartments, e.g., bone, the half-time may be con-

In the National Research Council report (1978), extensive information is given on concentrations of zinc in blood, urine and tissues (Chapter 6 pp. 123-145). The NRC report concluded that the mean serum-zinc concentration in humans is approximately 1 mg/L, the same in healthy men and women. The zinc content of whole blood will be about five times higher than the serum level.

In the National Research Council report (1978), extensive regulation of zinc in the mammalian body (Nordberg and Kojima, 1979). Doubt that this protein must play a very important role in the about the biological function of metallotionein, but there is no and Weser, 1977; Ryden and Deutsch, 1978). Much is still unknown lung zinc and copper in fetal livers (Bremner, et al. 1977; Hartman investigators of finding large amounts of metallotionein contain easily available pool for acute replacements of zinc in certain institutions. Similar indications are given by reports from several serve as a regulator of plasma zinc levels and constitute an liver parenchymal cells, a process that required synthesis of that glucocorticoids in vitro stimulated the uptake of zinc in Sobociński, et al. 1978). Failla and Cousins (1978) demonstrated changes (Bremner and Davies, 1975; Richards and Cousins, 1976; restrictive and bacterial infections have been shown to cause such and hot environments, burns, and exercise (Oh, et al. 1978). Food late in response to several environmental stresses, such as cool hepatic zinc was increased and metallotionein synthesis stimu- Richards and Cousins, 1975a,b, 1977). Recently, it was shown that the synthesis of metallotionein (Bremner and Davies, 1975;

since the concentration in the red cells is about 10 times the amount found in serum. A lowering of the serum concentrations of zinc may be seen in women taking contraceptive pills, during pregnancy, and as a result of certain stresses such as infections. In the same individual the zinc concentration in serum will be higher than in plasma mainly due to the release of zinc from platelets (Foley, et al. 1968). In 14 subjects the mean serum level was 1.15 mg/l and the mean plasma level 0.98 mg/l, the average difference being 16 percent.

The influence of age and sex on plasma zinc levels was studied by Chooi, et al. (1976). They found that in both males and females there was a decrease in plasma zinc from age 20 to age 90. Between men and women below the age of 50 no difference in plasma zinc levels could be noted between the sexes. However, females using contraceptive agents had lower zinc levels than women who did not take contraceptive agents. Average plasma levels in the groups studied were around 0.7 mg/l.

In a recent report, Hartoma (1977) stated that men had higher serum zinc levels than women. The average concentration in 154 male blood donors was 1.24 mg/l (range 0.74 to 2.2 mg/l), and in 95 women it was 1.11 mg/l (range 0.64 to 1.82 mg/l). The difference was highly significant according to the author. It was not stated to what extent the women took contraceptive pills. Hartoma also found that there was a slight tendency to a lowering of the serum concentration of zinc in men with increasing age, and that there was a significant correlation between serum zinc and serum testosterone in males aged 36 to 60 years. In men 28 to 35 years of age, there was a significant correlation between serum zinc and serum testosterone in males aged 36 to 60 years. In men 28 to 35 years of age, there was a significant correlation between serum zinc and serum testosterone.

The tissue concentrations of zinc are generally higher in the newborn. After the first year of life there are fairly small changes in the zinc levels in most organs except the kidney where the zinc concentrations are dependent on the accumulation of cadmium (Elinder, et al. 1977; Piscator and Lind, 1972; Prasad, 1976). In the liver the zinc level is constant during a lifetime. In the pancreas there is a decrease in zinc levels with increasing age on a wet weight basis, whereas if the pancreas values are calculated on an ash weight basis that decrease is not seen (Elinder, et al. 1977). This is in agreement with Schröder, et al. (1967). In the study by Elinder, the average concentrations of zinc in liver and pancreas were 45 (s.d. = 13.6) and 27 (s.d. = 7.2) mg/kg wet weight, respectively. In these organs the concentrations of zinc in the study by Elinder, the average concentrations of zinc in liver and pancreas were 45 (s.d. = 13.6) and 27 (s.d. = 7.2) mg/kg wet weight, respectively.

In the NRC report (Chapter 6 p. 129) it was stated that fasting among nonsmokers there was a tendency to decreased zinc excretion towards a higher zinc excretion in smokers than in nonsmokers. Ellinckx, et al. (1978) who studied the urinary excretion of zinc in different age groups. They found that there was a tendency in a higher zinc excretion in smokers than in nonsmokers.

age, there was a negative correlation, which was not significant. In these two studies plasma and serum levels, respectively, were lower and higher than earlier reported data which indicates that methodological problems in sampling and analysis may still exist. In both studies samples were taken in the morning after overnight fast.

TABLE 2

Zinc Concentrations in the Urine of Swedish People^t

Group	Number of Persons	Zinc, Average (a)* (mg/g of creatinine)	Standard Deviation (a)*	Zinc, Calculated Average (mg/24 hr)
Men, non-smokers (age in years)				
2 to 9	4	0.86	0.23	0.33
17 to 19	10	0.38	0.19	0.71
20 to 29	10	0.32	0.13	0.59
30 to 39	10	0.29	0.10	0.49
40 to 49	16	0.25	0.16	0.39
50 to 59	15	0.32	0.09	0.45
60 to 69	9	0.27	0.17	0.35
70 to 79	11	0.40	0.18	0.46
80 to 89	9	0.35	0.12	0.35
Men, smokers (age in years)				
40 to 49	5	0.35	0.19	0.55
50 to 59	5	0.39	0.09	0.55
66	1	0.32	--	0.41
75	1	0.27	--	0.31
Women, non-smokers (age in years)				
3	4	1.23	0.21	0.37
40 to 49	10	0.23	0.17	0.20
50 to 59	10	0.47	0.38	0.34

^tSource: Elinder, et al. 1978.

*a, arithmetic averages.

The homeostatic regulation of zinc absorption in the rat was studied by Evans, et al. (1973). Rats fed an optimal intake of zinc were compared to rats which had been on a diet for 7 and 13 days, respectively, containing less than 1 mg/kg of zinc. Whereas, in the controls the absorption was about 15 percent, measured as, in the rats after a one hour after examining the radioactivity in the carcass

Chapter 6 p. 123).

zinc were normally distributed, whereas zinc concentrations in renal cortex had a log-normal distribution. When the renal zinc bound to metallothionein (assuming a cadmium-zinc ratio of 1.0 in the prostate, where the concentration is about 100 mg/kg wet weight). In human semen concentrations of 100 to 350 mg/l have been reported. The zinc concentrations in hair will vary depending on age and geographical location (MRC Chapter 6 pp. 140-141). The average zinc concentration in hair from adults varied from 148 to 210 mg/kg. The newborn has zinc levels in hair similar to adults in the adult, but at age 1 to 4 the levels are lower than levels in adults (Hambridge, et al. 1972; Petering, et al. 1971). Zinc concentrations in hair will decrease during pregnancy (Baumslag, et al. 1974; Hambridge and Droege, 1974). The determination of zinc in hair has been used as a screening tool for zinc deficiency (Hambridge, et al. 1972). The total body store of zinc in adults humans has been estimated to be 2.3 mg for a 70 kg man (NRC 1974).

lected for seven days. The elimination of fecal zinc was similar age, an oral dose of 65Zn as the chloride. Feces were collected before sacrifice. One week before sacrifice each rat was given, by gavage, 600 mg/kg of added zinc beginning 7, 14, 21, or 42 days before 53 ppm zinc, and at different times groups were given a diet with by Ansari, et al. (1975). Male rats were given a diet containing The homeostatic regulation of ingested zinc was also studied

increased in proportion to the daily intake.

both total absorption and total fecal excretion of endogenous zinc supply was not sufficient. In the three highest exposure groups receiving 5.6, 10.6, and 18.2, indicating that in this study this receiving 38, 70, and 141 mg/kg, whereas it was lower in the groups fed of zinc. The daily zinc retention was the same in the groups relatively high absorption figure for the group receiving 141 mg/kg rats which were growing rapidly. This may also explain the relatively high absorption rapidly. These were mean living of 100 percent seems surprisingly high, but these were mean living which had been injected at the start of the experiment. The figure could be determined by measuring the turnover of radioactive zinc the true zinc absorption and the fecal excretion of endogenous zinc from 100 to 34 percent in inverse relation to the intake of zinc. adjusted to the respective intakes and the absorption of zinc was 10.6, 18.2, 38, 70, and 141 mg/kg. After six days the animals had six they were given a diet with the following zinc contents: 5.6, animals for zinc absorption in 36 mean living rats, where in groups of weigland and Krichgau (1978) studied the homeostatic mech-

tively, in the two experimental groups.

gastric dose of 65Zn, it was about 35 and 50 percent, respec-

new increase was seen, also in the heart but not in muscle tissue. Levels of 2,400 to 7,200 ppm in the diet, but at 8,400 ppm level a further increase in muscle tissue. No further increase was seen at in the heart and muscle tissue. No further increase was seen at tibia than controls, whereas there was no change in concentrations significantly higher levels of stable zinc in the liver, kidney, and fecal ^{65}Zn . Rats given 1,200 ppm zinc in the diet had significant higher levels of dietary zinc there was no further increase of the ^{65}Zn was eliminated in one week in contrast to 86 percent in the rats given 1,200 ppm zinc in the diet. At still greater content of the ^{65}Zn was any obvious signs of toxicity. In controls 65 percent or produce any obvious signs of toxicity. In controls 65 percent content of the diet did not affect weight gains, feed consumption, and after that feces were collected for one week. The high zinc sacrifice each rat was given ^{65}Zn as the chloride by gavage of from 1,200 to 8,400 ppm zinc for three weeks. One week before Ansari, et al. (1976) gave male rats dietary zinc at levels absorbed zinc and tissue stores of zinc.

They also show how rapid the exchange will be between diet. They also show how rapid the exchange will be between extreme capacity of the organism to handle excess zinc in the heart did not differ from controls. These results also show the heart did not differ from controls, muscle tissue, and the zinc level be noted. Levels in the kidneys, muscle tissue, and the diet. Only in the liver could a significant increase in the zinc were only slightly influenced by the high zinc content of the rapidly ^{65}Zn was eliminated. Tissue levels of stable zinc longer the exposure to the high zinc level in the diet the more length of exposure. Also analysis of tissues revealed that the exposure, whereas the fecal elimination of ^{65}Zn increased with in all groups except the control group irrespective of length of

The amount of radioactive zinc was, at all exposure levels, only a few percent of the amount found in the controls. There were no obvious changes with increasing dietary zinc, except in the tibia where, at the highest levels, there occurred an increase compared to the previous levels. In the heart and muscle tissue there was a slight but continuous decrease. In the liver and kidneys there was no change. The authors concluded that the data indicated that there was a good homeostatic control in the range 2,400 to 7,200 ppm. The authors also concluded that the homeostatic regulation was a good one week after calves given a diet containing 600 mg/kg zinc was much more effective in the rat than in calves. Stake, et al. (1975) found that calves given a diet containing 600 mg/kg zinc had considerably higher zinc levels in the liver, kidneys, and pancreas than calves fed a diet containing 34 mg/kg. There was, however, no change in heart or muscle zinc levels.

The topics of zinc essentiality and zinc deficiency have been extensively treated in the National Research Council report (1978) and also in a recent review by Prasad (1978). In 1934, it was shown by Todd, et al. (1934) that zinc was necessary for the growth of rats and since then many studies have been made on the development of the gonads. Prasad, et al. (1963) found that in certain villages in Egypt many subjects exhibited a syndrome characterized by dwarfism and anemia, hypogonadism, hepatosplenomegaly, rough and dry skin, and mental lethargy. There, young persons had a very low intake of animal proteins with bread as their staple food.

essentiality of zinc, including studies of humans.

In humans, zinc is necessary for normal growth and for normal development of the gonads. Prasad, et al. (1963) found that in certain villages in Egypt many subjects exhibited a syndrome characterized by dwarfism and anemia, hypogonadism, hepatosplenomegaly, rough and dry skin, and mental lethargy. There, young persons had a very low intake of animal proteins with bread as their staple food.

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Similar syndromes have been reported in other parts of the world. There are, however, studies that show that zinc deficiency with less pronounced symptoms may be more common than thought earlier. In the U.S., evidence of symptomatic zinc deficiency has been found in Colorado by Hambridge, et al. (1972). Zinc concentrations in hair were used as an index of the zinc status. Hambridge, et al. found that in 132 children ages 4 to 16, 10 children had hair zinc concentrations below 70 mg/kg, whereas most children had concentrations above 125 mg/kg. Eight out of ten of these children were found to have heights at the lower range for their age group. Poor appetite and a low intake of meat was thought to be one reason for the zinc deficiency. In these children hypogonadism (impaired taste acuity) was also found. After zinc supplement was normalized, 1-2 mg zinc sulfate/kg body weight/day for 1-3 months, this condition was paralleling the supplementation with zinc. In five children with hair zinc levels of 10 to 63 mg/kg before therapy, the levels were 67 to 170 mg/kg after four months of therapy. There are studies in other parts of the U.S. showing that low zinc levels were 67 to 170 mg/kg after four months of therapy. There are studies in other parts of the U.S. showing that zinc levels of 10 to 63 mg/kg before therapy, the levels were 67 to 170 mg/kg after four months of therapy. There are studies in other parts of the U.S. showing that zinc levels of 10 to 63 mg/kg before therapy, the levels were 67 to 170 mg/kg after four months of therapy.

main food. Zinc deficiency was demonstrated by the finding that zinc concentrations in plasma, red cells, and hair were decreased; that subjects had a higher turnover of radioactive zinc than normal; and that the excretion of zinc in feces and urine was less than in controls. Improvements were seen after oral administration of zinc, with a still greater effect observed upon additional protein supplementation.

As has been discussed earlier, human milk seems to contain a heteropatricia which generally occurs in children after weaning. There is also a rare congenital disease called acrodermatitis enteropathica which occurs in children after weaning.

(4) Zinc deficiency after operations such as intestinal bypass surgery (Atkinson, et al. 1978; Weissman, et al. 1978) The signs noted are generally changes in the skin and hypoguesia.

(3) Zinc deficiency after excessive alcohol ingestion (Ecker and Schroefer, 1978; Weissman, et al. 1978); and

(2) Zinc deficiency after intravenous hyperalimentation, which might lead to increased excretion of amino acids in urine, because of the large amounts of amino acids in zinc infusion fluids (Bergström and Leyden, 1978; Freedman, et al. 1975);

(1) Zinc deficiency in dialysis patients, which has been attributed to depletion of body zinc stores (Atkin-Thor, et al. 1978);

In addition to nutritional zinc deficiency, which is caused solely by a low dietary zinc intake, there are instances of zinc deficiency which are thought to have other causes. These are:

The reason for the signs and symptoms caused by the zinc deficiency is not clear, but it is known from a number of studies in a variety of organisms including human beings (NRC Chapter 8) that zinc is an essential constituent of many metalloenzymes. Typical examples of such metalloenzymes are alcohol dehydrogenase, carboxypeptidase, leucine aminopeptidase, alkaline phosphatase, carbonic anhydrase, RNA-polymerase, and DNA-polymerase. Also, thymidine kinase is thought to be a zinc dependent enzyme. Zinc may be involved in the synthesis and catabolism of RNA and DNA.

Levels in children's hair are not an uncommon finding (Prasad, 1978).

Zinc deficiency will not be covered in this section since it has been discussed in a previous section; the emphasis will be on the effects caused by excessive exposure to zinc via inhalation or

EFFECTS

strictions period.

Prasad, et al. (1978b) have recently reported on experimental zinc deficiency in humans. They studied four male volunteers who were hospital patients with various diseases. They were given a diet containing Zn at a level of about 3 mg/day for several weeks. In order to decrease the zinc intake it was necessary to give sub-jects cereal protein instead of animal protein during the study. In all subjects considerable weight losses occurred during the zinc depletion period. The plasma zinc level decreased significantly in all subjects and in 3 of 4 subjects there was a decrease cantly in all subjects and in 3 of 4 subjects there was a decrease in zinc excretion. Connective tissue was analyzed in two patients; during the period of low zinc intake thymidine kinase activity could not be detected, whereas after zinc supplementation it became close to the normal values. Also, plasma alkaline phos-phatase activity decreased along with a decrease in plasma lactate dehydrogenase activity during zinc depletion. In the connec-tive tissue the RNA and DNA ratio showed changes during the re-tire tissue the RNA and DNA ratio showed changes during the re-

factor or factors necessary for the absorption of zinc. Signs in this disease may come from many organs, among them the skin, central nervous system, and the gastrointestinal tract. As in other zinc deficiencies in children, there will be retarded growth and hypogonadism. Large oral doses of zinc will correct the condition.

Most of our knowledge about metal fume fever and its relationship to exposure to zinc oxide fumes comes from the beginning of the century when there was extensive research on this acute type of poisoning (Drinker, et al. 1927, 1928; Sturgis, et al. 1927). Reviews on metal fume fever, often also containing case reports, have been published in large numbers (Anseline, 1972; Heggsted, et al. 1945; Kehoe, 1948; Roberts, 1957). Metal fume fever is described in all textbooks on occupational hygiene. In summary it should also be mentioned that metal fume fever has not only been associated with inhalation of zinc oxide fumes, but with many other metal fumes which may produce similar symptoms.

Acute, Subacute, and Chronic Toxicity

have been exposed to zinc oxide fumes (Hilderman and Taylor,
There was a report on acute emphysema in cattle reported to
respectively.

It was calculated that the subjects inhaled 48 and 74 mg zinc,
were exposed to zinc oxide fumes at a level of 600 mg zinc/m³.
the disease. In a study by Sturgis, et al. (1927) two subjects
of data on the levels of zinc oxide fumes in air that might cause
proteins which might act as sensitizing agents. There is a lack
metal fume penetrates deep into the alveoli, and combines with
mechanisms for this reaction. One reasonable theory is that the
there is no definite evidence for any of the proposed different
metal fume fever. Several theories have been put forward, but
McCord (1960) that there is an allergic basis for the mechanism of
also been given the name "Monday fever." It has been suggested by
further exposure will not cause new symptoms. This disease has
when the worker has not been exposed for a couple of days, and
fume fever generally occurs at the beginning of the working week
metal fume fever, nor does it cause long-term sequelae. Metal
is the most prominent. There have never been any fatalities from
influenza-like symptoms such as headache, fever, hypertension, sweat-
ing, and muscle pains. Among the laboratory findings leukocytosis
fever is symptom occurrence within a few hours after exposure.
The symptoms may persist for 1 to 2 days and are characterized by
not capable of producing the disorder. Typical for metal fume
deep into the alveoli. Zinc oxide dust or other metal dusts are
duced metal fumes (McCord, 1960; Rohrs, 1957) which can penetrate
metal fume fever only appears after exposure to freshly pro-

Batchelor, et al. (1926) made an extensive investigation of malized workers exposed to zinc in a smelter in New Jersey. The authors

Millichken, et al. (1963). The effects of inhalation of zinc chloride in smoke from smoke bombs have also been described by Schmalz (1974) who reported on 11 cases, of which 2 had very severe reactions including edema of the lungs. However, no severe sequelae were seen. In one case, how-ever, it was almost two years before the lung function was nor-mal.

Further details on exposure to zinc chloride are provided by the hydrochloric acid component in the smoke may contribute. The pointed out that the toxic action may not only be due to the zinc spaces may rapidly lead to severe disease, but it should be tarry exercises. Accidental inhalation of such smoke in confined coming from so-called "smoke bombs" which are often used in military exercises. Major component in smoke inhalation of zinc chloride which is the major component in smoke that other metals (such as cadmium) might have been responsible either that cattle are extremely sensitive to zinc oxide fumes or but the extremely severe condition caused by the fumes showed mal's examined. In this case, a galvanized material was suspected liver, kidney, and lungs were not above normal values in two animals examined. Autopsy showed severe changes in the lungs with edema, emphysema, and hemorrhages. Zinc concentrations in time all three died. Autopsy showed severe changes in the lungs and arc welding of galvanized pipe were done during remodeling of the barn. Three heifers were severely affected and within a short time all three died. Autopsy showed severe changes in the lungs with edema, emphysema, and hemorrhages. Zinc concentrations in the lungs were not above normal values in two animals examined. Autopsy showed severe changes in the lungs and all three died.

pointed out that this smelter was well suited for studies on
 arsenic effects of zinc since the amounts of lead, cadmium, and
 chronic effects of zinc were very low compared with other types of zinc
 arsenic in the ore were never processed in other parts of the U.S. Of a total work force
 of 1,620 men, a number of workers were selected from different
 work areas for the special studies. Twelve men were selected from
 bag rooms where zinc oxide was handled. From a zinc oxide packing
 house five men were selected; four of them never wore respirators.
 From another zinc oxide plant two men were selected and two men
 were selected from a plant handling metallic zinc. Finally, three
 workers from a lithopone packing house were selected. A number of
 bag houses an average concentration of 14 mg/m³ was observed. In
 other workplaces mean concentrations were generally below 35
 mg/m³. In the zinc dust plant a maximum concentration of 130
 mg/m³ was measured. The 24 subjects under went a number of ex-
 aminations which included x-rays, physical examinations, inter-
 views, blood pressure measurements, and measurements of zinc in
 urine, and feces. Regarding the laboratory findings, it
 may be noted that 14 of the 24 men showed a slight leukocytosis;
 hemoglobin was reported to range between 72 and 97 percent with an
 average of 81 percent (100 percent is assumed to be 160 mg/l).
 Twenty-four-hour zinc elimination via feces in controls was re-
 ported to vary from about 4 to 20 mg, with an average of 9.32 mg,
 which is in good agreement with present daily values. In the ex-
 posed subjects, 24-hour excretion of zinc via feces averaged 46.8
 mg which indicates an exposure via the gastrointestinal tract or
 posed subjects, 24-hour excretion of zinc via feces averaged 46.8

4, and 7 weeks and at the end of the experiment. For most parts micron. A number of lung function tests were performed after 2, 4, or 8 hours a day to a concentration of 15 mg/m³ of zinc oxide, at particle size less than 1 micron. They were exposed for 1, 4, or 8 hours a day to a concentration of 15 mg/m³ of zinc oxide. The rats were divided into groups so lungs in an 84-day study. The rats were divided into groups so Pistorius (1976) studied the effect of zinc oxide on rat

levels around 100 mg/m³ in several places.

Also, the total dust was high in some workplaces with 120 mg/m³, with mean concentrations varying from 2 to 20 mg/m³. Nitrogen oxides were high in some workshops, the highest being exposed to substances other than zinc oxide. For example, levels is obvious that in this study many of the workers must have been also in control groups changes were noted during the workday. It enzyme activities were determined before work and after work. Other laboratory investigations gave no conclusive results. Some laboratory investigations showed a tendency to leukocytosis, but value of 58 mg/m³ was found during welding on one occasion. Variied between 1.7 and 18 mg/m³ of zinc oxide, but a maximum experienced metal fume fever several times. Exposure levels work in the shipyard and 10 shipyard workers not exposed to zinc oxide were used. Interviews showed that most of the workers had operations. As a control group, 10 healthy subjects who did not 60 shipyard workers who were exposed to zinc oxide in different Chmielewski, et al. (1974a,b) reported on the examination of

a smelter for decades without any symptoms of chronic disease. Authors was that the workers could be exposed to zinc compounds in massive excretion into the intestine. The conclusion of the

Zinc stearate is a compound other than zinc oxide which is often encountered in the plastic industry and is suspected of causing lung disease. Votila and Noro (1957) reported on a fatal case involving a worker employed for 29 years in a rubber plant. The autopsy showed the cause of death was a diffuse fibrosis of the lungs which histochimical examination of the lungs showing increased deposits of zinc. However, no quantitative determinations of the zinc content of the lung were made. The role of zinc stearate as a cause of chronic lung disease has since then been discussed by Harding (1958) and by Weber, et al. (1976). Harding gave rats intratracheal instillations of 50 mg of zinc stearate which caused the deaths of about half of the animals. In the survivors (living up to 259 days after instillation of the compound) fibrosis could not be detected. Harding also found that the zinc stearate had disappeared from the lungs within 14 days. Weber, et al. described autopsy findings in a man who was employed for the last eight years of his life in a plastics industry and who was exposed to zinc stearate. Fibrosis was found in the lungs with the zinc content of 62 mg/kg of lungs on a dry weight basis (Weber, et al. 1976). The same authors found that 30 persons from the same area had concentrations between 3.3 to 69.3 mg/kg of zinc in lungs. The man had also had other occupations, but his exposure to silica quartz in another occupation could not explain the fibrosis. The authors concluded that zinc stearate could not have caused lung disease.

tion of inhaled zinc from the lungs, and that the absorbed zinc is rapidly eliminated from the body through the homeostatic mech-

During the last years there have been some reports on copper deficiency in human beings after treatment with zinc. Prasad, et al. (1978a) and Porter, et al. (1977) have reported hypocupremia

Hallbook and Lanner (1972) gave 13 patients with leg ulcers zinc sulfate in oral daily doses of 600 mg, corresponding to 135 mg of zinc per day. Treatment lasted for 18 weeks. Fourteen patients were given a placebo. Blood counts, liver function tests, and urine analysis did not show any significant differences between patients given zinc and the placebo. Serum levels of zinc rose among patients with an initial level of <1.1 mg/l from an average of 0.95 mg/l to 1.57 mg/l after six weeks of treatment, but no further increase was noted. Among patients with an initial serum level >1.1 mg/l no increase in zinc levels was noted during the 18 weeks of treatment. Copper concentrations were not measured.

In animal experiments it has been shown that zinc may interfere with copper metabolism and that when the intake of copper is low, excessive zinc may induce a copper deficiency and anemia (NRC Chapter 9 pp. 256-257; Underwood, 1977; Hamilton, et al. 1979; Murthy and Petering, 1976). The animal data indicate that prolonged excessive intakes of zinc may constitute a hazard in patients treated with oral zinc supplements.

Lev^{els} were between 0.84 and 1.92 mg/l. During the study a number of laboratory investigations were undertaken on several occasions, but copper levels were not determined. No ill effects could be noted from the treatment with zinc and there were no changes in

addition, lead in the amount of 800 mg/kg. The groups were compared containing 5,400 mg/kg of zinc and another group received, in areas near smelters. Willoughby, et al. (1972) gave foals a diet especially, have been described and related to exposure to zinc in smelters. In foals, some symptoms, lameness and joint afflictions lead poisoning has occurred in horses living near lead-zinc and 2,040 mg/kg in two analyzed livers.

Liver were extremely high, measured on a dry matter basis, 1,430 that copper levels were lower than normal. Zinc concentrations in myocardium, kidneys, and liver. There were also some indications findings showed severe pulmonary emphysema with changes in both effected that they died or had to be slaughtered. The post-mortem enteritis. One one farm, 7 out of 40 cows were so severely exposure was only for a couple of days but it resulted in severe would correspond to an intake of 140 g of zinc per cow per day. cows had an intake of about 7 kg/day of these dairy nuts, which that the zinc concentration was 20 g/kg. It was stated that the by dairy nuts which had been contaminated by error with zinc so described by Allien (1968), the zinc poisoning of cattle was caused zinc poisoning has occurred in cattle. In the outbreak treated (sickle-cell anaemia and coeliac disease).

be noted that in both studies patients patients with severe diseases were 660 mg zinc sulfate may be too high for long treatment. It should were seen, but Porter, et al. pointed out that the daily doses of correct the hypocupremia. No chronic effects of the treatment mg/day, i.e., 150 mg zinc per day. In both cases it was easy to after a long-term treatment with zinc sulfate in doses of 660

Aughey, et al. (1977) gave zinc (as the sulfate) to mice for up to 14 months in drinking water at a concentration of 500 mg/L. The concentrations of zinc in feed for controls and exposed animals were not stated. That zinc is readily absorbed was seen by a rapid rise in plasma concentrations of zinc during the first days of exposure. During six months no difference between the first days and exposed animals could be shown regarding zinc concentrations in the liver, spleen, and skin nor was there any difference between the sexes. Histological examination showed that several endocrine glands were affected by the administration of zinc. Hyperthyroidism was found in the adrenal cortex; in the pancreatic islets and in the pituitary gland changes consistent with hyperactivity were noted.

Paraded with a control group and a group given only the excessive amounts of lead. It should be mentioned that the groups consisted of only 2 or 3 animals each. In three animals given excessive amounts of zinc, bone changes, especially in the epiphyseal areas of the long bones, were noted as a first sign. Later the animals had difficulties in standing and walking. In animals given lead and zinc the symptoms associated with exposure to zinc dominated. There were fewer effects from the exposure to lead and zinc than in animals given only lead. It should be noted that in this experiment, exposure to zinc was extremely high, but taken together with the other reports on actual findings in animals living near smelters, it is obvious that exposure to zinc in high amounts may constitute a hazard to horses.

Kang, et al. (1977) gave rats, by pair-feeding, diets containing 1.3, 55, and 550 mg zinc/kg of feed for four weeks. The animals were killed after that time and tissue concentrations of zinc and a number of other metals were determined. The low zinc diet gave typical signs of zinc deficiency, whereas there was no difference in weight gains and food efficiency ratios in the two groups given higher amounts of zinc; this fact, according to the authors, suggested that the highest level (550 mg/kg) was not toxic. Liver and kidney concentrations of zinc were slightly higher in the group given the largest amount of zinc, but no difference was noted in the heart. Iron concentrations in liver were higher in the group given the highest amount of zinc, but no difference in the heart. Zinc concentrations in the liver and kidney could be seen between the two highest zinc levels. In the kidney there was also a tendency for decreasing iron concentrations with increasing zinc intake as well as for copper, but there was no difference between the two highest zinc levels, nor practically any difference between the two highest dose levels, nor was there a difference in magnesium.

In pigs given zinc in the diet in concentrations ranging from 500 to 8,000 mg/kg, Brink, et al. (1959) found that signs of toxicity in the form of weight gain and feed intake were seen at levels above 1,000 ppm. In pigs, given from 2,000 ppm and higher, deaths occurred as soon as two weeks after exposure and severe gastrointestinal changes were seen with hemorrhages. There were also signs of brain damage due to hemorrhages. Changes in the joints were also seen, mainly in the form of swollen joints. In pigs given zinc in the diet in concentrations ranging from 500 to 8,000 mg/kg, Brink, et al. (1959) found that signs of toxicity in the form of weight gain and feed intake were seen at levels above 1,000 ppm. In pigs, given from 2,000 ppm and higher, deaths occurred as soon as two weeks after exposure and severe gastrointestinal changes were seen with hemorrhages. There were also signs of brain damage due to hemorrhages. Changes in the joints were also seen, mainly in the form of swollen joints. In pigs given zinc in the diet in concentrations ranging from 500 to 8,000 mg/kg, Brink, et al. (1959) found that signs of toxicity in the form of weight gain and feed intake were seen at levels above 1,000 ppm. In pigs, given from 2,000 ppm and higher, deaths occurred as soon as two weeks after exposure and severe gastrointestinal changes were seen with hemorrhages. There were also signs of brain damage due to hemorrhages. Changes in the joints were also seen, mainly in the form of swollen joints.

Acute effects of hemodialysis have been described by Gallerly,
et al. (1972). A woman on home dialysis used water stored in a
galvanized tank and two hours after the first dialysis at home she
had symptoms including nausea, vomiting and fever. Similar severe
symptoms were experienced by her at two subsequent dialyses, but
subsided between dialyses. Dialyses at the hospital were then
done without any symptoms, but she had symptoms again when she
started dialysis at home. At new admission to the hospital she
was found to be severely anemic. It was then found that the zinc
concentration in the tank water was 6.25 mg/l. The patients's

It has been reported that zinc and copper could be introduced in excessive amounts into the blood during hemodialysis (Blomfield, et al. 1969). Petrite and Row (1977) described nine cases of anemia in dialysis patients due to the release of zinc from a galvanized iron tubing in the dialysis system. Copper levels were not measured in these cases but there was a rise in hemoglobin concentrations after removal of the source of zinc.

In a woman given total parenteral nutrition after an operation, acute zinc poisoning occurred due to an error in prescription. During a period of 60 hours she received 7.4 g of zinc sulfate. She became acutely ill with pulmonary edema, jaundice, and oliguria, among other symptoms. The serum zinc concentration was 42 mg/L. In spite of treatment she remained oliguric and hemodilysis did not improve renal function. She died after 47 days of illness (Brooks, et al. 1977).

Liver samples from these pigs, levels of zinc above 1,000 mg/kg wet weight were found.

The relationship between zinc and cancer has been reviewed earlier by the NRC (1978) (Chapter 7 pp. 208-209, Chapter 9 pp. 231-234 and Chapter 10 pp. 258-261) and by Sunderman (1971). It was concluded that during certain experimental conditions, injection of zinc salt into the testes could induce testicular tumors. There was no evidence that zinc given via the oral route or parenteraly could cause tumors. However, zinc is of interest with regard to cancer since zinc seems to be indirectly involved by being necessary for DNA and RNA synthesis. It has been shown that zinc-deficient rats tumor growth was reduced (Pethering, et al. 1967; Dewys, et al. 1970). These earlier findings have recently been confirmed in other studies.

The effect of different levels of dietary zinc on the development of chemically-induced oral cancer in rats has recently been studied by Wallenius, et al. (1979) and Mather, et al. (1979). In the study by Wallenius, et al. (1979), three groups of female rats were fed diets for three weeks which contained 15 mg/kg, 50 mg/kg, and 200 mg/kg of zinc, respectively. The palatal mucosa was then painted with the carcinogen 4-nitro-quinoxaline-n-oxide three times a week. The animals were killed after cancer could be observed macroscopically in the oral cavity. It was noted that the tumor growth was reduced at the higher zinc levels. The tumor incidence was 100% at the control level, 50% at the intermediate level, and 10% at the highest level. The tumor size was also reduced at the higher zinc levels. The results indicate that zinc may have a protective effect against oral cancer.

Teratogenicity, Mutagenicity, and Carcinogenicity

Zinc concentration in red cells was 35 mg/l and after six weeks dialysis in the hospital it was reduced to 12 mg/l. During the same period plasma levels decreased from 7 mg to 1.58 mg/l. Blood copper was not decreased.

cience in itself has any etiological role in human cancer. However regarding human beings, there is no evidence that zinc deficit 21 percent and zero (0) percent.

deficiency and at a total of four doses the corresponding figures were given figures of 79 and 29 percent, respectively, for tumor incidence between groups was most significant; a total of eight doses the groups given the lowest doses of the carcinogen, the difference fed an adequate intake of zinc. It was also noted that in animals more carcinomas of the esophagus were found than in animals on an adequate intake of zinc-deficient carcinogen. A general finding was also that in zinc-deficient animals on an adequate intake of zinc, but which were given the deficient animals than in controls, but they were also lower in zinc. As expected, zinc levels in the esophagus were lower in zinc gen. Finally, there was one experiment where the exposure was only for two weeks for a total of four doses of the carcinogen for four weeks and animals were sacrificed 63 days after the start of exposure. Finally, there was one experiment where the carcinogen was similar but the carcinogen was administered after four weeks later. In a third experiment the carcinogen was given were killed at the end of exposure and some animals were killed five weeks later. Some animals were killed with the length of exposure of nine weeks. Some animals were killed at the end of exposure and some animals were killed after four weeks with an another experiment the carcinogen was administered after four weeks on these diets the carcinogen methylbenzylnitrosamine was administered by intragastric intubations twice weekly in doses of 2 mg/kg body weight for 12 weeks. In another experiment the group of rats was fed a diet containing 7 mg/kg of zinc. After 12 weeks on the diet the carcinogen methylbenzylnitrosamine was developed of cancer is the study by Fong, et al. (1978). One group of rats was fed a diet containing 60 mg/kg of zinc and one group of rats was fed a diet containing 7 mg/kg of zinc. After 12 weeks on these diets the carcinogen methylbenzylnitrosamine was developed by the same diet.

ever, many studies have been performed on the levels of zinc in both malignant and nonmalignant tissues in human beings. The zinc concentrations have been found to be both low and high and Chapter 10 pp. 259-261). As an example it has been shown that in cancer of the esophagus in human beings zinc concentrations were lower than normal which is in accordance with the above mentioned experiments on rats (Lin, et al. 1977). However, there is one organ in the human being where there seems to be a more consistent pattern, the prostate gland. It has been discussed earlier that zinc concentrations in the prostate normally are very high. There has been a consistent finding that in cancer of the prostate there is a decrease in zinc in the carcinomatous tissue or in hyperrophic prostate. These authors also reported that the cadmium levels were higher in the carcinomatous tissue than in the normal or hyperrophic prostate. In the study by Habib, et al. (1976), zinc concentrations in the neoplastic tissue were less than half of the concentrations in normal tissue or in hyperrophic prostate. These authors also reported that the cadmium has been implicated as a possible cause of prostate cancer and since there are interactions between cadmium and zinc, this might have some bearing on the problem of the relationship between zinc and cancer of the prostate. Habib (1978) has reviewed the role of zinc in the normal and pathologic prostate.

zinc in the hyperplastic tissues as in normal tissue. There is one exception; the study by Gyorkay, et al. (1967) found considerably increases in zinc levels in hyperplastic tissue - more than three times the normal.

The mutagenic effects of zinc have been discussed by the National Research Council (Chapter 10 p. 261) which could not find literature that suggested that zinc is mutagenic in animals and human beings nor have any new data appeared on this subject. The same conclusions are made with regard to teratogenesis. The zinc might have an effect since long-term supplements with large amounts of zinc will cause disturbances in copper metabolism.

In a study by Cox, et al. (1969), it was shown that if rats were fed a diet containing 4,000 ppm of zinc during gestation, zinc concentrations increased. Ketcheson, et al. (1969) fed rats zinc concentrations increasing up to 5,000 mg of zinc/kg during gestation. Even diets containing up to 5,000 mg of zinc/kg during gestation, zinc concentrations increased. At that level malformations were not observed, but there was a reduction in the copper concentrations of the fetal liver.

A brief statement in a report by Kumar (1976) states that in a small group of women supplements of zinc administered during the third trimester of pregnancy in a dose of 100 mg of zinc sulfate per day (23 mg zinc per day) caused premature births and one still-birth in four consecutive subjects. Kumar then made studies per day (23 mg zinc per day) caused premature births and one per day (23 mg zinc per day) caused zinc sulfate.

(it is not quite clear how the dose was calculated, but it is in rats and gave them a daily supplement of 100 ppm zinc orally to rats and a small group of women supplements of zinc administered during the third trimester of pregnancy in a dose of 100 mg of zinc sulfate per day (23 mg zinc per day) caused premature births and one still-birth in four consecutive subjects. Kumar then made studies per day (23 mg zinc per day) caused zinc sulfate.

exposure to cadmium would cause changes in the distribution of ture up to 1974 was reviewed and discussed. It was concluded that discussed in the NRC report (Chapter 10 pp. 261-268) and the literature between cadmium and zinc were extensively discussed.

Cadmium

(1977) and NRC (Chapter 7 pp. 186-187). Between zinc and other metals have also been reviewed by Underwood international meeting and reported (Norrborg, 1978). Interactions between metal interactions have recently been discussed at an international meeting and reported (Norrborg, 1978). Such metals may also have an influence on the metabolism of zinc. Other metals may but it is also possible that excessive intakes of other metals may affect zinc could influence the metabolism of iron and copper, takes of zinc could influence the metabolism of iron and copper, other metals may occur. It was demonstrated that excessive intake effects of excessive intakes of zinc, interactions between zinc and As has already been discussed in the section concerning effects.

INTERACTIONS OF ZINC WITH OTHER METALS

180).

might have an adverse effect on the fetus (NRC Chapter 7 pp. 179- mentioned in animal experiments that zinc deficiency during pregnancy between zinc and copper, excessive zinc intakes during prolonged times could have an adverse effect on the fetus. It is well documented that has been recommended, but due to the known interaction between zinc and copper, excessive zinc intakes during pregnancy result in absorption of the implants. Supplementation for pregnant women has been recommended. Supplementation for pregnant animals there was a significant increase in the number of other nutrients in the diet was not stated. In the zinc-supple-

percent zinc sulfate solution"). The concentration of copper and stated in the report "received additinally 150 ppm zinc as a 2

ly used laboratory animals), exposure to cadmium will result mainly small laboratory animals. In the rat especially (the most commonences between some large mammals (e.g., man, horse) compared to Elinander and Piscator (1978) showed that there are clear differences with regard to the human situation. A review of the literature by formed, there might be some difficulties in drawing conclusions although a large number of animal studies have been performed (Friborg, et al. 1974).

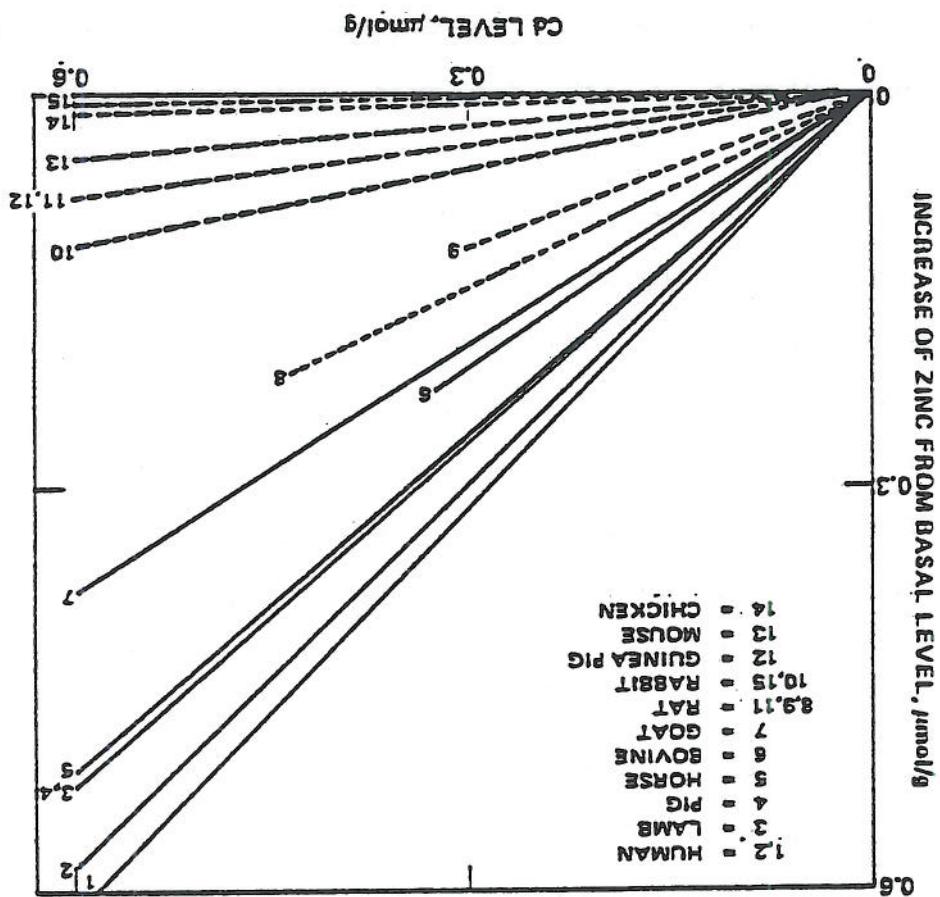
Cadmium related to the occurrence of renal tubular dysfunction renal cadmium corresponds to the critical level which has been estimated for thiominein would be close to zero (Nordberg, et al. 1979) and that 200 mg/kg wet weight of cadmium the amount of zinc in metallothionein would increase. It was also shown that at a level of about zinc will increase. It with increasing cadmium concentrations the ratio of cadmium to about equimolar amounts of zinc and cadmium in metallothionein, whereas at low levels of cadmium in the kidney there are that whereas at low levels of cadmium in the kidney have been shown ence of cadmium in metallothionein. It has recently been shown 1978). The increase in renal zinc is also related to the occurrence both in human beings and in horses (Elinander and Piscator, 1977, these earlier findings have recently been confirmed in new studies up to cadmium concentrations of about 60 mg/kg wet weight. The increases in cadmium and that this increase is nearly equimolar horses the increase in renal concentrations of zinc is parallel to and kidney. It has also been shown that in both human beings and laters. In animals on marginal zinc intakes there could be a zinc deficiency in certain organs parallel with the increase in liver zinc with increases in liver and kidney where cadmium also accumulates.

143 mg/kg in the animals at the higher level of exposure. The testes of zinc-deficient animals were 104 mg/kg, compared to 17.2 mg/l of cadmium in drinking water. Zinc concentrations in diet contained 40 mg zinc/kg. The exposure in that experiment was zinc, 5 mg/kg feed, whereas such lesions were not seen when the testicular and pulmonary lesions in rats on a marginal intake of zinc (Lal 1976) found that oral exposure to cadmium could cause levels are affected.

Rat fetuses a decrease of copper and iron occurs before the zinc content increased. Data by Choudhury, et al. (1978) indicate that in the dam paralleling the accumulation of cadmium as previously in the fetus is thought to be due to retention of zinc significantly. This is since cadmium does not pass the placental barrier to any orally. Since cadmium was found in rat pups that had been given cadmium birth weight were found in the fetuses. Pond and Walker (1975) showed that both low zinc and copper concentrations and decreases in mineral distribution in the fetuses. If animals are exposed to cadmium during the gestation period, this may also influence the is known to cause effects on the fetuses. If animals are exposed to between species are illustrated in Figure 1. Zinc deficiency alone concussions from experiments done with rats. The differences between increases in renal copper concentrations make it reasonable to conclude that one must be cautious when drawing is rather small. On the other hand, exposure to cadmium causes increases in hepatic zinc, whereas the increase in renal zinc

Increase of zinc as a function of increasing cadmium concentration in kidney of 11 different species. The references are in the paper by Elinder and Piscator (1978). The data are taken from nine publications.

FIGURE 1



and combinations of these metals were added in amounts of 100 mg/kg of molybdenum. To these diets zinc, copper, or molybdenum containing 18 mg/kg of copper, 70 mg/kg of zinc, and less than 1 cinnamon and Buncle (1965) fed groups of rats a basic diet with copper.

available and that at such sites there would be no competition for zinc binding sites on plasma albumin would be larger number of zinc binding sites on the zinc deficient rats a finding by suggesting that in the zinc deficient rats a influence on the uptake of ^{65}Zn . The authors tried to explain from the gut, but in zinc-supplemented rats excess copper had an active amounts of copper did not influence the uptake of ^{65}Zn Evans, et al. (1974) found that in zinc deficient rats excess-

Murthy and Peterling, 1976; Allen and Klevay, 1978).

factor with regard to cholesterol levels (Peterling, et al. 1977; since been shown that it is the copper status that is the main curved with an increasing zinc-copper ratio in the diet. It has though Klevay (1973) found that in rats hypercholesterolemia occurring heart disease. The main reason for this may be that the copper content of the typical American diet is less than the coronary heart disease. The main reason for this may be that the ratio between copper and zinc in the American diet contributes to gested by Klevay (1975) and Klevay and Forbusch (1976) that the zinc and giving copper supplementation. It has also been suggested which can be easily corrected by decreasing the intake of mia, may cause copper deficiencies in human beings and result in ane-

The resorption rate in the uterus was lower and there was a larger results showed that in females deficient in both calcium and zinc day of gestation, and the fetuses were removed and examined. The of calcium per kg of feed. The animals were killed on the last of calcium diet which contained the same amount of zinc but 15 mg deficient diet containing 0.4 mg zinc per kg or a zinc-deficient diet containing 0.4 mg zinc per kg. Between a zinc-deficient diet of gestation, female rats were given either a zinc-deficient diet interaction between zinc and calcium. Beginning on the first day study by Hurley and Tao (1972) shows an interesting example of influence when zinc intake is marginal. Also, Underwood (1977) has reviewed the relationships between zinc and calcium. The influence except for some indications that calcium could have an sorption except for some indications that calcium could absorb calcium levels in the diet do not influence zinc absorption included that calcium levels in the diet do not influence zinc absorption by NRC (1978) (Chapter 7 pp. 184-185). It was concluded that calcium on absorption of zinc from the gut.

Calcium

Per or molybdenum ingested at the same time as zinc. These data indicate that the levels of zinc retained and excreted are affected only by zinc dietary levels and not by levels of copper. Influence the retention or tissue distribution of the isotope. That even the very high level of copper or molybdenum did not bone retention and decreased urinary excretion of the isotope but found that an increase in dietary zinc resulted in an increased active zinc. After four days the animals were killed. It was of the experiment all animals were given an injection of radio-zinc. The length of the experiment was seven weeks. At the end mg/kg of copper, 1,800 mg/kg of molybdenum, and 5,000 mg per kg of

In the pups the zinc to copper ratio was considerably lower in the tended to decrease in the pups as a result of iron restriction. That the zinc to copper ratio in spleen increased in dams but both the dams and pups were killed and examined. It was found 5, 29, and 307 mg/kg of iron. Eighteen days after parturition zinc. Sherman, et al. (1977) gave pregnant rats diets containing metabolism, but much less is known about the effects of iron on As mentioned earlier, high intake of zinc may affect iron

Iron

metals can interact with each other. Not be available to the fetus. This study shows how two essential from bone and thus the large amount of zinc stored in bone would cienet and high calcium intake there would be no release of zinc and transported to the fetus, whereas in animals on a zinc-deficient diet. This suggested that zinc was released from bone during the release of calcium. This zinc could then be available deficienit diet. This also lower zinc content in the bones of rats on the calcium diet. This was interpreted as calcium being withdrawn from cienet diet. The bone during pregnancy to provide calcium to the fetus. There was a reduction in both ash weight and total calcium content of the femur in the females given the zinc-deficient and calcium-deficient diet. This was 57 percent. Analyses of maternal bone showed there was group was 57 percent. The corresponding figure for zinc-deficient and calcium-deficient females on the zinc-deficient diet showed malformations whereas the zinc-deficient diet. Eighty-three percent of the fetuses from the corresponding figure for zinc-deficient and calcium-deficient females on the zinc-deficient diet. Eighty-three percent of the rats given only number of live births per litter than among the rats given only

group. There were also reductions in the zinc levels in liver and caused reduced plasma zinc levels also in the high zinc level zinc group but increased erythrocyte zinc. Further exposure of lead. Lead exposure decreased the plasma zinc in the low level given drinking water containing 5 and 50 mg/l of zinc and 100 mg/l in tissues as was shown by El-Gazzar, et al. (1977). Rats were lead will also have an influence on the zinc concentrations less. It was concluded that main interaction was in the gut with high zinc intake, but also the hematological changes were increased. The lead concentrations in tissues were lower in animals dietary zinc concentrations the symptoms of lead toxicity decreased dietary zinc concentrations of 50 and 200 mg lead per kg fed. They found that with higher given 50 and 200 mg lead per kg fed. Of three dietary levels of zinc (8, 35, and 200 mg/kg) on rats take of zinc. Cerklewski and Forbes (1976) studied the influence of zinc. There was a lower uptake of lead in animals with high intakes; there was a lower uptake of zinc and there seem to be some interactions exposure to lead and zinc and there can be stimuli.

Lead

for both iron and zinc. It was concluded that there were some common mucosal binding sites but was inhibited by adding iron to the duodenal loop system used. Zinc in iron-deficient mice and found that zinc uptake from the hamilton, et al. (1978) studied the intestinal absorption of clated with decreased ratio of zinc to copper in the tissues. were seen between groups with high and low iron intake. In the iron-deficient pups increased levels of serum lipids wereasso-

both on contraceptives and not on contraceptives. The greatest HESS, et al. (1977). Uterine zinc excretion decreased in women women on a low intake of zinc, copper, and iron was studied by the influence of oral contraceptives on the excretion of zinc in that contraceptive pills have an influence on zinc metabolism.

In the previous chapters it has been mentioned several times

Interactions Between Zinc and Drugs

high lead and high zinc intake together. Lead exposure to zinc caused large reductions in serum iron than the exposure to zinc caused increases in ALA excretion caused by lead was reduced by the additional exposure to the high level of zinc.ings indicate that the increase in ALA excretion caused by lead calcium in the diet, 0.9 and 0.1 percent, respectively. The findings therefore. These diets were also combined with two levels of tions of 5,000 mg/kg of lead or 6,300 mg/kg of zinc and combinations of 5 mg/kg of zinc and 7 mg/kg of lead and then groups were given added mg/kg of zinc and 7 mg/kg of lead. Thawley, et al. (1977) gave rats a basic diet containing 30

al. 1978; Thomasino, et al. 1977).

al. 1976; Bordet, et al. 1976; Finelli, et al. 1975; Thawley, et the excretion of ALA seen in lead-intoxicated rats (Abdulla, et regarding the ALA dehydratase activity, and that zinc decreases and in vitro it has been shown that zinc is antagonistic to lead dependent enzyme, in blood. In a number of studies both in vivo

is the effect of zinc on the activity of ALA dehydratase, a zinc An effect which has attracted great interest the last years tition of zinc.

tibia of both groups. There was no change in the brain concentra-

The usual intake of zinc in these women before the study started was estimated to be about 10 mg/day. During the study the average intake averaged only 0.17 mg/day. At the beginning of the study, before the zinc intake was lowered, the average excretion of zinc in urine was 0.36 and 0.4 mg, respectively, for the group on contraceptives and for the control group. These data indicate that whereas contraceptives will have relatively little influence on zinc metabolism during normal zinc intake, they may have a more profound influence when the zinc intake is low. In this study the zinc intake was extremely low.

Many other drugs, especially drugs with chelating properties, increase the excretion of zinc. Substances in food, such as tannins, can influence the absorption of zinc. Alcohol will have an influence on zinc metabolism especially if a state of chronic phytate, can influence the absorption of zinc. Such cases often have low serum levels of zinc and an increased alcoholism has been reached with circrohotic changes in the liver.

Such cases often have low serum levels of zinc and an increased influence on zinc metabolism especially if a state of chronic alcoholism has been reached with cirrhoctic changes in the liver.

excretion.

change was in the contraceptive group with a decrease of 83 percent; a 62 percent decrease was seen for those not on contraceptives.

The present standard for drinking water, 5 mg/l, is based on organoleptic effects, i.e., some people will recognize the bitter taste caused by zinc present at such levels. The World Health Organization (WHO) has also proposed that the level should be 5 mg/l; however, the USSR has established a limit for zinc at 1 mg/l for other than health reasons (NAS, 1977).

There is no acceptable daily intake for zinc in food. As mentioned earlier, zinc is an essential nutrient and there has been no reason to restrict the zinc levels in food.

In 1974, the National Academy of Sciences recommended that adults should have an intake of 15 mg of zinc per day, that pregnant

The National Institute of Occupational Safety and Health (NIOSH, 1975) has recently reviewed the occupational hazards of exposure to zinc oxide and no changes were suggested regarding the existing standard for zinc oxide of 5 mg/m³. The American Conference of Governmental Industrial Hygienists (ACGIH, 1976) has an enforceable threshold limit value (TLV) for zinc oxide of 5 mg/m³ and adopted a workplace standard for zinc oxide of 5 mg/m³. The Occupational Safety and Health Administration (OSHA) (29 CFR 1910.1000) has a workplace standard for zinc oxide of 5 mg/m³, 8-hour time-weighted average. The TLV value has also been adopted in other countries. For zinc chloride a limit of 1 mg/m³ has been adopted by ACGIH and OSHA also adopted a standard of 1 mg/m³ for zinc chloride.

Existing Guidelines and Standards

CRITERION FORMULATION

Studies on experimental animals and on human beings given zinc for zinc is an essential element and is not a carcinogenic agent.

Basis and Derivation of Criterion

Persons subjected to such exposure at special risk because zinc oxide fumes may cause acute reversible reactions which may put copper may constitute another risk group. Occupational exposure to zinc oxide via food, for curing of various diseases caused by zinc deficiency or to promote wound healing may constitute a group at deficiency or to promote wound healing may cause copper deficiency or to cure various diseases caused by zinc deficiency may cause reversible health effects. Patients treated for excessive intake of zinc by people with a tendency to copper deficiency might cause reversible health effects. Patients treated for months or years with large oral doses of zinc salts, about 10 times the intake via food, for curing of various diseases caused by zinc deficiency may interfere with copper and other minerals, since zinc may interfere with copper and other minerals.

Special Groups at Risk

Smoking will contribute even less. A milligram of zinc emitting industries, be in the order of only a few tenths of average exposure to zinc via ambient air will, even in the vicinity may cause an additional daily intake of 2 to 10 mg of zinc. The may cause an additional daily intake of 2 to 10 mg of zinc. The zinc via drinking water will be only a few percent of the intake via food, but for some individuals the zinc concentration in tap water to 20 mg/day. For the majority of the population, the intake of ent intake of zinc via food for the adult U.S. population is from 10 It has been well established in several studies that the pres-

Current Levels of Exposure

mg/day of zinc (Table 3) (NAS, 1974). women should have an intake of 20 mg/day, lactating women should have 25 mg/day, and that pre-adolescent children should have 10 mg/day of zinc (Table 3) (NAS, 1974).

*Source: NAS (1980)

Age (Yrs.)	mg/day	Age (Yrs.)	mg/day	Age (Yrs.)	mg/day	11 - Adults	10.0	1 - 3
0.0 - 0.5	3.0	4 - 6	4.0	7 - 10	5.0	10.0	0.5	

Recommended Allowances (RDA) for Zinc*

TABLE 3

of 135 to 150 mg of zinc has been well tolerated by patients given long-term oral administration of zinc sulphate in daily doses available, the current criterion will be reconsidered. Therefore, it is reasonable that the current level of 5 mg/l become maintained for water quality criterion. As additional data become known instances of adverse effects occurring at current standard. Standard of 5 mg/l, based on organoleptic effects. There are no concentrations generally well below the current drinking water for this essential metal. Zinc is naturally present in water at the presence of zinc in drinking water contributes to the RDA at the present time.

The most common reported effect of high-level exposure to zinc is copper deficiency, which is readily reversible. The effect occurs at exposure levels at least an order of magnitude above the RDA for zinc. The data on special groups at risk for zinc-related copper deficiency are too sparse to include in criteria evaluation for zinc. The data on special groups at risk for zinc-related RDA for zinc. The data on special groups at least an order of magnitude above the RDA for zinc. Zinc is naturally present in water at the presence of zinc in drinking water contributes to the RDA at the present time.

The toxicological data base for evaluating water quality criteria for zinc is inadequate. While there is no evidence that zinc is carcinogenic, there is a lack of usable data on chronic effects. Most animal studies reported in the literature fail to include specific exposure data, while studies with humans are generally from special groups (e.g., patients receiving high-dose zinc therapy) case reports of accidental high exposure or based on data either case reports of accidental high exposure or based on data from special groups (e.g., patients receiving high-dose zinc therapy). The toxicological data base for evaluating water quality criteria for zinc is inadequate. While there is no evidence that zinc is carcinogenic, there is a lack of usable data on chronic effects. Most animal studies reported in the literature fail to include specific exposure data, while studies with humans are generally from special groups (e.g., patients receiving high-dose zinc therapy).

For long periods if the copper status is adequate. Exposed persons, indicate that large doses of zinc can be tolerated therapeutically purposes, together with observations of occupationally

the compound to promote wound healing. In patients with metabolic diseases such treatment might cause reductions in serum copper levels. Using a safety factor as high as 10, this means that an additional intake of 15 mg of zinc does not constitute any hazard. This corresponds to an intake of 2 liters of water containing 7.5 mg Zn/L. This concentration is above the present standard for drinking water which is 5 mg/L based on organoleptic effects.

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