United States
Environmental Protection
Agency

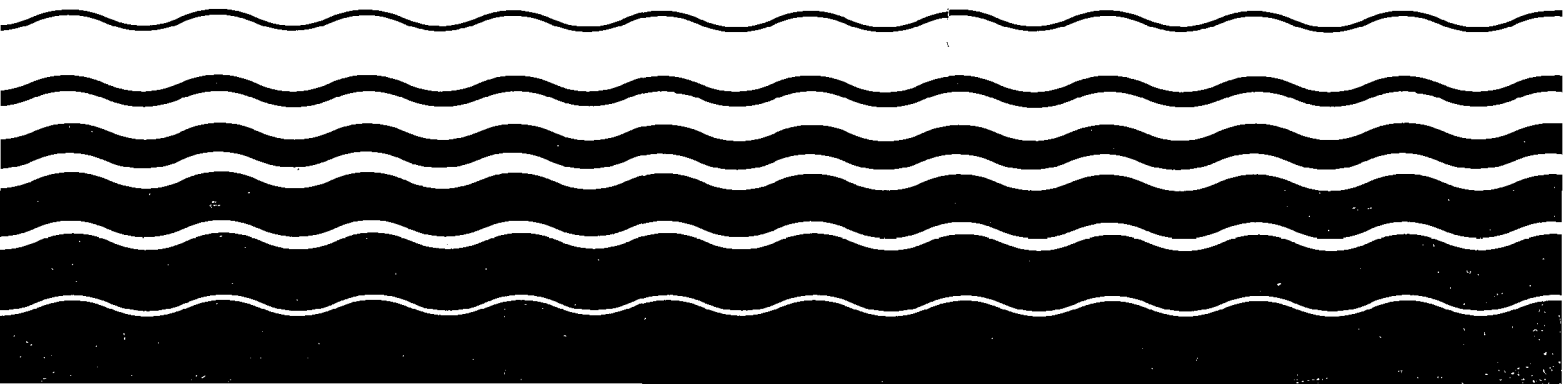
Office of Water
Regulations and Standards (WH-553)
Washington DC 20460

August 1980
EPA-440/4-81-016

Water

EPA

An Exposure and Risk Assessment for Zinc



DISCLAIMER

This is a contractor's final report, which has been reviewed by the Monitoring and Data Support Division, U.S. EPA. The contents do not necessarily reflect the views and policies of the U.S. Environmental Protection Agency, nor does mention of trade names or commercial products constitute endorsement or recommendation for use.

REPORT DOCUMENTATION PAGE	1. REPORT NO. EPA-440/4-81-016	2.	3. Recipient's Accession No.

Title and Subtitle

An Exposure and Risk Assessment for Zinc

5. Report Date: August 1980

6.

Author(s) Perwak, J.; Goyer, M.; Nelken, L.; Schimke, G.; Scow, K.; Walker, P.; and Wallace, D.

8. Performing Organization Rept. No.

Performing Organization Name and Address

Arthur D. Little, Inc.
20 Acorn Park
Cambridge, MA 02140

10. Project/Task/Work Unit No.

11. Contract(C) or Grant(G) No.
(C) 68-01-3857
(G)

Sponsoring Organization Name and Address

Monitoring and Data Support Division
Office of Water Regulations and Standards
U.S. Environmental Protection Agency
Washington, D.C. 20460

13. Type of Report & Period Covered: Final

14.

Supplementary Notes

Extensive Bibliographies

16. Abstract (Limit: 200 words)

This report assesses the risk of exposure to zinc. This study is part of a program to identify the sources of and evaluate exposure to 129 priority pollutants. The analysis is based on available information from government, industry, and technical publications assembled in August 1980.

The assessment includes an identification of releases to the environment during production, use, or disposal of the substance. In addition, the fate of zinc in the environment is considered; ambient levels to which various populations of humans and aquatic life are exposed are reported. Exposure levels are estimated and available data on toxicity are presented and interpreted. Information concerning all of these topics is combined in an assessment of the risks of exposure to zinc for various subpopulations.

17. Document Analysis a. Descriptors

Exposure
Risk
Water Pollution
Air Pollution
Effluents
Waste Disposal
Food Contamination
Toxic Diseases
Zinc

b. Identifiers/Open-Ended Terms

Pollutant Pathways
Risk Assessment

c. COSATI Field/Group 06F 06T

18. Availability Statement	19. Security Class (This Report)	21. No. of Pages
Release to Public	Unclassified	180
	20. Security Class (This Page) Unclassified	22. Price $17.50

EPA-440/4-81-016
August 1980

AN EXPOSURE AND RISK ASSESSMENT FOR ZINC

by

Joanne Perwak
Muriel Goyer, Leslie Nelken, Gerald Schimke, Kate Scow
Pamela Walker, and Douglas Wallace
Arthur D. Little, Inc.

and

Charles Delos
U.S. Environmental Protection Agency

EPA Contract 68-01-3857

Monitoring and Data Support Division (WH-553)
Office of Water Regulations and Standards
Washington, D.C. 20460

OFFICE OF WATER REGULATIONS AND STANDARDS
OFFICE OF WATER AND WASTE MANAGEMENT
U.S. ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

FOREWORD

Effective regulatory action for toxic chemicals requires an understanding of the human and environmental risks associated with the manufacture, use, and disposal of the chemical. Assessment of risk requires a scientific judgment about the probability of harm to the environment resulting from known or potential environmental concentrations. The risk assessment process integrates health effects data (e.g., carcinogenicity, teratogenicity) with information on exposure. The components of exposure include an evaluation of the sources of the chemical, exposure pathways, ambient levels, and an identification of exposed populations including humans and aquatic life.

This assessment was performed as part of a program to determine the environmental risks associated with current use and disposal patterns for 65 chemicals and classes of chemicals (expanded to 129 "priority pollutants") named in the 1977 Clean Water Act. It includes an assessment of risk for humans and aquatic life and is intended to serve as a technical basis for developing the most appropriate and effective strategy for mitigating these risks.

This document is a contractors' final report. It has been extensively reviewed by the individual contractors and by the EPA at several stages of completion. Each chapter of the draft was reviewed by members of the authoring contractor's senior technical staff (e.g., toxicologists, environmental scientists) who had not previously been directly involved in the work. These individuals were selected by management to be the technical peers of the chapter authors. The chapters were comprehensively checked for uniformity in quality and content by the contractor's editorial team, which also was responsible for the production of the final report. The contractor's senior project management subsequently reviewed the final report in its entirety.

At EPA a senior staff member was responsible for guiding the contractors, reviewing the manuscripts, and soliciting comments, where appropriate, from related programs within EPA (e.g., Office of Toxic Substances, Research and Development, Air Programs, Solid and Hazardous Waste, etc.). A complete draft was summarized by the assigned EPA staff member and reviewed for technical and policy implications with the Office Director (formerly the Deputy Assistant Administrator) of Water Regulations and Standards. Subsequent revisions were included in the final report.

Michael W. Slimak, Chief
Exposure Assessment Section
Monitoring & Data Support Division (WH-553)
Office of Water Regulations and Standards

TABLE OF CONTENTS

TABLE OF CONTENTS (Continued)

LIST OF TABLES

LIST OF FIGURES

ACKNOWLEDGMENTS

The Arthur D. Little, Inc., task manager for this study was Joanne Perwak. Other major contributors were Muriel Goyer (human effects), Leslie Nelken (environmental fate), Gerald Schimke (materials balance), Kate Scow (biological fate), Pamela Walker (materials balance), Douglas Wallace (biotic effects and exposure and monitoring data), Melba Wood (monitoring data), and Alfred Wechsler (technical review).

I. EXECUTIVE SUMMARY

MATERIALS BALANCE

Approximately 1.19 million metric tons (MT) of zinc were consumed in the U.S. in 1977, about half of which was imported. Zinc is used primarily in metallic form in galvanizing (41%), alloys and die casting (36%), brass (12%), and rolled zinc (3%) in construction, transportation, electrical, machinery and other industries. The remainder (8%) is used as zinc oxide and other zinc compounds, which are used in a wide variety of products, such as plastics, paper, paints and cosmetics.

Less than 10% of the zinc supply is recycled domestically. An unknown amount is accumulating within the economic system, and the remainder is released to the environment, primarily as solid wastes disposed of to land. Refuse comprised of spent products containing zinc, ore mine tailings, metals working wastes, coal ash, and municipal and industrial sludges constitute major sources of landfilled zinc. In addition, significant quantities of zinc are agriculturally landspread as fertilizer adjuvant.

The largest input of zinc to water results from erosion of soil particles containing natural traces of zinc (45,400 MT/yr). Culturally accelerated erosion accounts for 70% of this soil loss; geologic or natural erosion constitutes the other 30%. However, as this source is dilute and widely dispersed it is unlikely to result in significantly elevated aquatic concentrations. On the other hand, urban runoff (5250 MT/yr), inactive mine drainage (4060 MT/yr), and municipal and industrial effluents (17,000 MT/yr combined) are smaller but more concentrated sources, capable of affecting many local areas. Drainage from active mining areas is considerably less than from inactive areas due to the disposal methods currently employed.

POTW represent the largest total point source zinc discharges, receiving contributions from water supply and distribution system corrosion, combined sewer area runoff, industrial wastes, and human excrement. Industries with large discharges of zinc directly to water include iron and steel, zinc smelting (primarily from a single mill), and possibly plastics and electroplating.

The total quantity of zinc estimated to be emitted to air (7130 MT/yr) is a small portion of the total environmental release. Refuse incineration, coal combustion, and some metals working industries constitute the major sources. Along with releases of zinc through metal corrosion and tire abrasion, these sources contribute to urban runoff contamination.

DISTRIBUTION OF ZINC IN THE ENVIRONMENT

Zinc in ambient water is usually found at concentrations of less than 50 ug/l. However, in many locations concentrations of 100-1000 ug/l are found. The fact that higher concentrations are more common in New England, the Southeast, the Missouri River Basin, the Rio Grande River Basin and the Upper Colorado, appears to be correlated with mining activities in these areas. However, in all river basins there are some locations with zinc concentrations of 100-1000 ug/l.

Zinc has a tendency to absorb to sedimentary material. Consequently, anthropogenic discharges of zinc in excess of levels naturally in equilibrium with aquatic sediments result in removal from the water column and enrichment of sediments. Severe zinc contamination thus tends to be confined to the region of the source. Zinc in the water column is primarily in the form of the free ion.

Zinc is generally found in soils at concentrations between 10 mg/kg and 300 mg/kg, with a mean of about 50 mg/kg. Soils near highways and smelters have been found to contain higher concentrations, due to deposition of zinc released in tire abrasion and stack emissions.

The mobility of zinc in soil depends on the solubility of the compound and, to some extent, on the soil properties. Zinc in a soluble form, such as zinc sulfate, is fairly mobile in most soils. However, as relatively little land disposed zinc is in soluble form, the slow rate of dissolution will limit mobility. Consequently, movement toward groundwater is expected to be slow unless zinc is applied to soil in soluble form (such as in agricultural applications) or accompanied by corrosive substances (such as in mine tailings). The transport of soil zinc may also result from surface runoff or entrainment of particles into the atmosphere.

Annual average airborne zinc concentrations in urban areas of the United States are generally less than 1 ug/m^3. Although data are sparse, higher airborne concentrations of zinc would be expected in the vicinity of iron and steel-producing plants and zinc smelters. Atmospheric emissions of zinc, consisting primarily of zinc sorbed to submicron particulate matter and the oxide of zinc, are expected to be short-lived in the atmosphere, with deposition upon soil and pavement occurring as fallout and washout.

EFFECTS OF ZINC

Zinc is an essential trace element in human and animal nutrition; the recommended dietary allowance for humans is 3-15 mg/day in humans. Zinc deficiency in humans has been associated with such effects as growth impairments, inhibition of sexual maturation, loss of appetite, inability to gain weight, skeletal abnormalities, perakeratotic esophageal and skin lesions, and hair loss.

Moderately high levels of zinc appear to have few adverse effects on humans or animals; the metal has not been shown to be either carcinogenic or mutagenic. Human survival has been reported after ingestion of up to 12,000 mg of metallic zinc, and most individuals appear capable of ingesting 150 mg zinc on a daily basis without adverse effect. Vomiting and diarrhea, acting to reduce further assimilation, are

generally the threshold effects. However, it is zinc's disagreeable metallic taste which constrains the drinking water criterion to 5 mg/l, well below any emetic threshold.

Inhalation of zinc oxide at concentrations of 15 mg/m^3 of zinc or above produces fever, malaise, headache, and occasional vomiting, thus necessitating the occupational exposure standards currently in effect.

The effects of zinc on aquatic organisms are of more concern. Several fish kills in recent years have been attributed to zinc from runoff and discharges from mining areas and smelters. However, the concentrations causing mortality were generally not well documented, and in many cases, high levels of other metals were also present.

In the laboratory, avoidance reactions have been observed in rainbow trout at concentrations as low as 5.6 ug/l. Effects on growth, reproduction and survival are reported in various freshwater fish species after chronic exposure to concentrations of 106-1150 ug/l. There are not enough data to permit generalizations concerning invertebrates as a group. The proposed fresh water criterion ranges from approximately 15-80 ug/l depending on hardness.

Acute toxicity studies have been conducted for many species of freshwater fish. LC_{50} values range from 90-103,000 ug/l, with salmonids and striped bass reported as being the most sensitive. Invertebrates are, with some exceptions, sensitive to the same range of concentrations.

The limited information available suggests that marine invertebrates are less susceptible than freshwater species. Marine invertebrates such as oysters and crabs exhibit growth reductions at 50-125 ug/l.

A strong negative correlation between water hardness and zinc toxicity has been confirmed for freshwater organisms. The effects of temperature, pH, and other water quality parameters are not as well understood.

EXPOSURE AND RISK

Humans are primarily exposed to zinc through ingestion; the dietary intake of an average teenage male has been estimated to be 18.6 mg zinc/day. Dietary supplements may provide up to an additional 75 mg zinc per tablet. The mean intake of zinc in drinking water is 0.4 mg/day (maximum of 26 mg/day). Negligible quantities are inhaled in ambient air. Since humans are able to tolerate 150 mg/day without adverse effects, little risk appears to be associated with these exposures.

Exposure of aquatic organisms to 100-1000 ug/l total zinc is common in the United States, especially in New England, the Western Gulf and the Southeast regions. Since calcium hardness appears to mitigate the toxicity of zinc, risk may be greater in New England and parts of the Southeast, which have soft water.

Salmonids and invertebrates are acutely sensitive to zinc concentration in the range of 100-1000 ug/l. Over 20% of the water samples taken nationwide have zinc concentrations exceeding 100 ppb. About 25% of all samples exceed the proposed chronic exposure water quality criterion. However, there is some uncertainty in estimating risk from laboratory toxicity data coupled with ambient monitoring data. Organisms in the environment may be somewhat less susceptible to toxicity than those in the laboratory due to differences in the make-up of the two systems. Compared to laboratory waters, where the toxic free ion of zinc can be expected to predominate, a portion of zinc in environmental waters may be adsorbed to solids or, under certain conditions, complexed with organic or inorganic material. In addition, acclimation may occur in environments receiving chronic exposures.

Consequently, estimation of the actual ecological risk due to zinc requires closer examination of areas having elevated aquatic zinc levels, employing both field and laboratory measures of stress. Also needed is a better understanding of the relationship between toxicity and chemical speciation of zinc.

II. INTRODUCTION

The Office of Water Planning and Standards, Monitoring and Data Support Division of the Environmental Protection Agency is conducting a program to evaluate the exposure to and risk of 129 priority pollutants in the nation's environment. The risks to be evaluated included potential harm to human beings and deleterious effects on fish and other biota. The goal of the task under which this report has been prepared is to integrate information on cultural and environmental flows of specific priority pollutants and estimate the risk based on receptor exposure to these substances. The results are intended to serve as a basis for developing suitable regulatory strategy for reducing the risk, if such action is indicated.

This report is intended to provide a brief, but comprehensive, summary of the production, use, distribution, fate, effects, exposure, and potential risks of zinc. There are a number of problems with attempting such an analysis for zinc. Since the purpose of this report is to provide a basis for regulation, it is important to identify sources. However, zinc is an element commonly found in the earth's crust and natural sources to waterways can be significant. Thus in any analysis of discharges or runoff, it is important to distinguish background concentrations or natural sources from anthropogenic sources. We have attempted to do this to the extent possible, but in discharges from Publicly Owned Treatment Works (POTW) facilities, for example, it is difficult to trace back to the sources, natural or anthropogenic

In addition, the aquatic chemistry of zinc is complex. Other metals are commonly found with zinc, making the situation more complicated due to possible interactions. We have used information available on the aquatic chemistry of zinc to draw conclusions regarding specific fate pathways as related to sources.

Finally, zinc is a nutritional requirement, and zinc deficiency could be considered a risk. However, for the purposes of this risk assessment

we have discussed zinc deficiency cursorily to establish a range of acceptable doses. We have concentrated on assessing the risks due to exposure to high levels of zinc.

This report is organized as follows:

- Section III contains information on the production, discharge (point and non-point) and disposal of zinc.

- Section IV describes available monitoring data and a consideration of the fate of zinc in five specific pathways.

- Section V considers reported effect levels for humans and aquatic organisms.

- Section VI discusses exposure scenarios for humans and aquatic organisms.

- Section VII discusses risk to various subpopulations of humans and aquatic organisms.

III. MATERIALS BALANCE

A. INTRODUCTION AND METHODOLOGY

In this section, data on sources of zinc and pathways of entry into the environment are identified. Current and past EPA reports, readily available literature and personal contacts with EPA and industry experts at Arthur D. Little, Inc., were used to estimate environmental loadings of zinc. The environmental compartments (air, land, water, etc.) initially receiving and transmitting the element were studied, as were the locations at which the environmental loadings take place. There are many uncertainties in this analysis: current releases have not been identified from all sources, past releases are not well documented, and future releases may occur at undefined locations. Nevertheless, sufficient information is available to indicate the nature and scale (temporal and geographical) of environmental discharge of zinc.

B. MATERIALS BALANCE CHECKLIST

Zinc is one of the major common metals (iron, aluminum, copper, lead, zinc). It is moderately abundant in the earth's crust with an average concentration of about 70 ppm. Soil content of zinc varies depending upon geologic composition and land use, ranging between 10 and 330 ppm. Soils tested near highways have shown higher zinc concentrations due to runoff from rubber tire wear, automobile transmission oil, and galvanized highway structures and automobile parts. Industrial areas contain roughly twice as much zinc as rural areas; a test at Grand Rapids, Michigan, showed 56.6 ppm zinc in urban and industrial areas and 22.1 ppm and 21.1 ppm in nearby agricultural and residential areas, respectively (NRC, 1979).

In 1977, total U.S. industrial demand was 1.19 million metric tons. The projected annual growth rate for zinc production is 2.0% through

the year 2000. Domestic reserves are approximately 21.8 million metric tons; therefore, United States production is not expected to meet the domestic demand. Worldwide reserves are estimated to be 154.2 million metric tons, sufficient for projected demand (Versar, 1978). Table III-1 summarizes the commercial sources and uses of zinc.

Environmental releases from production as well as consumption and uses, disposal and reclamation, are presented in Table III-2 and visualized in Figure III-1. Zinc is consumed in galvanizing, metal alloys manufacturing and die-casting, brass products, iron and steel production, vulcanization of rubber, plastic products, paper products, and in various compounds used in paints and cosmetics. The zinc production industry itself, iron and steel industries, and galvanizing operations or loss from galvanized products account for the major environmental releases.

1. Primary and Secondary Zinc Production

Zinc is mined at nearly 30 mines and produced at six smelters in the United States which provide a major source of environmental inputs; a typical flow sheet for the zinc production process is shown in Figure III-2. Table III-3 quantifies zinc production and beneficiation by state with consequent water and land emissions. The major portion of zinc ore is mined by conventional underground mining methods. The five states of Missouri, Tennessee, Idaho, New York and Colorado account for over 85% of all zinc mined. Active zinc mines are shown in Figure III-3. Following extraction, it is crushed in the mills and concentrated by differential flotation. Although losses to the atmosphere are relatively small, some loss does occur during blasting, ore handling, crushing, and through fugitive dust emissions from tailings. Primary production presents a source of waterborne zinc, particularly when water pumped from the mine is utilized in the concentrating process. Other stages in the production process with potential for waterborne zinc emissions include: grinding at the mill; flotation cells in zinc, lead and copper operations; thickening (concentrate thick-

Table III-1

Summary of U.S. Zinc Supply and Demand (1977)

	Supply Metric Tons (MT)	Consumed (MT)
Domestic Production		
Mine Production (net)	415,406	
Secondary Production	77,095	
Imports, Exports, Stocks		
Imports (metal)	523,339	
Imports (ores & concentrates)	111,561	
Imports-Exports (compounds)	13,605	
Industry Stocks (1/1 - 12/31)	49,885	
Consumptive Uses		
Galvanizing		488,266
Zinc Alloys Mfg.		428,720
Brass Products		142,906
Zinc Oxide Production		47,637
Rolled Zinc		35,727
Other Zinc Compounds		47,635
Total	1,190,891	1,190,891

Source: Versar, 1978.

Note: The above figures are for one year. There is considerable statistical variation from year to year; consequently, these do not reflect average values.

Table III-2

Summary of Environmental Releases of Zinc (MT/Year)

	Air	Direct Aquatic	POTW	Land
Mine Production	Δ[1]	73[1]	None[1]	30,660[1]
Smelting	114[1]	1,100[12]	None[1]	22,710[1]
Secondary Productivity	754[3]	Δ[2]	Δ[1]	N/A
Galvanizing Production	903[2]	Δ[2]	Δ[1]	N/A
Zinc Alloys Mfg.	110[1,2]	Δ[2]	Δ[1]	2,090[1,2]
Brass Products	10[1,2]	Δ[2]	Δ[1]	180[1,2]
Iron & Steel Production	814[1,2]	2,588[2]	None[1]	43,067[1,2]
Coal Combustion	1,420[1]	450[12]	Δ[1]	14,240[1]
Coal Mining	Δ[1]	1,500[2]	None[1]	Δ[1]
Copper Mining	Δ[1]	4[1]	None[1]	6,512[1]
Other Metallic Ore Mining	Δ[1]	1[1]	None[1]	None[1]
Paper Products	Δ[2]	1,100[2]	Δ	N/A
Plastic Products	Δ[2]	1,700[2]	N/A	N/A
Electroplating	Δ[1]	414[1,9]	1,620[1,9]	966[1,9]
Inactive Mines	Δ	4,060[13]	None	-
Other Industries***		270[12]	960[12]	
Area Sources:				
Galvanized Mat'l Decay	None	*	*	432,800[1]
Tire Abrasion	N/A	*	*	6,808[4,5]
Agricultural Applications	N/A	*	None	56,000[6,7]
Suspended Sediment	None	45,400[3,8]		
Incineration/Refuse	3,000[1]	None	None	57,000[1]
POTW	None	7,814[10]	-	14,269[10]
Total	7,125	66,474	-	687,302
Urban Runoff Component**	-	5,250	2,906	-

*Portions of these releases enter directly or indirectly through the water compartment through groundwater, surface runoff, etc.; however, due to the uncertain nature of the release, contribution to water cannot be identified quantitatively.

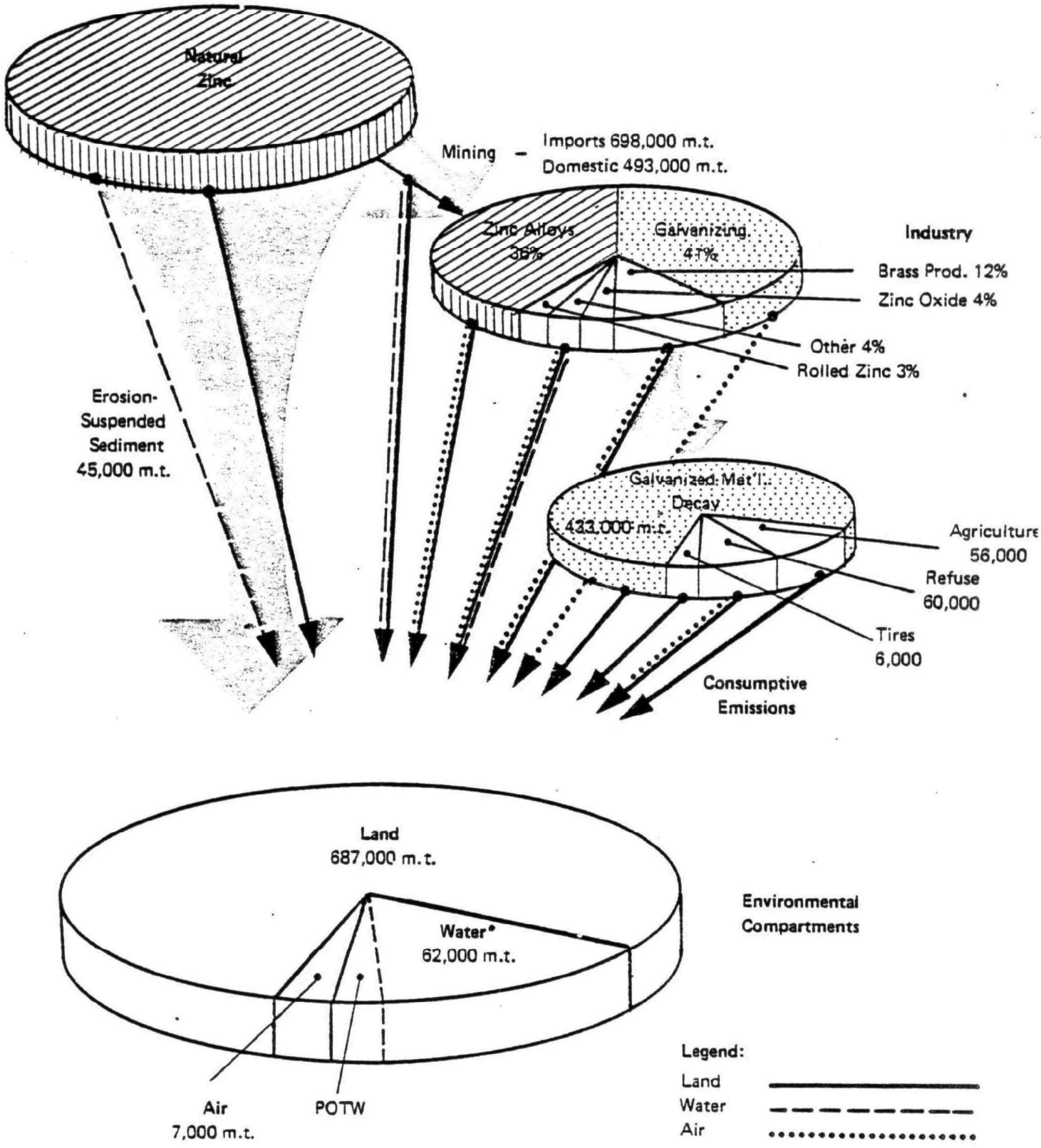
**Galvanized material decay and tire abrasion are among the sources of zinc to urban runoff.

***Includes 624 MT to POTW from carwashes.

Δ Insignificant.

Sources:

[1]Arthur D. Little, Inc., Industry Experts
[2]Versar, 1978
[3]NRC, 1979
[4]Christensen et al., 1979
[5]DOT, 1978
[6]SRI, 1979
[7]Anderson, 1977
[8]Wischmeier, 1976
[9]Bureau of Mines
[10]Table III-9
[11]EPA, September, 1979
[12]Versar, [yet] unpublished communication, 1979
[13]Martin and Mills, 1976



Note: Boundaries between receiving medium are often undefined and/or changing: Zinc apparently released to one compartment can result in another.

* Includes discharge to POTW = 7814 m.t.

Source: Arthur D. Little, Inc.

FIGURE III–1 ENVIRONMENTAL BEHAVIOR OF ZINC

Figure III-2
TYPICAL FLOWSHEET - LEAD/ZINC ORES

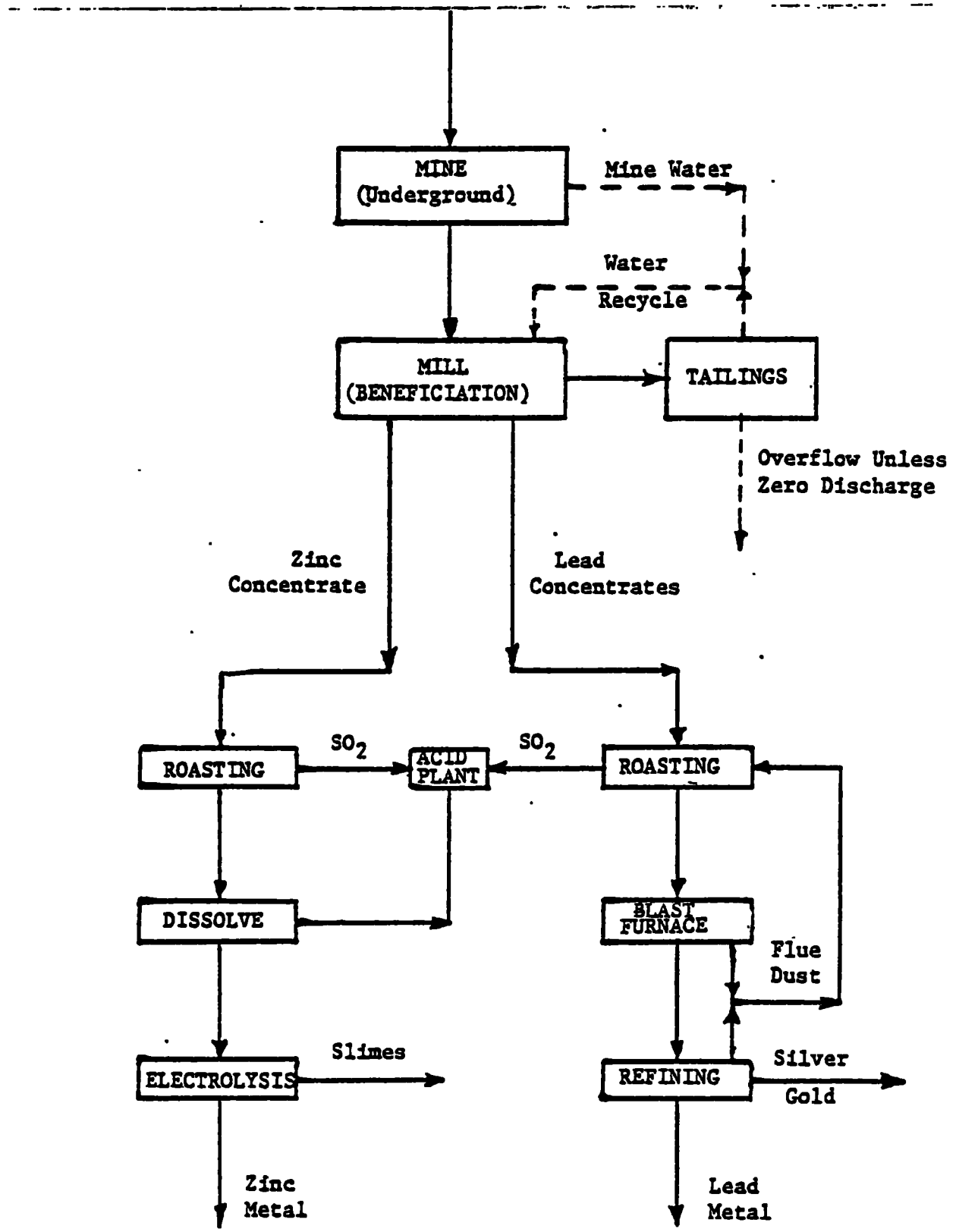


Table III-3

Zinc Releases from Mining and Milling Activities

(Zinc - Lead - Lead/Zinc Mines)

	Ore Mined 1000 MT/Yr	Concentrate Produced 1000 MT/Yr	Zinc Released to Water MT/Yr	Zinc Deposited as Solid Wastes MT/Yr
New York	1,124	131	0.712	5,000
Tennessee	2,260	137	0.537	1,600
Missouri	7,663	611	1.873	10,650
Pennsylvania	348	-	7.877	900
Idaho	1,185	144	54.512	3,050
New Mexico	122	69	0.180	360
Colorado	903	125	0.947	2,920
Wisconsin	334	16	4.006	1,800
Virginia	541	31	1.749	2,500
Utah	196	37	-	600
Arizona	83	5	-	240
Washington	273	16	0.199	1,040
TOTAL	15,032		72.592	30,660

Source: Arthur D. Little, Inc., Estimates and EPA, NPDES data.

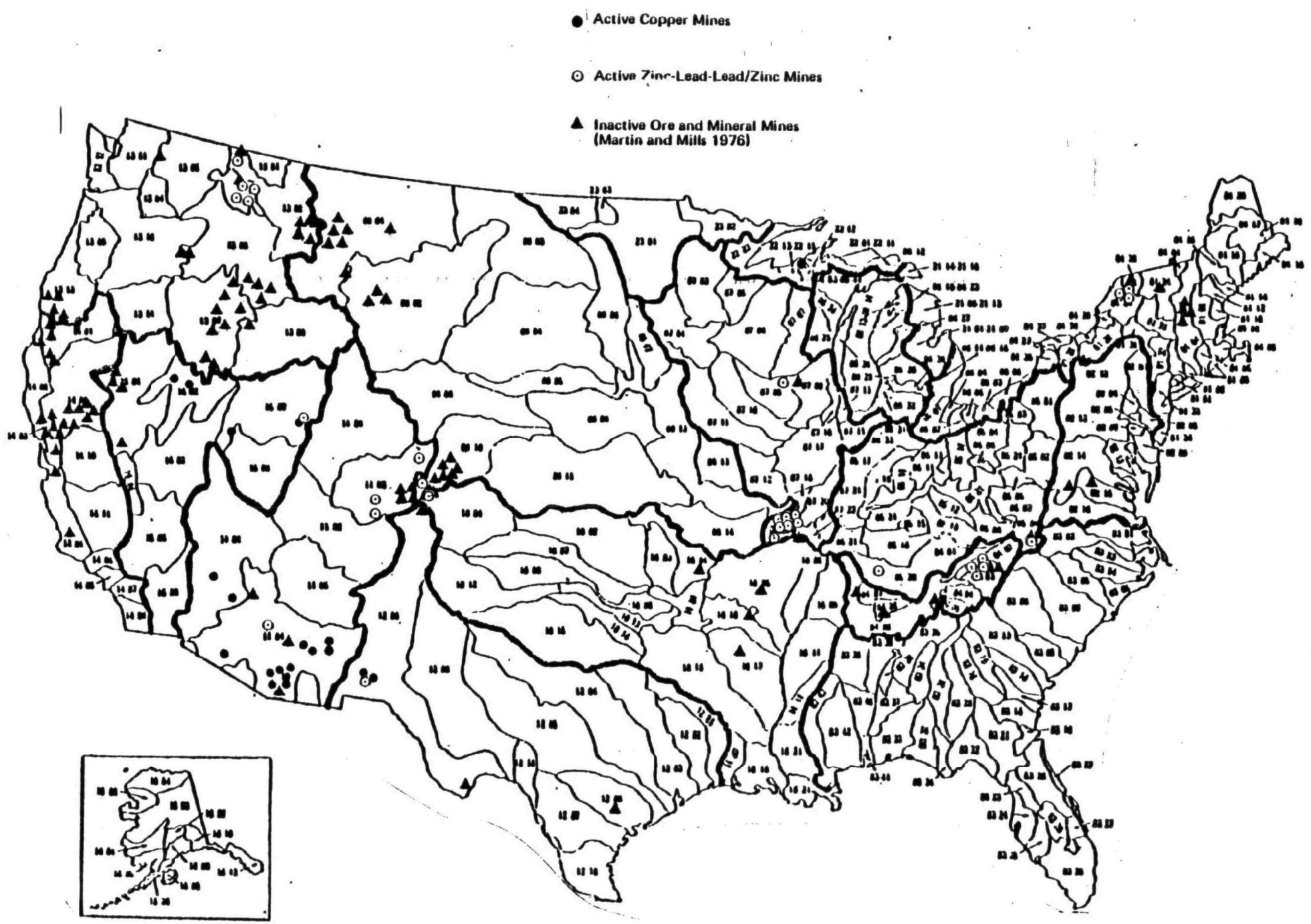


FIGURE III–3 LOCATION OF ACTIVE AND INACTIVE MINES IN THE UNITED STATES

Nc N rs es EP/ ver in igi ons

eners are either discharged or recycled); and settling (treatment lagoons) (NRC, 1979). The effluent limitation guidelines of the EPA for zinc content in water effluents from these operations is 0.50 mg/l (30-day average) effective July 11, 1978 (Federal Register). Prior to regulation, most discharges were in the range of 0.50 mg/l with some higher. Currently all mines and mills operate within that qualification except one mill (in Idaho) which is in the process of upgrading that situation. At the present time, there are no regulations for solid wastes. The zinc content in tailings is that zinc which could not be recovered. It is in the form of zinc sulfides and silicates in locked silica waste particles and is insoluble. All of the mines included in the survey presented in Table III-3 are underground mines and essentially have no solid mining waste piles.

Drainage from mine tailings ponds presents an additional source of zinc to the environment. At active mine sites, the contribution is minimal and is accounted for in direct aquatic discharges. Associated tailing ponds are controlled by containment structures such that ordinary runoff is routed through treatment facilities. Therefore, zinc discharge to streams emanating from active mine tailing piles is included in the determination of total mining discharge to water, presented in Table III-2. Significant storm events in which flooding occurs can provide a rare additional aquatic discharge from this source.

Mining operations studied in the above analysis were active; however, runoff from abandoned or inactive mines represents an additional source. A study conducted in 1976 compiled local analyses of stream quality data upstream and downstream of abandoned/inactive mine sites. Daily loads of various constituents, including zinc, were determined. Load factors were extrapolated for each mine and combined with associated waste areas to obtain an annual loading of 4060 metric tons of zinc (Martin and Mills, 1976). Emphasis should be placed on the rough nature of this approximation. This release is independent of active mine operations and therefore is included in Table III-2.

The contribution of zinc smelting operations to the environment occurs in the roasting, sintering and electrolytic process and through vertical retorts. Concentrated zinc ore goes through a roasting procedure to drive off sulfur dioxide, SO_2, and convert the zinc sulfide, ZnS, to zinc oxide, ZnO. This process releases over 50% of the total atmospheric emission during primary production (EPA, 1972). In sintering, lead and cadmium impurities are volatilized and discharged into the stream from which they are extracted. The sintering process also prepares the material for the reduction system. Atmospheric emissions are controlled, usually by bag-houses; therefore, the major portion of environmental release from the smelting operations is to land disposal areas.

Secondary or recycled zinc is an important source of zinc in the United States today. Zinc scrap materials are composed of residual scrap materials and metallic scrap. It can be recovered by remelting, as in the case of brass, or by a sweating and distillation process for other scraps. Releases of zinc to the atmosphere from these processes average 9 kg/ton/produced (NRC, 1979). Total emission from this source is estimated to be 754 metric tons for 1972. As secondary production becomes a more important source of zinc in the future, total zinc emissions will increase.

2. Production in which Zinc is a Byproduct/Contaminant

Zinc is a byproduct/co-product/contaminant in the mining and production of several other metals and in the manufacture of various chemical compounds and products. Lead is frequently mined in conjunction with zinc. Lead and lead related operations are included in the information presented in Table III-3. Of the 38 major copper mines in the United States producing 250 million metric tons of ore, and subsequently 1.50 million metric tons of copper, few have any appreciable zinc content in effluents or solid wastes. Copper mines indicating detectable concentrations of zinc are presented in Table III-4; remaining copper mines either have no discharge or contain only trace

Table III-4

Zinc Released by Copper Mining Operations*

	Effluents		Solid Waste in Tailings	
	Daily Discharge m^3/day	Zn Discharge Per Year MT	Accumulated Total Amount of Zinc MT	Zinc Added per year MT
Underground Mine, Tennessee	32,700	0.48	32,000	4,000
Open Pit Mine, Montana	35,960	0.66	72,480	3,020
Underground Mine, Michigan	121,120	1.77	Trace Only	
Open Pit Mine, Utah	84,400	1.54	7,500	157
TOTAL		4.45	111,980	7,177

*Mining operations presented are those with detectable zinc concentrations; others operate without discharge or contain zinc only as a trace element.

Source: Arthur D. Little, Inc., Estimates and EPA, NPDES data.

amounts of zinc (Arthur D. Little, Inc., Industry Experts, 1979). Several additional metallic ores have effluents containing small amounts of zinc; those that are detectable are shown in Table III-5. Solid wastes at these sites contain zinc, but again, only as a trace element.

Zinc appears in the ash of most coals only in traces. However, the approximately 400 million metric tons of coal burned each year yields about 40 million metric tons of coal ash per year containing a total of 14,240 metric tons of zinc. Table III-6 shows regional variation of zinc content in coal ash. This solid waste is stored in ponds and piles, and some is recovered. Atmospheric emission from coal combustion is variable and, to date, poorly documented. Based upon conservative estimates that approximately 1.0% of the coal ash escapes to the environment, roughly 1420 metric tons of zinc would be emitted annually.

Zinc oxide is widely used in U.S. industry. Its most important application is in production of rubber. Zinc oxide can be produced chemically or by direct or indirect pyrometallurgic methods. Zinc emissions to the atmosphere from this production was estimated as high as 7300 metric tons in 1969 (NRC, 1979).

3. Environmental Release of Zinc During Consumptive Use

Of the 1.19 million metric tons of zinc consumed in 1977, 84% was used directly in metallic form (Versar, 1978). Emissions from consumptive uses of zinc are presented in Table III-2. Manufacturing uses of zinc include galvanizing, brass products, die castings and rolled zinc. The construction industry consumes the largest quantity of zinc in the following applications: protective coating for structural steel, roofing, siding, guttering, and reinforcing bars. Galvanized sheet is the standard duct material for air conditioning, ventilating, and heating systems. In architectural construction, brass and zinc-bearing bronze are frequently used for door and window frames, railings,

Table III-5

Zinc Releases by Other Metallic Ore Mining Operations

	EFFLUENTS	
	Daily Discharge m^3/day	Zinc Discharge Per Year MT
South Dakota: Gold Mine	625	0.01
New Mexico: Molybdenum Mine	11,000	0.08
Idaho: Silver Mine, A	682	0.01
Idaho: Silver Mine, B	3,133	0.03
Colorado: Molybdenum Mine	11,000	0.12
California: Tungsten Mine	33,000	0.24
Arkansas: Bauxite A	41,640	0.30
Arkansas: Bauxite B	7,190	0.05
Arkansas: Vanadium	2,682	0.02
New York: Titanium	21,000	0.23
TOTAL		1.09

Source: Arthur D. Little, Inc., Estimates and EPA, NPDES Data.

Table III-6

Zinc Content in Coal Ash by Region

	Zinc Content per Volume Ash (%)	Zinc in Coal Ash MT/Yr
Eastern Region (Appalachia, etc.)	0.0230	3,340
Interior Province (Illinois, Kansas, Ohio, etc.)	0.0743	8,090
Western Region (Wyoming, Montana, Utah, etc.)	0.0258	2,810
TOTAL		14,240

Source: Arthur D. Little, Inc., Estimates and EPA, NPDES data.

panels, spandrels, and building hardware. Brass fittings are used in plumbing and heating systems as faucets, valves, traps, pump casings, and brass condenser and heat exchanger tubes (Versar, 1978). Although the nature and quantities of these industrial applications are fairly well defined, the discharges related to use and ultimate disposal cannot be predicted accurately.

The protective nature of zinc coatings to many metals indicates its frequent environmental release; its function is to be sacrificially corroded. Assuming a 15- to 20-year average life span for galvanized products, up to 430,000 metric tons may be released annually. If galvanized products are distributed in proportion to population distribution, regional release would be as shown in Table III-7.

Although the quantity of zinc consumed by the electroplating industry is relatively small by comparison to other metals, e.g., nickel, the process releases some zinc to the aquatic environment. Based on estimated consumption by the Bureau of Mines (BOM, 1979; and American Iron and Steel Institute, 1978) approximately 25,000 metric tons of zinc are consumed annually for electrolytic galvanizing of sheet and strip metals; an additional 5,000 metric tons of zinc are consumed annually for miscellaneous electroplating applications. Because of zinc's relatively minor role in the industry, these estimates might vary slightly ($\pm$1,000 to 5,000 metric tons). Conservative estimates by both BOM (1979) and Arthur D. Little, Inc., conclude that no more than 10% of that consumed is released to the environment (i.e., 3,000 metric tons released). EPA reports that 54% of the electroplating operations discharge to POTW's; of those remaining, approximately 70% of the zinc is captured by treatment systems and disposed on land as sludge and the remainder is discharged directly to water.

An alternate estimate of zinc discharge due to electroplating suggested that approximately 38,900 metric tons are released (Versar, 1979). Considering the approximate consumption of zinc by the in-

Table III-7

Other Zinc Releases to Environment by Region

	Tire Abrasion MT/Yr	Galvanized Decay MT/Yr
New England (ME, NH, VT, RI, CT, MA)	361	25,500
Middle Atlantic (NY, NJ, PA)	780	82,600
East North Central (OH, IN, IL, MI, WI)	1,295	87,300
West North Central (MN, IA, MO, ND, SD, NE, KS)	580	37,200
South Atlantic (DE, MD, DC, WV, VA, NC, SC, GA, FL)	1,217	62,700
East South Central (KY, TN, AL, MS)	510	29,000
West South Central (AR, LA, OK, TX)	770	41,100
Mountain (MT, ID, WY, CO, NM, AZ, UT, NV)	345	16,400
Pacific (WA, OR, AK, HI, CA)	950	51,000
TOTAL	6,808	432,800 *

Source: Arthur D. Little, Inc., Estimates; DOT, 1978; Christensen, 1979; and Bureau of Mines, 1978.

*Averaged over five year period, Bureau of Mines.

dustry (30,000 ±5,000 metric tons), perhaps the sampling used to determine concentrations was unrepresentative.

The transportation industry is a consumer of galvanized sheet steel, die-cast alloys, and brass. The majority of die castings are used for automobile components. Brass is used for radiators, tubing, and decorative trim. Galvanizing is effective in protecting steel products such as railroad equipment, ship hulls, aircraft, buses, trucks, trailers, and large-scale industrial equipment. Zinc-rich paint, which is developing a fast growing market, is used to supplement galvanized steel for automotive underbody protection. Zinc is a minor constituent of automobile fuels and lubricating oils.

A remaining 13% of zinc consumed in metal is distributed in a variety of miscellaneous applications: battery cases, weather-stripping, litographic plates, sacrificial anodes for ship hulls, offshore drilling rigs and production (Versar, 1978).

Zinc and its components are being used increasingly in the chemical-metallurgic, ceramic, fertilizer, paint, paper, plastics, rubber, textile and electronic industries. As a binding material, zinc oxide aids in the vulcanization process in tire making; by weight, tires are estimated to contain 0.73% zinc (Christensen et al., 1979). Abrasion on road surfaces during normal tire wear contributes 6800 metric tons zinc to surrounding soil and water systems as shown in Table III-7, column 1 (Christensen, 1979; DOT, 1978). Zinc concentration in soils near highways has been reported as a function of distance from the road and depth below the ground surface at four locations (NRC, 1979). Concentrations eight meters from the road exceeded 140 ppm above the background level. Considerable variability was indicated between different locations, but the data were analyzed to estimate an average zinc accumulation of 13 pounds per mile. The probable range of values is estimated at between 6.5 and 20.3 pounds per mile. Using the aver-

age value and paved road mileage in the United States,[1] it is estimated that some 11,537 MT of zinc has accumulated within 60 meters of U.S. highways.

Table III-8 shows the geographic regional distribution of the zinc accumulation along roads, and compares it with the annual amount of zinc released through tire abrasion. It can be seen that the amount accumulated generally represents less than two years worth of loss from tire abrasion. This would indicate that a large percentage of abrasion loss finds its way directly to water.

4. Other Sources

Urban runoff transports a significant portion of environmentally emitted zinc. Numerous sources are responsible for this occurrence; however, due to the variable conditions surrounding zinc consumed in the urban environment, it is difficult to determine quantitative estimates of specific sources to urban runoff. As shown in Table III-2, urban runoff is not an independent emission of zinc, rather is comprised of the other sources listed in the table; to prevent double counting, urban runoff is presented separately from the summation of zinc releases.

Zinc contained in urban runoff was estimated using EPA information gathered nationwide on combined sewer overflows and stormwater discharges (EPA, 1977). Zinc concentrations were obtained from a variety of field studies (Shaheen, 1975; Pitt, 1978; Illinois EPA, 1978; Mattraw; Colston, 1974) and results ranged from 87 µg/litre to 750 µg/litre. Based on a zinc concentration of 250 µg/litre, 5250 metric tons of zinc is discharged in urban runoff each year.

[1] Road mileage is based on bituminous (or higher grade) surface roads in the United States.

Table III-8

Regional Distribution of Zinc Accumulation Near Paved Roads

	Accumulation[1] MT	Tire Abrasion MT/Yr	Equivalent Time Years
New England (ME, NH, VT, RI, CT, MA)	504	361	1.4
Middle Atlantic (NY, NJ, PA)	1,163	780	1.5
East North Central (OH, IN, IL, MI, WI)	2,104	1,295	1.6
West North Central (MN, IA, MO, ND, SD, NE, KS)	1,225	580	2.1
South Atlantic (DE, MD, DC, WV, VA, NC, SC, GA, FL)	1,965	1,217	1.6
East South Central (KY, TN, AL, MS)	1,076	510	2.1
West South Central (AR, LA, OK, TX)	1,366	770	1.8
Mountain (MT, ID, WY, CO, NM, AZ, UT, NV)	1,042	345	3.0
Pacific (WA, OR, AK, HI, CA)	1,092	950	1.1
TOTAL	11,537	6,808	1.7

[1] NRC (1979) presents data from which average roadside accumulation has been estimated as $13^{+7.3}_{-6.5}$ pounds of zinc per mile of highway. Regional mileage figures are based on bituminous, or higher grade, surfaced roads.

Galvanized material decay represents the most significant potential source to urban runoff; if a correlation between population distribution and galvanized material consumption were assumed, approximately 70% of all galvanized material (roughly 300,000 MT annually) would be located in the nation's cities and susceptible to decay. The viability of this assumption is questionable, however, particularly when considering zinc utilized in highway guardrails and other construction items used frequently in rural areas. Automobiles are an additional potential source of zinc in urban runoff. Here again, however, the urban component is ambiguous: tire abrasion occurs in non-urban areas due to high speed travel, yet fast stops, tire skidding and dripping of transmission oil potentially contribute to urban areas. It is difficult, therefore, to determine what portion of the 6,808 MT of zinc released by tire abrasion is in urban runoff although undoubtedly it contributes. These are the most apparent zinc bearing constituents, however, others might include decay of materials in which zinc is a constituent (other non-ferrous metals) and atmospheric fallout from fuel oil and coal combustion, incineration, soil erosion, and other industrial and construction activities.

As mentioned earlier, zinc is a natural constituent of soil and plant life. Frequently, crop plants are seriously deficient in zinc content. Therefore, in agriculture, zinc compounds are used as plant and animal nutrients as well as fungicides and wood preservatives. The most common compound used is zinc sulfate, mostly as a micronutrient. In 1977, agricultural fertilizers discharged approximately 33,000 metric tons of zinc (SRI, 1979). Additional agricultural uses released 26,000 agricultural discharges based upon distribution of crop lands (Anderson, 1978).

Suspended sediment load in streams and waterbodies is a regularly occurring phenomenon which transports soil elements through the water compartment. The average total suspended load on the Mississippi River measured near its mouth is 258 million metric tons annually

(Todd, 1970). Based on the assumption that the Mississippi basin is roughly 40% of the total U.S. area, and that natural soil content of zinc varies between 10 and 300 ppm with a mean of 50 ppm (NRC, 1979), suspended sediment naturally contributes about 32,200 metric tons of zinc per year to water. Wischmeier reports an average of 3.6 billion metric tons of sediment transported by water annually, with about 25% of that reaching major streams (Wischmeier, 1976). Based on Wischmeier's determination and the above zinc concentration, suspended sediment contributes 45,400 metric tons to water each year. The latter, more conservative estimate is presented in Table III-2.

5. Municipal Disposal

The total amount of zinc treated in POTW is estimated to be about 22,000 metric tons. To make this estimate we examined the available data and used a flow-weighted mean concentration of zinc in POTW influent and flow-weighted mean removal efficiencies of primary and secondary treatment plants.

A substantial number of studies addressing the composition of POTW influent and effluent have been accomplished in recent years. Many of the studies are of single POTW, and there is considerable variability in the nature of the study, the quality of the reporting, and the indicated range of values for zinc concentration. Several studies present data and conclusions based on groups of POTW which were investigated. Of the studies examined, none present data from a truly representative cross section of POTW in the United States. However, one (Sverdrup and Parcel, 1977) presents a relatively consistent data set on 103 POTW clustered mainly in the Midwest, with some additional plants in California, New Jersey, New York, and elsewhere in the Southeast. The authors of the Sverdrup and Parcel study concluded that their data describe "typical" POTW with regard to heavy metals. The study emphasized collection of data on secondary treatment plants, and consequently the report has only a small number of primary plants in the sample. Using data presented in the study, a flow-weighted mean

for zinc concentration in the influent of the 103 POTW was calculated by the following formula:

$$\bar{C} = \frac{\Sigma^{103} C_i V_i}{\Sigma V_i} = 610 \text{ µg/l}$$

C_i = concentration of i^{th} POTW
V_i = flow volume of i^{th} POTW

The authors of the Sverdrup and Parcel report concluded that POTW meeting secondary treatment standards removed an average of 72% (range 45% to 96%) of the zinc in the influent. This conclusion is drawn from data on 22 of the 103 plants which both operated to these standards and had sufficient data on all parameters of interest to allow analysis. Sverdrup and Parcel noted that while influent concentrations reported elsewhere in the literature agreed with their data, the removal efficiencies reported elsewhere tended to be lower. They suggested that the explanation could be that other analyses included some POTW not meeting secondary standards. In any event, 81% was the flow-weighted mean of the removal efficiencies for the 22 plants. Data were presented on removal efficiency for 10 primary treatment facilities in addition to the 22 secondary plants. The median value of removal efficiency for the primary plants was 39% while the flow-weighted mean was 17%. The latter was used in our calculations to estimate partitioning between sludge and release to the aquatic environment.

We have little data on improved metals removal during advanced treatment. However, we assumed that metal removal efficiencies would be related to solids removal, and used data in the 1978 Needs Survey to characterize metals removal efficiency in Advanced Secondary and Tertiary Treatment plants (EPA, 1978 Needs Survey). Based on the Needs Survey, 28% of the total flow from POTW undergoes primary treatment,

39% secondary, 18% advanced secondary, and 14% tertiary treatment. Advanced secondary is assumed to remove 88% of zinc, while tertiary treatment is assumed to remove 86%.

Table III-9 summarizes the POTW zinc budget based on the above assumptions and shows a total loading to POTW of 22,083 metric tons, of which 7814 metric tons of zinc is discharged by POTW to the aquatic environment, while 14,269 metric tons is discharged to land. An earlier estimate (Versar, 1978) indicated a total loading of 21,300 metric tons which agrees well with that estimated in this study.

POTW discharge represents a major source of zinc to the water environment. The sources of zinc in POTW influent are not completely defined. However, assuming that the zinc concentration in tap water is characterized by the reported 2595 sample average of 194 µg/l (U.S. HEW, 1970), 7023 metric tons/year could be contributed from this source. Human waste is also a contributor to this source. Zinc is found in every human tissue and tissue fluid; total body zinc for an average man weighing 70 kg is estimated to be 2.3 g. It is, therefore, the most prevalent trace metal in tissue (NRC, 1979). Zinc content in human excrement varies from 7 to 12 mg daily (NRC, 1979). Using an average of 9.5 mg/capita/day and 164 million persons served by POTW (EPA, 1978 Needs Survey), a 568 metric ton loading of zinc would be contributed from excrement alone.

An average residential loading of 128.5 mg/capita/day was reported recently on the basis of an extensive sampling survey of four cities (Arthur D. Little, Inc., 1979). If this loading is representative of POTW users throughout the country, then some 7692 metric tons of zinc would be contributed. However, the average zinc concentration in the tap water of these four cities was reported to be 67 µg/l (as opposed to 194 µg/l from the HEW sample). At this average concentration the tap water would contribute 2422 metric tons. Excluding the contributions due to tap water and body wastes yields a net residential loading estimate of 4702 metric tons.

Table III-9

Summary of POTW Zinc Budget

	Treated Flow (MGD)[1]	Zinc Loading to POTW (MT)[1][2]	Treatment Removal Efficiency	POTW Discharge (MT)	
				To Sludge	To Water
Primary treatment	7,525	6,341	.17[3]	1,078	5,263
Secondary	10,137	8,543	.81[3]	6,920	1,623
Advanced secondary	4,731	3,987	.88[4]	3,509	478
Tertiary	3,812	3,212	.86[4]	2,762	450
Total	26,205	22,083	.65 (overall)	14,269	7,814

[1] EPA 1978 Needs Survey, FRD-2.

[2] L(MT/yr) = flow (MGD) x 610 (10^{-6} g/l) x 3.785 (l/gal) x 365 (day/yr) x 10^{-6} ($\frac{MT}{g}$) = 0.8427 x flow.

[3] Flow-weighted mean value calculated from Sverdrup and Parcel Associates data, February 1977.

[4] Assume advanced treatment removes Zn proportionately to TSS--estimated from tables 17, 27, 31 of EPA 1978 Needs Survey, FRD-2.

Urban runoff is another constituent of zinc in POTW discharge. Although it is difficult to estimate the percentages of zinc from tire abrasion, galvanized material decay and other urban sources, POTW effluent obtains a substantial portion of its zinc from these releases. Table III-10 summarizes the major sources of zinc to POTW influent. The 5264 kkg of zinc unaccounted for may have industrial and commercial sources, or may represent an underestimation of the contribution from urban runoff.

Reliable data on concentration of trace metals in incineration and refuse operations throughout the United States are sparse. Based upon an assumption that of approximately 200 million metric tons of municipal refuse disposed of in the United States each year, zinc concentration is 0.03% of the total source. As much as 5% or 3000 metric tons is emitted to air via incineration; solids leaving the incinerator or bypassing it altogether release the remaining 95% or 57,000 metric tons to land (NRC, 1979 and SRI, 1979).

C. SUMMARY

The release of zinc to the environment occurs in all environmental compartments as indicated in Table III-2. The land compartment is the largest receptor of zinc released to the environment. Primary production of zinc, iron and steel production, coal combustion, and refuse disposal are the major sources of zinc as solid waste. Highway surface runoff, galvanized metal decay and agricultural application provide the major distributive source of zinc to land.

The water compartment receives zinc concentrated discharges from POTW and directly from industries. Suspended sediment and galvanized material decay, tire abrasion, and agricultural applications provide a major non-point source contribution of zinc to the aquatic environment.

TABLE III-10

Sources of Zinc to POTW (kkg/year)

Tap Water	7,023
Car Wash	624
Human Body Wastes	568
Residential Loading*	4,702
Urban Runoff**	2,906
Electroplating	<1,620
Unknown***	4,640
Total	22,083

*Excluding contributions from body waste and tap water.

**Includes maximum storm water discharge potentially contributing to POTW's, however this entire quantity does not necessarily reach POTW.

***This estimate is determined to be the difference between known contributors to POTW and total POTW loading.

D. REFERENCES

American Iron and Steel Institute. 1978. Annual Statistics, Washington, D.C.

Anderson, J.R. 1977. Land use and land cover changes - a framework for monitoring. J. Research U.S. Geologic Survey 143-152.

Arthur D. Little, Inc. 1979. Sources of toxic pollutants found in influents to sewage treatment plants. VI. Overall interpretation. Report on EPA Contract No. 68-01-3857.

Arthur D. Little, Inc. 1972. Economic Impact of Anticipated Pollution Abatement Costs - Primary Zinc Industry, Parts 1, 2.

Bureau of Mines. 1977. Minerals Year Book 1977: Zinc. Washington, D.C.

Cammarota, V.A., Jr. 1979. Bureau of Mines, Metals Section, Personal Communication.

Christensen, E.R. et al. 1979. Zinc from automobile tires in urban runoff. ASCE, J. of Environ. Engineering Div. 165-170.

Council for Agricultural Science and Technology. 1976. Application of Sewage Sludge to Cropland-Appraisal of Potential Hazards of Heavy Metals to Plants and Animals. Ames, Iowa (EPA #PB-264-015).

Hydroscience. 1978. Nonpoint Sources: An Assessment of Pollutant Loadings to Lakes and Rivers in North Central Texas. Arlington, Texas.

Martin, H.W. and W.R. Mills. 1976. Water Pollution Caused by Inactive Ore and Mineral Mines. Cincinnati, Ohio (EPA #600/2-76-298).

National Research Council. 1979. Zinc. Baltimore: University Park Press.

Sittig, M. 1975. Environmental Sources and Emissions Handbook. Park Ridge, N.J.: Noyes Data Corporation.

SRI International, Casey, S.E. 1979. Agricultural Sources of Zinc.

Svedrop and Parcel and Associates, Inc. 1977. Study of Selected Pollutant Parameters in Publicly Owned Treatment Works. (Draft.) Task Order No. 7 under EPA Contract 68-01-3289.

Todd, D.K. 1970. The Water Encyclopedia. Port Washington, New York: Water Information Center.

Tracor-Jitco. Production and Use of Zinc.

Trinity River Authority of Texas. 1978. Planning and Environmental Division. Watershed Runoff Water Quality and Sediment Analysis in North Central Texas.

United States Environmental Protection Agency. 1972. AP-42 Compilation of Air Pollutant Emission Factors. Washington, D.C.

United States Environmental Protection Agency. 1976. Considerations Relating to Toxic Substances in the Application of Municipal Sludge to Cropland and Pastureland. Washington, D.C.

United States Environmental Protection Agency. 1977. Heavy Metal Pollution From Spillage at Ore Smelters and Mills. Washington, D.C.

United States Environmental Protection Agency. 1977. Nationwide Evaluation of Combined Sewer Overflows and Urban Stormwater Discharges. Volumes I, III. Washington, D.C.

United States Environmental Protection Agency. 1977. State and Local Pretreatment Programs (Federal Guidelines). Washington, D.C.

United States Environmental Protection Agency. 1979. Needs Survey - Convergence and Treatment of Municipal Wastewater. Summaries of Technical Data. (FR D-2). Washington, D.C.

United States Department of Transportation. 1978. Highway Statistics 1977. Washington, D.C.

Versar. 1978. Gross Annual Discharge to Waters/Zinc.

Versar. 1978. Materials Balance of Zinc.

Versar. 1979. Estimates on Electroplating.

Wischmeier, W.H. 1976. Cropland Erosion and Sedimentation." Agricultural Research Service, USDA. Control of Water Pollution from Cropland. Vol. II. Washington, D.C.

IV. DISTRIBUTION OF ZINC IN THE ENVIRONMENT

A. MONITORING DATA

1. Zinc in Aquatic Environments

a. Zinc in Water

Zinc content in seawater is in the range of 1 to 27 ug/l zinc with a median at about 8 ug/l, while zinc content in uncontaminated freshwater is usually somewhat higher, but less than 50 ug/l.

Of the 19 river basins covered by STORET* surveys, several have high ambient concentrations of zinc in their waters and sediments.

Tables IV-1 and IV-2 provide a complete list of the watersheds surveyed. Figure IV-1 is a compilation of the data for the entire U.S.; the mode in the graph indicates that the majority of the water samples had zinc concentrations in the range of 10-100 ug/l. The corresponding mode for sediment samples is for 1-10 ppm.. Of the 19 major river basins, New England (or the Northeast) had the highest ambient aqueous zinc concentrations. A breakdown of the data for the Northeast into minor river basins have mean levels of less than 50 ug/l, several have mean concentrations exceeding 200 ug/l (the Piscatagua River and estuary, the Presumpscot River, and Lake Champlain). Figure IV-2 shows minor river basins having high levels of zinc in 1978 (STORET). It is apparent that in many areas the mean concentration is greater than 60 ug/l and observations greater than 120 ug/l are common. There appears to be some relationship between high concentrations and mining areas (also shown in Figure IV-2). In addition, high concentrations appear around urban/industrial areas.

In a comparison of natural vs. anthropogenic levels of zinc, surface water samples were taken in 15 of the major EPA water basins. The mean total zinc in benchmark stations ranges from 12.8 ug/l to 246 ug/l. Benchmark stations are located in areas presumably unaffected by anthropogenic sources. Zinc content in other (non-benchmark) stations ranges from 39.1 ppb to 296 ug/l. The factor difference (mean benchmark/mean non-benchmark) within basins ranged from 0.16 to 12.1.

*The water quality data base developed by EPA.

Table IV-1

Total Zinc in Ambient Waters

Region (Number of Observations)	Percentage of Positive Observations			
	1-10 ug/l	10-100 ug/l	100-1,000 ug/l	>1,000 ug/l
New England (7,846)	6	40	45	8
Mid Atlantic (16,005)	20	57	18	5
Southeast (11,776)	16	49	30	3
Great Lakes (7,641)	17	64	15	2
Ohio (10,551)	14	66	19	1
Tennessee (5,503)	17	62	18	3
Upper Mississippi (7,459)	11	64	13	11
Lower Mississippi (6,360)	18	73	7	<1
Souris & Red of North (1,265)	13	79	7	<1
Missouri (6,248)	16	57	24	2
Arkansas & Red (8,697)	23	57	17	1
Western Gulf (623)	12	54	31	<1
Rio Grande & Pecos (646)	10	50	28	7
Upper Colorado (2,163)	18	53	26	3
Lower Colorado (944)	21	63	13	2
Great Basin (769)	35	55	10	0
Pacific Northwest (3,395)	27	51	14	7
California (3,986)	21	55	21	3
Alaska (322)	35	55	9	1
Hawaii (537)	41	55	4	<1
United States (102,736)	16	55	22	4

Source: STORET

Table IV-2

Zinc in Sediment in U.S. River Basins

Region (Number of Observations)	Percentage of Positive Observations			
	1-10 ppm	10-100 ppm	100-1,000 ppm	1,000-10,000 ppm
New England (457)	2	61	33	3
Mid Atlantic (842)	7	55	36	2
Southeast (1,254)	20	70	9	<1
Great Lakes (1,062)	3	42	47	6
Ohio (372)	1	87	12	1
Tennessee (95)	0	65	31	4
Upper Mississippi (185)	5	75	16	3
Lower Mississippi (625)	9	86	4	<1
Souris & Red of North (11)	18	73	9	0
Missouri (114)	10	75	16	0
Arkansas & Red (189	10	66	16	8
Western Gulf (864)	6	80	12	1
Rio Grande & Pecos (66)	8	86	6	0
Upper Colorado (76)	16	59	24	1
Lower Colorado (29)	24	76	0	0
Great Basin (0)	*	*	*	*
Pacific Northwest (54)	2	61	33	4
California (183)	5	79	16	0
Alaska (0)	*	*	*	*
Hawaii (130)	<1	25	75	0
United States (6,608)	8	65	24	2

*No data

Source: STORET

FIGURE IV-1

DISTRIBUTION OF TOTAL ZINC IN AMBIENT WATERS IN THE U.S.

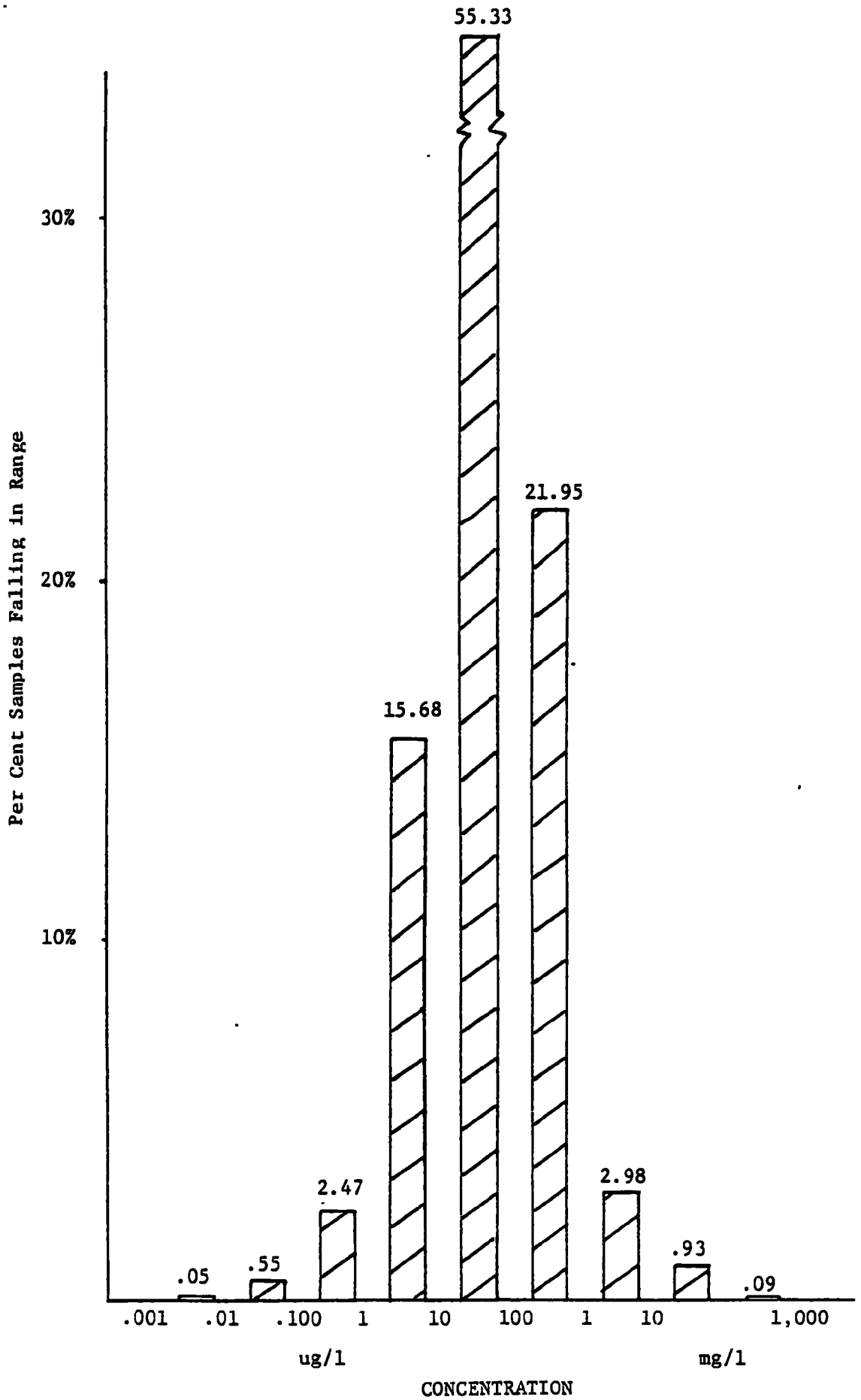


Table IV-3

Northeast, Major Basin 1, Total Zinc in Water for 1978*

Minor Basins (see reference map)		Number of Measurements	Total Zn (ug/l)		
			Mean	Max.	Min.
1	Quinnipiac River & Western Conn Coastal (1974)	664	6.3	570.0	0.01
2	Housatonic River	112	85.8	1110.0	0.02
3	Pawcatuck River & Eastern Conn Coastal (1977)	60	2.4	50.0	0.0
4	Connecticut River	263	71.1	1140.0	0.04
5	Thames River	128	50.0	600.0	0.13
6	Narragansett Bay	29	88.3	360	20.0
9	Merrimack River	121	45.6	510	0.0
10	Piscataqua River & New Hampshire Coastal (1977)	137	353.4	5000	10.0
14	Presumpscot River and Casco Bay	24	1345.9	10100	2.0
15	Adroscoggin River	30	19.4	110	2.0
16	Kennebec and Sheepscot Rivers	17	11.9	49	0.0
24	Lake Champlain	10	202.0	450	10.0
25	St. Lawrence River	10	19.0	30	10.0
27	Niagara River (1975)	61	82.9	1000	0.0
29	Oswego River	31	28.1	130	10.0
30	Mohawk River	59	25.1	240	0.0
32	Middle Hudson River	17	27.6	50	0.0
33	Lower Hudson-New York Metro.	288	62.3	862	0.0
34	New Jersey Coast	234	56.2	586	10.0
35	Lake Erie Shore & Minor Tribs. (1976)	88	62.6	405	0.0

*Minor basins with less than 10 observations have been excluded, remarked data was excluded.

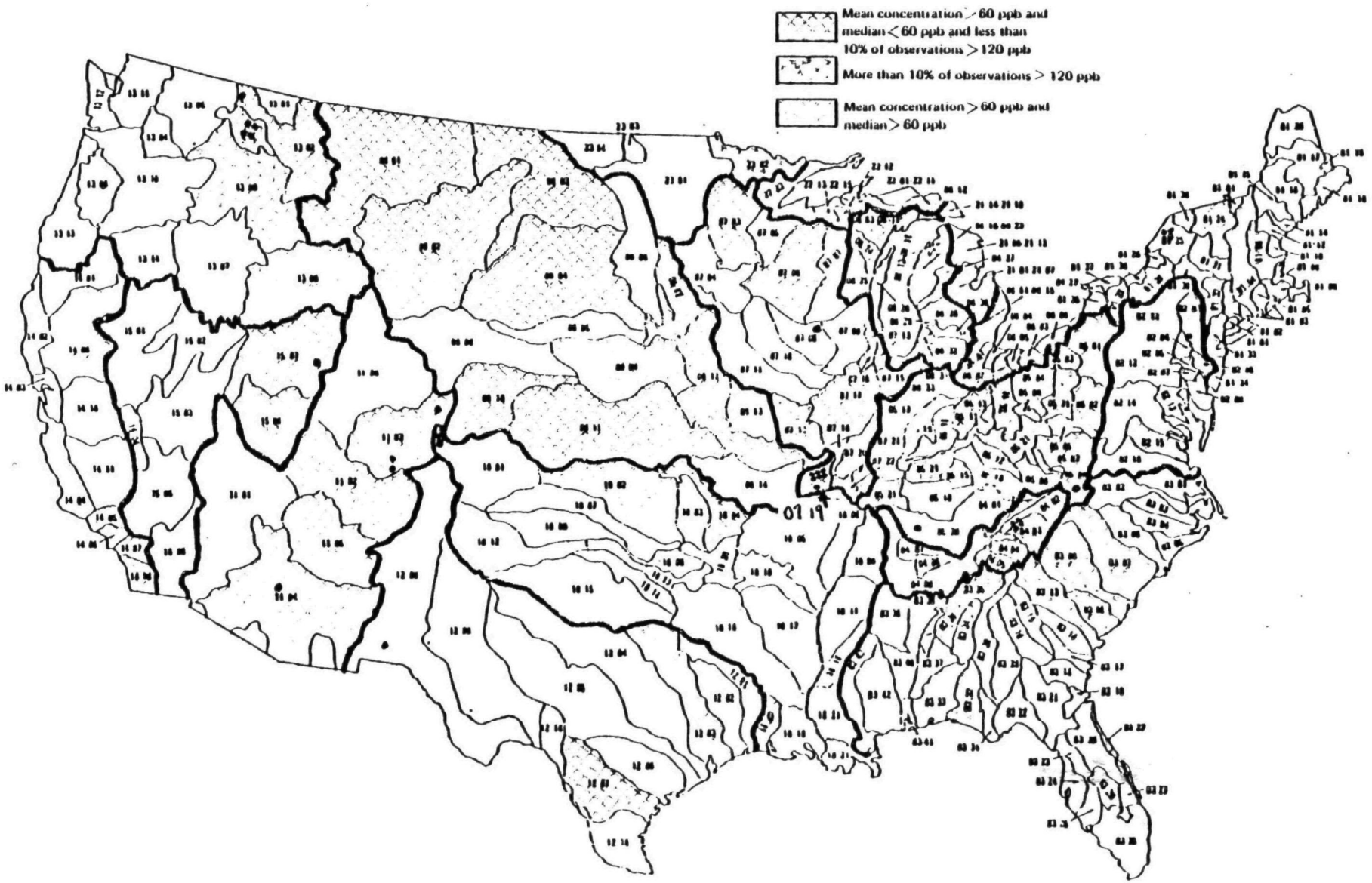


FIGURE IV–2 ZINC CONCENTRATION IN U.S.MINOR RIVER BASINS

The mean dissolved zinc range from 5.04 ug/l to 42.7 ug/l in naturally pristine areas. Dissolved zinc in non-benchmark stations ranged from 20.6 to 13,000 ug/l. The factors of difference ranged from 0.5 to 896.6 (STORET).

b. Streams Receiving Mined and Milled Wastewaters

In 1971, Wilson and Bolter (1972, as reported by NRC, 1979) sampled two mines for zinc discharge. One mine had a discharge of 1045 ug/l zinc and a resultant stream concentration (distance downstream not given) of 231 ug/l. In the other mine, the discharge was 411 mg/l and the resultant stream concentration was 328 ug/l. The study noted that zinc content of the streams returned to baseline levels within a few miles of the source.

Another study (Mink et al., 1970, as reported by NRC, 1979) compared the zinc concentrations in two forks of the Coeur d'Alene River. In the north fork, which has little mining activity, the zinc concentration during low volume flow was about 100 ug/l. In the south fork, which has considerable mining activity, concentrations were as high as 21,000 ug/l.

Jennett and Foil (1979) tested stream waters for zinc content in a lead and zinc mining area in southeast Missouri. The maximum concentrations found at 23 sampling sites were 36.0 mg/l total zinc, and 36.0 mg/l dissolved zinc. The average dissolved zinc concentrations between all the stations was approximately 1.1 mg/l, with a median level of .024 mg/l. All the high total and dissolved readings were associated with rainfall runoff.

c. Sediment

STORET data indicate that bottom sediments in U.S. river basins normally contain between 1 and 1,000 ppm of zinc, which is two to four orders of magnitude greater than concentrations found in river water. The distribution of observations is: 8% between 1 and 10 ppm, 65% between 10 and 100 ppm, 24% between 100 and 1,000 ppm, and 2% between 1000 and 10,000 ppm. The regions with the highest sediment concentrations are the Great Lakes, the mid-Atlantic states, New England, and the Pacific Northwest (see Table IV-2 and Section VII). For a more detailed description of zinc in sediment, see Section VI-B.

2. Zinc in Aquatic Organisms

The amount of zinc in aquatic tissues is dependent on both the water concentration and dietary intake; levels in freshwater fish are similar to marine species. Whole body zinc concentrations in aquatic animals are generally at least one order of magnitude higher than in the aqueous medium. In a study by the National Marine Fisheries Service (as cited in NRC, 1979), oysters were found to have the highest average whole body zinc concentration at 202 ppm. Other mollusks and crustaceans were generally much lower in zinc, with average levels of 12.5 ppm. Zinc concentrations in finfish range from not detected at the ppb level for the sablefish to 93.60 ppm for silver perch, with an average residue of 6.5 ppm (NRC, 1979).

3. Zinc in Plant Tissue

There are two separate mechanisms for zinc uptake, including sorption (absorption, adsorption and ion exchange) and metabolic assimilation. For the alga Golenkinia paricispina, zinc uptake involves ion exchange sites created by photosynthetic removal of CO_2. Planktonic algae and free floating vascular plants can obtain zinc from water but not from sediments; benthic algae and rooted plants can obtain zinc from both water and sediments. The concentration in brown seaweed varies with concentration in seawater. This is also true for the algae Chlorella. The differences between plant species in concentration factors is due to differences in ion exchange properties of their surfaces. Seasonal variations in zinc content range widely and is species dependent (NRC, 1979).

In terrestrial plants, zinc uptake by plant roots is dependent on diffusion and is independent of mass flow. Zinc uptake is also dependent on the soil solution concentration and on the ability of the soil to replenish zinc. Ranges of zinc uptake are from 2 to 4000 mg/day (by fresh weight). Zinc uptake and translocation in tomatoes, soybeans and squash plants increase with soil concentration. When zinc levels drop below 20 ppm dry weight in leaves, deficiencies may occur.

Concentrations of zinc in plant tissue are greatest in young plants and later decrease because of dilution and redistribution as tissues mature (NRC, 1979).

Concentration factors of zinc vary widely with species. They range from a low of 400 for Laminaria digitata to a high of 1100 for Fucus vesiculosis (both algal species) (NRC, 1979).

4. Zinc in Soil

a. Normal Soil

Normal soils contain between 10 and 300 ppm zinc, averaging about 50 ppm. In uncontaminated (by man) soil, zinc content is generally the same as the parent rock from which the soil is formed. Near sulfide deposits zinc content is generally higher. A recent study of 863 U.S. soils (see p. 31 of NRC, 1978) indicate an average of 54 ppm zinc in soil.

b. Soil Adjacent to Highways

Zinc levels in soils are generally higher near highways. In one study, zinc levels eight meters from a road ranged from 54 to 172 ppm in the top 5 cm of soil. In the top 10 to 15 cm of soil, zinc levels ranged from 11 to 72 ppm (NRC, 1979).

c. Soil Near Smelters

In studies of zinc contamination in soil near six smelters (Goodman and Roberts, 1971; Burkett et al., 1972; and Miesch and Huffman, 1972, as reported by NRC, 1978) high levels were found in the top few cm of soil (as high as 12,200 ppm in the top 10 cm of the soil 200 m from a smelter in Poland). Also apparent is considerable downward movement in some cases (as high as 467 ppm zinc in soil at a depth of 40 to 50 cm 200 meters from the same Poland smelter). Finally, the data also indicate decreasing concentrations of zinc in soil with increasing distance from the plant. Zinc levels seem to return to near normal levels at distances greater than 10 km from the smelters.

Another study (Buchauer, 1973, as reported by NRC, 1979) indicated higher concentrations of zinc in the soil. Within 1 km of the plant, zinc concentrations ranged from 50,000 to 80,000 ppm at a depth of 0 to 15 cm, with organic matter containing as much as 135,000 ppm of zinc. Again, in the study the concentration of zinc in soil fell off sharply with distance from the plant.

5. Zinc in Air

a. In Urban Communities without Mines or Smelters

The concentration of zinc particulates in urban areas throughout the United States range from 0.1 to 1.7 ug/m^3. Annual average airborne zinc concentrations throughout the United States, as indicated by data from the National Air Sampling Network (U.S. HEW, 1968, as reported by NRC, 1979) are generally less than 1 ug/m^3, ranging from <0.01 ug/m^3 to 1.60 ug/m^3. Sources of zinc in these urban atmospheres include motor vehicles, fuel oil and coal combustion, incineration, soil erosion, and industrial, commercial and construction activity.

b. Near Zinc Smelters

Although data are not available on the concentration of zinc in the air near zinc smelters, the data provided above on soil concentration near zinc smelters indicate that high concentrations are likely in the surrounding areas. As discussed above, zinc concentrations in soil return to near normal levels at distances of approximately 13 km. High levels of zinc in the soil close to the smelters suggest high concentrations may also be present in air.

B. ENVIRONMENTAL FATE

1. Overview

a. Methodology

The environmental fate of anthropogenic zinc is described as a result of discharges by processes which contribute significant quantities of the metal to the air, water and soil. The discussion emphasizes the form of zinc specific to the discharge, and its subsequent transport

upon release to the environment. A general overview of the environmental chemistry of zinc conducted by Versar (1979a) has been used as the basis to formulate judgments concerning the direction and rate of transport zinc assumes in an ecosystem. Studies available in the literature support the observations noted. Biological pathways have been treated separately from physico-chemical and bulk transport pathways. Uptake of zinc by terrestrial and aquatic biota are discussed in terms of the emission source. Again, the general uptake and metabolism of zinc by biota was reviewed by Versar (1979a,b).

b. Major Environmental Pathways

The major pathways of physical transport and qualitative rates at which transport occurs are designated in Figure IV-3. Atmospheric emissions (Pathway 1) have been segregated by point source and dispersive emissions. Combustion processes, such as incineration, smelting and coal combustion contribute to localized pollution; dispersive sources, such as emissions of zinc from automobile use, contribute to the concentration of zinc found in urban runoff. Pathway 2 follows the flow of zinc which originates from solid waste disposal dumps, and mine tailings. As environmental controls restrain further discharges to air and water, the quantity of zinc disposed upon land surfaces can be expected to increase. Zinc discharged with industrial process effluents into local surface waters or publicly owned treatment works (POTW) is reviewed in Pathway 3. The fate of zinc in POTW is described in Pathway 4.

Figure IV-4 gives a more general overview of all major pathways of anthropogenic zinc. The major impact on the land compartment (mostly at specific disposal sites) and the underlying groundwaters is to be noted. The migration of groundwaters containing zinc to nearby surface waters has not been shown in this figure since (1) the process is very slow, and (2) the current magnitude of this transport pathway is far from being well documented. Also not represented here is the high concentration of zinc in sediments with respect to the overlying water and the steep profile of zinc concentrations in soils subject to contamination

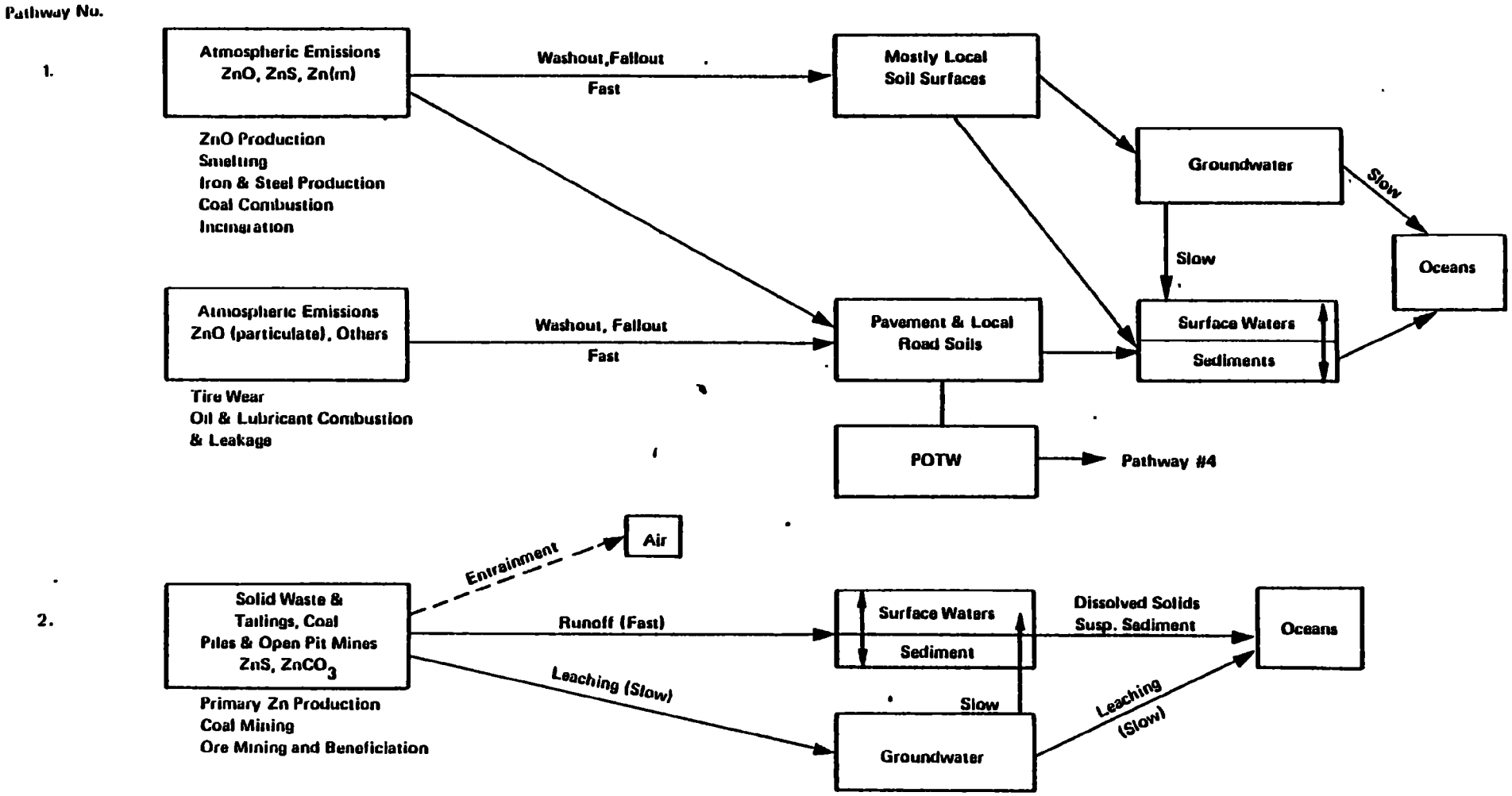


Figure IV-3 MAJOR ENVIRONMENTAL PATHWAYS OF ZINC EMISSIONS

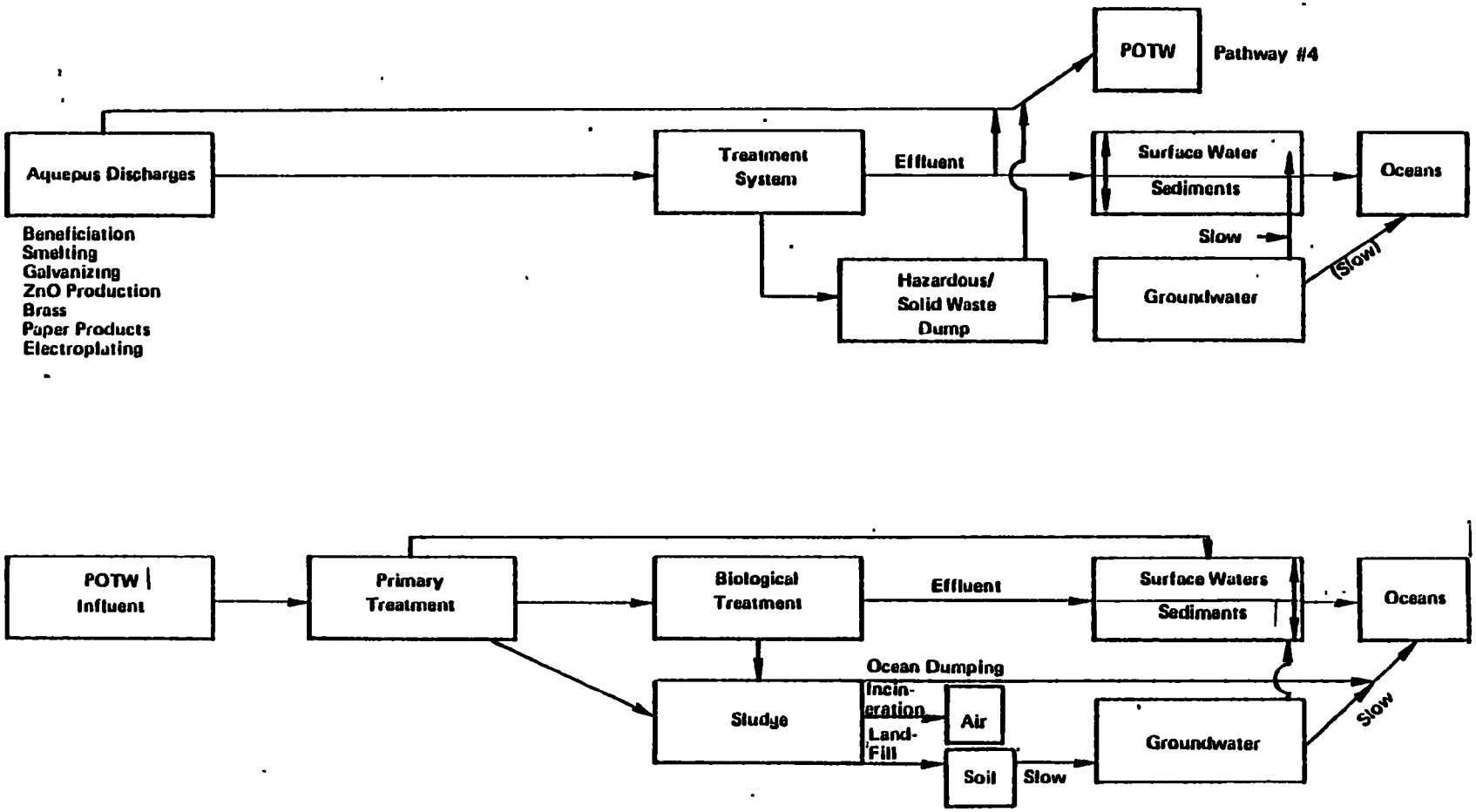
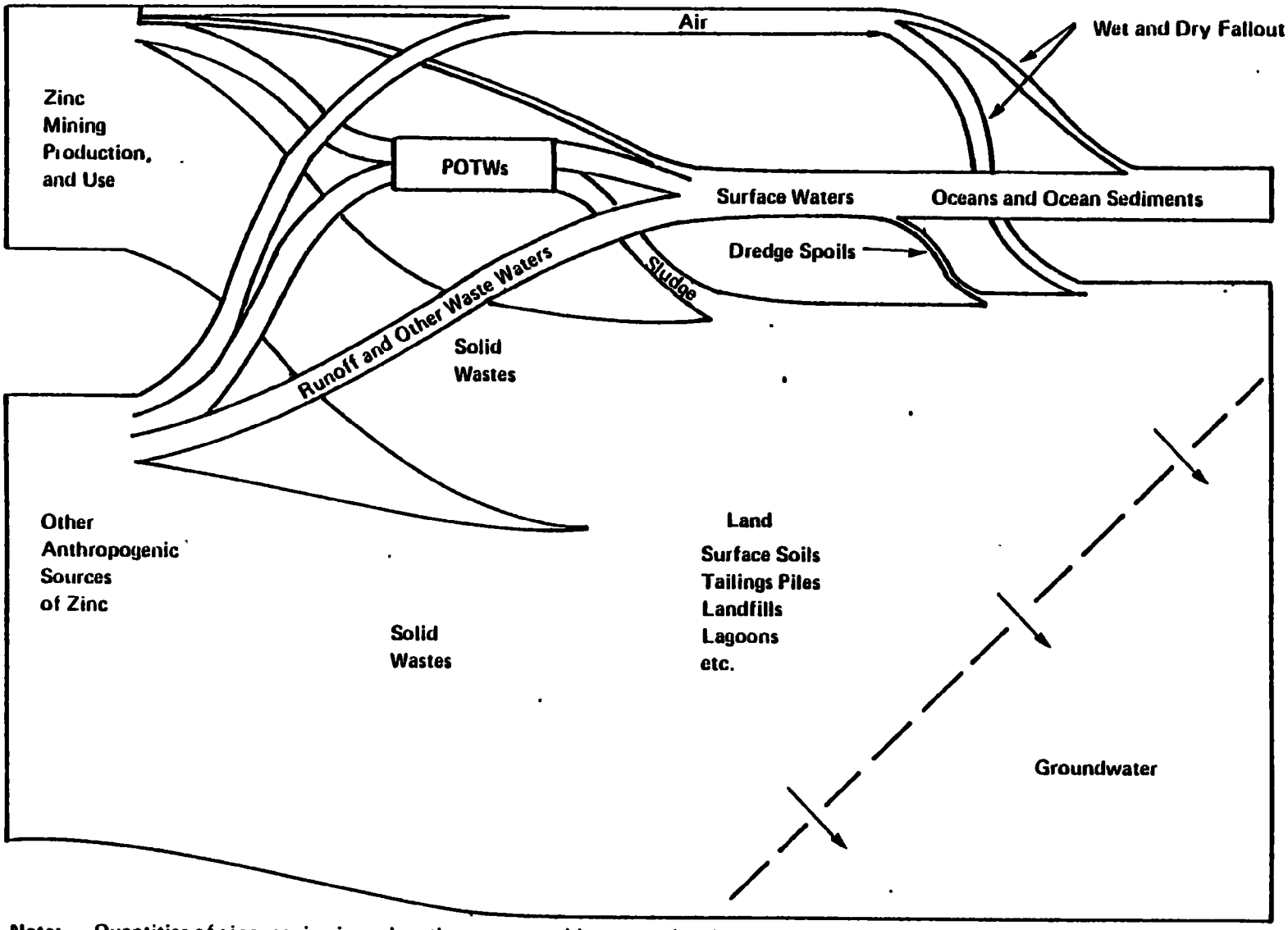


Figure IV-3 MAJOR ENVIRONMENTAL PATHWAYS OF ZINC EMISSIONS (Continued)



Note: Quantities of zinc moving in each pathway are roughly proportional to the thickness of each pathway shown. Slow movement from groundwaters to surface waters not shown.

Figure IV-4 SCHEMATIC DIAGRAM OF MAJOR PATHWAYS OF ANTHROPOGENIC ZINC RELEASED TO THE ENVIRONMENT IN THE U.S. (1979)

by airborne zinc. This figure also indicates the relative contributions of the zinc industry and all other human activities to the major zinc pathways; the major contribution of the latter category is to be noted.

c. Biological Transport

Biological transport of zinc includes uptake by plants from soils and water. Terrestrial biota are exposed to zinc from atmospheric deposition on the soil, landspread sludge and purposeful application as a nutrient to promote growth, or as a fungicide. In aquatic environments, biota are subject to zinc contamination by any process which eventually leads to transport in freshwater surface environments, or oceans.

Zinc is an essential nutrient to all organisms, and large bioconcentration factors are observed in nature. Bioconcentration factors for zinc go as high at 10^4 times the concentration of zinc in the water column. The most active accumulators of zinc appear to be the periphyton community (the attached submerged macroscopic plants and animals) and the benthic feeders. Zinc is not, however, biomagnified through the food chain. The major mechanisms for bioaccumulation in water are direct ingestion, and uptake from water via sorptive processes. The rate of bioaccumulation increases with increasing water temperature, pH, and hardness, although zinc is more toxic in acidic and soft waters. Documentation exists for reduced zinc uptake by algae in the presence of competing ions (K^+, Na^+, Mg^{++}), and by oysters as the salinity of the water decreases. One study found that only 0.6% of the zinc introduced to a model estuary ecosystem resided with the biotic compartment after 100 days; the remainder partitioned into the sediments.

d. Important Fate Processes

Zinc is concentrated in the sediments in aerobic waters sorbed primarily to hydrous iron and manganese oxides. Zinc also sorbs to clays, and to a lesser extent, organic materials. The bulk of zinc transported in the water column is in association with the dissolved solids (Perhac, 1974). In anaerobic waters, zinc will exist in the reduced phase as zinc sulfide. Bioaccumulation by the biota is greatest for algae and

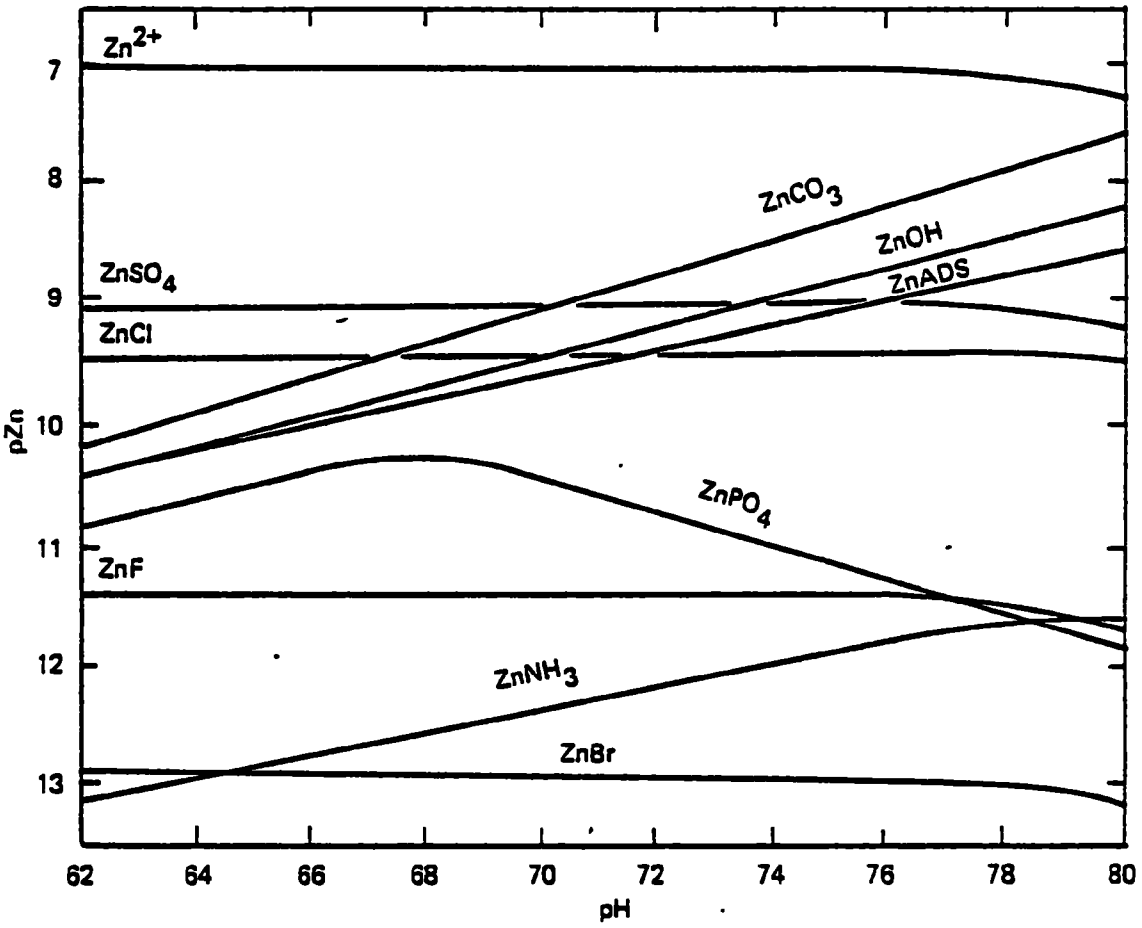
benthic feeders. In terrestrial plants, accumulation of zinc is promoted by low soil pH; in contrast, algae accumulate more zinc with increasing pH.

Atmospheric emissions of zinc will consist mostly of zinc sorbed to submicron particulate matter and the oxide of zinc. A large percentage of the zinc is expected to be short-lived in the atmosphere; dry fallout and washout of zinc particulates will contribute to deposition upon local soils.

2. General Fate Discussion

a. Aqueous Complexation

The concentration of soluble zinc in water is directly related to parameters such as pH, the oxidizing potential of the water (indicated as pE), the presence of other competing ions (Ca^{++}, Mg^{++}, Fe^{+++}), and the existence of other precipitating and complexing agents (OH^-, S^-, $CO_3^=$, $PO_4^{\equiv}$). Generally in a low pH environment, or at low alkalinities, zinc will remain as the free ion; at pH values above 8.0 or in waters of higher alkalinity, zinc will begin to complex predominantly with the carbonates, hydroxides and organic ligands. An early study by Hem (1972) demonstrated that the theoretical equilibrium species in a system comprised of 10^{-3} M carbon dioxide, and sulfur and 10^{-5} M zinc would be Zn^{++} ion below pH 7.5, and soluble zinc hydroxide forms above this pH. Within the pE and pH range considered, solid zinc sulfide, zinc hydroxide and smithsonite ($ZnCO_3$) were stable forms. For environmental applications, the model developed by Vuceta and Morgan (1978) may be a more adequate representation of a real system. Using a number of inorganic and organic cations and ligands, as well as a compound exemplary of surface sorption sites, the authors found that the model predicts that the zinc ion is the predominant species at pH 7. Zinc species present in minor concentrations (10^{-9}-10^{-10} M) were the chloride, sulfate, carbonate and hydroxide. Figure IV-5 illustrates the speciation as a function of pH.



Source: Vuceta and Morgan (1978).

FIGURE IV–5 SPECIATION OF Zn(II) IN NATURAL FRESH WATERS AS A FUNCTION OF pH IN PRESENCE OF 1.55×10^{-4} ha/L SiO_2

b. Absorption to Sediments and Suspended Solids

The above model is also capable of partitioning zinc between the dissolved and sorbed state. Using SiO_2 as a representative of available colloidal surface area, the model predicted that zinc would remain in the dissolved state. Introduction of iron and manganese oxides to the system caused significant adsorption of zinc. This distribution also is dependent upon the quantity of surface area for adsorption. Figure IV-6 reveals that copper, mercury, lead, nickel and cobalt will sorb before zinc for the same quantity of surface area. Metal adsorption to colloidal surfaces may be enhanced by organic ligands such as humic acids which coat a thin film over particles (Dana and Leckie, 1978).

c. Soils

The behavior of zinc in soils is dependent upon the adsorption properties of the soil, as well as the pH and redox potential of the soil solution. In aerobic soils, the solubility of zinc is controlled by $Zn_4(OH)_6(SO_4)$, at a pH of 5 and carbonate and sulfur concentration of 10^{-3} M; under anaerobic conditions, ZnS is the controlling species. Zinc is easily sorbed in soils; it is exceeded by copper, but not by lead and cadmium in adsorbing potential. Figure IV-7 illustrates the strong dependence of heavy metal adsorption onto hydrous oxides and soil particulates as a function of pH. At a pH of 5-6, adsorption is the principal means of removing zinc from solution. As the pH continues to increase, precipitation will become the dominant mechanism of removing zinc. Below a pH of 5, adsorption of zinc becomes insignificant. The presence of organic ligands such as humic acids, enhances metal adsorption at low pH values. Out of five ligands tested, humic acid was the most effective in this respect.

d. Summary Statement

The concentration and speciation of soluble zinc in the water column is dependent upon pH, pE, other cations and ligands, as well as colloidal surface areas. Within the pH range of concern, the Zn ion predominates in the water column. Sorption to hydrous iron and manganese oxides

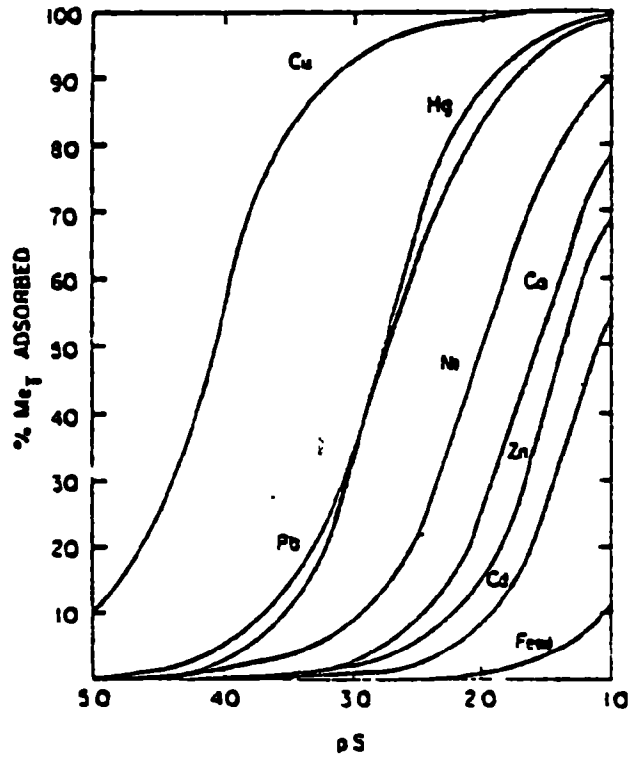
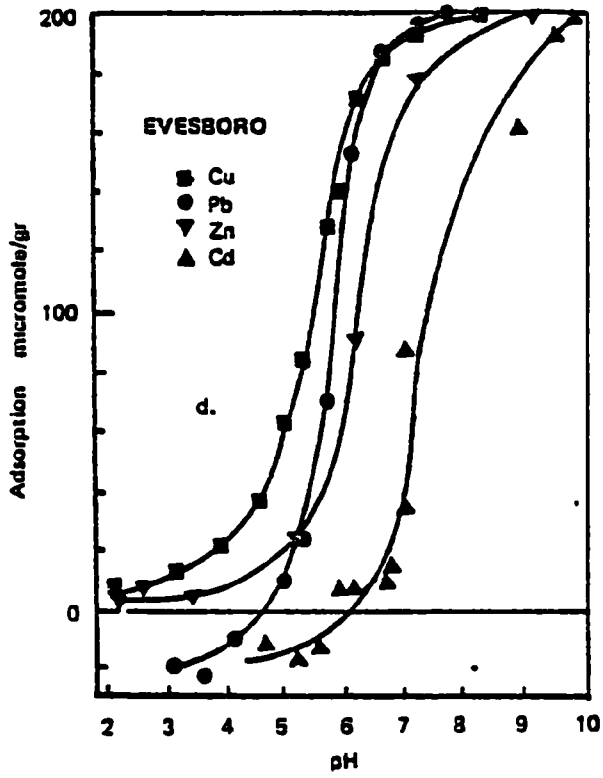


Figure IV-6: Adsorption of Heavy Metals in Oxidizing Fresh Waters (pH = 7, pE = 12, $pCO_2 = 10^{-3.5}$ atm., pCt-4.16) as a function of surface area of SiO_2 in ha/L. pS = -log (SiO_2) ha/L.

Reference: Vuceta and Morgan (1978).



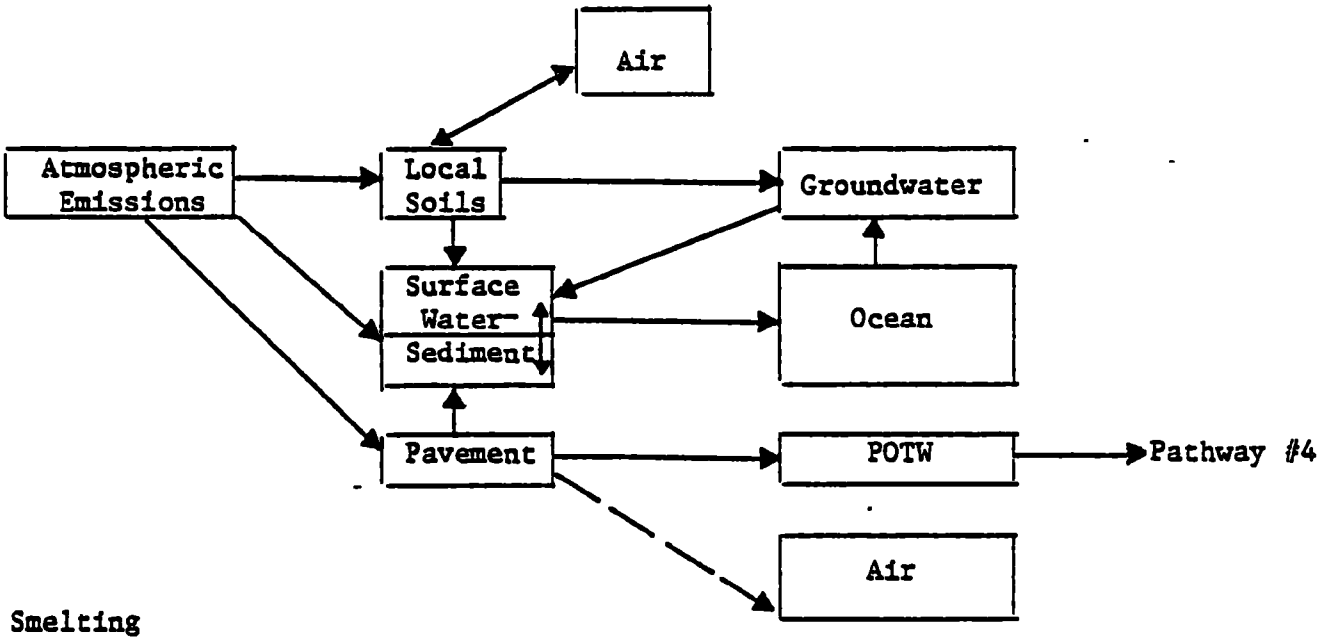
Reference: Huang (1977)

Figure IV-7 ADSORPTION OF HEAVY METALS ON SOIL MINERALS AND OXIDES

concentrates zinc onto suspended solids and sediments. In soils, zinc adsorbs within a pH range of 5-6, with the presence of organic ligands such as humic acids, enhancing this tendency. In acid environments, the zinc iron will be available in the soil solution.

3. Physicochemical Pathways

a. Pathway #1 - Atmospheric Transport



Smelting
Iron and Steel
Coal Combustion
Incineration
Primary Zinc Producers
Phosphate Processors

Sources: Pathway #1 describes the fate of atmospheric emissions of zinc as a result of thermal processes such as incineration and smelting, and from automobile use and other dispersion sources. The major industries which are responsible for zinc emissions into the atmosphere comprise iron and steel producers, primary zinc producers (from smelting operations), industries which combust coal, and refuse incineration operations. All of these emissions can be considered point sources of pollution and amount collectively to 6,100 kkg/yr. For the most part, the oxide of zinc (ZnO) is released. Small quantities of $ZnSO_4$, ZnS, as dust, and elemental zinc, as a vapor, are also a result of these processes.

A portion of the zinc found in urban atmospheres is due to the tire wear and combustion or leaking of lubricants and fuels (NRC, 1979). Tires contain 1.5% zinc by weight and wear at a rate of 1.2 kg/million km. Fuel and lubricating oils contain 30-1500 ppm zinc. The emissions from motor vehicles are small submicron particles with zinc sorbed in quantities of 0.1-10 ppm. Other sources contributing to urban area emissions are soil erosion, oil and coal combustion, and incineration.

The residence time of zinc in the troposphere, and distance travelled prior to deposition, are functions, in part, of the particle size of the airborne contaminant. NRC (1979) states that a horizontal retort distillation operation for the smelting of zinc produces particulates 34% of which are less than 2.5 μm in diameter, 35% range from 2.5 - 5.0 μm, and 31% are greater than 5.0 μm.

Most of the particulates are scavenged by control systems. However, submicron particulates from dusts (ZnS) and zinc oxide fumes escape the collectors. In fact, Jacko et al. (1975) found that trace metals exhibit a preference for smaller particulate sizes, as demonstrated in the emissions from a scrubber controlled municipal incinerator. One reason given to explain this occurrence, in addition to the existence of submicron metallic oxide fumes, is the selective adsorption of the trace metals upon small particulates due to the greater surface area to volume ratio. The work of Coles et al. (1979) supports this trend. Zinc partitioned amongst coal fly ash particulates in the following manner: 68 ppm on 18.5 μm fraction; 189 ppm on 6.0 μm; 301 ppm on 3.7 μm; 590 ppm on 2.4 μm.

Deposition on Soils: Once in the atmosphere, deposition of the particulates via rainout or dry fallout proceeds quickly, resulting in a mean residence atmospheric time of 7-30 days (Versar, 1979b). Most of this deposition will occur in the local vicinity of the emission source. Some of the zinc will clearly be transported over much greater distances; the fallout of this material may vary significantly from location to

location depending on climatic and other factors. In one study of marine sediments about 100 km offshore (south) of Los Angeles, the concentration of anthropogenic zinc in the surface layers (top 2 cm) was used to estimate a deposition rate of 0.2 μg $Zn/cm^2/yr$ (Bertine and Goldberg, 1977). The sample location was in an area that would not have been affected by wastewater discharges to the ocean, but would be affected by surface winds from the Los Angeles area. Thus, the authors suggested that the deposition rate calculated was due to atmospheric transport and fallout.

Figure IV-8 illustrates the deposition of zinc particulates from three smelters as a function of distance away from the smelter. Zinc concentrations in surface soils are seen to rise dramatically within a 10 km radius of such sources. The zinc concentrations were measured within the top few centimers of soils (0-10 cm). In all cases, the concentration of zinc far exceeds that of background levels, which is on the order of 50 - 54 ppm (NRC, 1979). In one case, the zinc content in the soil ranged from 50,000 - 80,000 ppm within 1 km of the emission source.

Most of the particulate zinc which is deposited exists as the oxide. The more soluble $ZnSO_4$ may also be present as well as zinc ion absorbed to particulate matter. Initial mobility will depend upon the solubility of the zinc compounds deposited. Zinc oxide is fairly insoluble in water ($\sim$1.6 mg/l at 29°C) as is zinc sulfide ($\leq$6.9 mg/l at 18°C); zinc sulfate however, is highly soluble in water (86 g/100 g water at 80°C).

Figure IV-9 demonstrates the mobility of zinc within the soil profiles near three different smelters. The bulk of the zinc inhabits the top 0 to 10 cm of soil. The concentrations fall off rapidly below this depth although the concentrations of zinc encountered exceed typical background concentrations. Versar (1979b) states that zinc, once soluble, is highly mobile in the soil environment due, in part, to weak sorption upon clay minerals, iron and manganese hydrous oxides, and organic matter. This mobility increases as the pH level of the soil solution

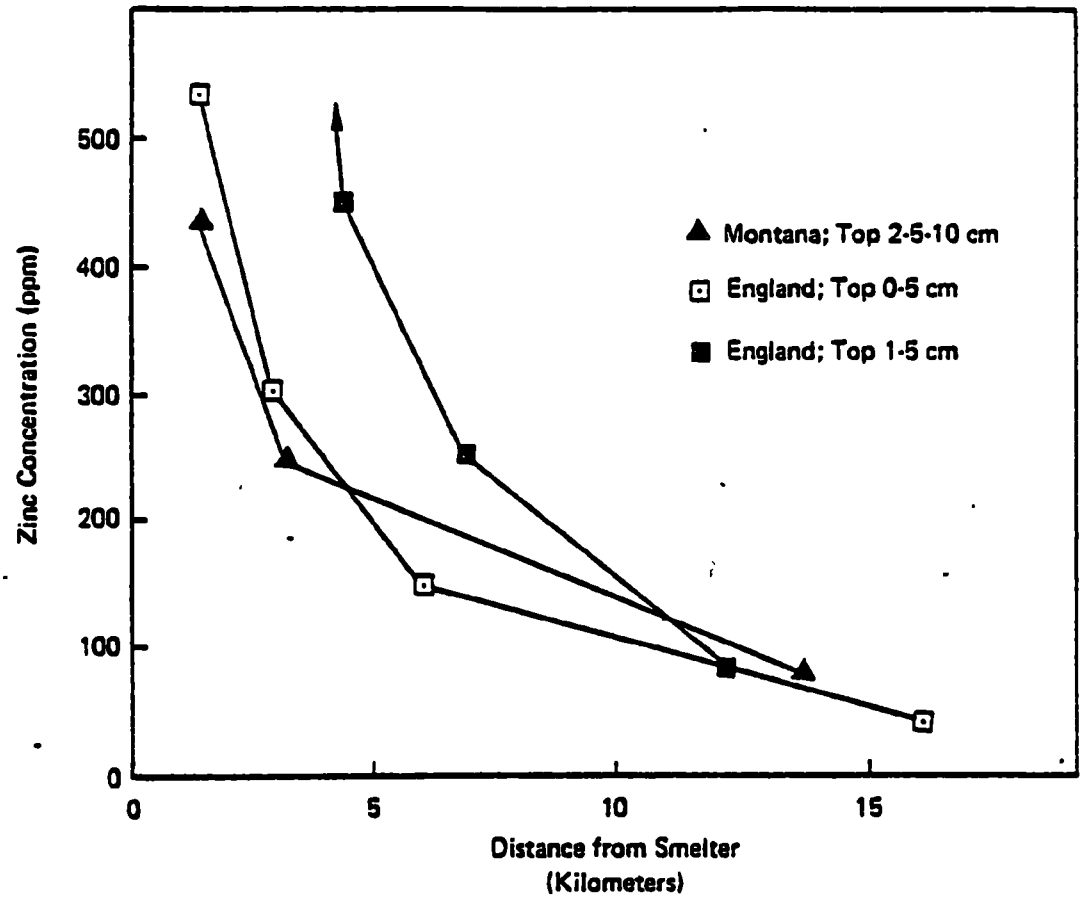


Figure IV-8 ZINC CONTENT OF SOILS NEAR SMELTERS

Source: NRC (1979)

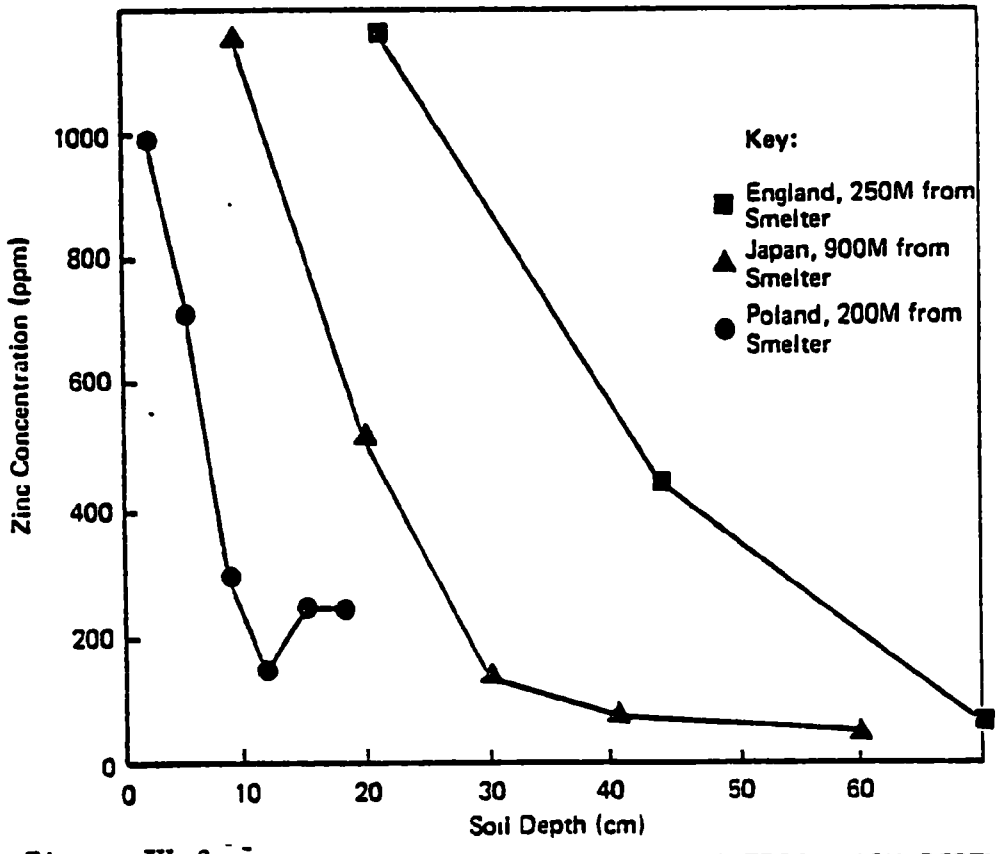


Figure IV-9 ZINC CONTENT OF SOILS NEAR SMELTERS vs. SOIL DEPTH

Source: NRC (1979)

decreases. An example of zinc mobility in soils vs pH can be found in the work of Huang et al. (1977) which concluded that mobility was greatest below a pH of 5-6. Transport through the soil profile of oxidized soils will be dominated by the Zn^{++} ion, making possible bioaccumulation and groundwater contamination (Versar, 1979b).

According to Versar, the deposition of zinc upon the soil surface allows, of course, for the entrainment of soil particles containing zinc back into the atmosphere. This cycle is partially dependent on groundcover and soil moisture, and will continue indefinitely with the same type of chemical phenomena dictating the transport of airborne zinc. Surface runoff of soil particulates will also result in the introduction of zinc to surface waters and sediments. Bioaccumulation of deposited zinc will be another pathway both among terrestrial and aquatic organisms, and will be discussed in a later section.

Road Surfaces: Most of the zinc released to the local urban atmospheres immediately falls out onto road surfaces and local soils. That which deposits upon a pavement is subject to runoff into the nearest drainage system. The concentration of zinc found in street surface runoff after a fairly heavy rain in seven cities ranged from 0.03-0.95 kg/curb mile; the average was 0.34 (NRC, 1979). Of the metals Cr, Cu, Ni, Hg, Pb and Cd, zinc comprised the largest percentage (40%) of the total heavy metals found in street runoff. The zinc distribution on the street by land use classification was: 38% residential, 44% industrial and 24% commercial. The runoff will flow in the direction of either natural or man-made drainage basins. The fate of zinc as it enters a Publicly Owned Treatment Work (POTW) will be discussed separately. In streams, zinc is quickly sorbed by clays, hydrous iron and manganese oxides and - to a lesser degree - by organic matter. The stream sediments become highly enriched by zinc; the bulk of zinc transported in the water column is associated with dissolved solids (Perhac, 1974). A more detailed explanation of this fate process will be discussed in Pathway #2. The ultimate sinks for zinc transported with stream suspended solids or in the dissolved state are: 1) lake sediments, and 2) ocean sediments.

Figure IV-10 illustrates the pattern of deposition of zinc onto soils as a function of distance from a highway. The plots are derived from the average of four studies found in Table 3-3 in a review of zinc performed by the NRC (1978). From the graph it appears that soils at the 10-15 cm depth are hardly affected by zinc deposition as a function of distance whereas the top soil surface layer contains a high of about 112 ppm Zn at an 8 meter (.005 mi.) distance from the highway to about 35 ppm at 32 m (0.02 mi.) from the highway.

Groundwater: Groundwater contamination by the zinc ion will be a function of a number of parameters, the two most obvious being the depth to the groundwater table, and the composition of the soil.

Jennett and Linneman (1977) state that the potential for heavy metal (Zn and Pb specifically) contamination of shallow aquifers (<300 ft. deep) from wastes applied to land may be large. However, one can say that the transport of zinc from soil to groundwater will be a slow process (in comparison to deposition) and can be considered the "rate determining step" in the environmental transport of zinc via the pathway.

Surface Waters and Oceans: Transport of zinc from groundwaters to surface waters and oceans (in Pathway #1) will have a negligible effect on these waters due to the slowness of the process and the dispersed nature of the initial pollution.

Summary Statement: Wet and dry deposition will quickly remove zinc released to the atmosphere as a result of industrial combustion processes, and non-point source atmospheric emissions. Zinc will accumulate in the top few centimeters of the soil as the insoluble oxide, or sorbed to hydrous iron and manganese oxides, clays and organic matter. A soil solution pH less than about 6, promotes the dissolution and transport of the zinc ion on the soil profile. Groundwater contamination may occur over time.

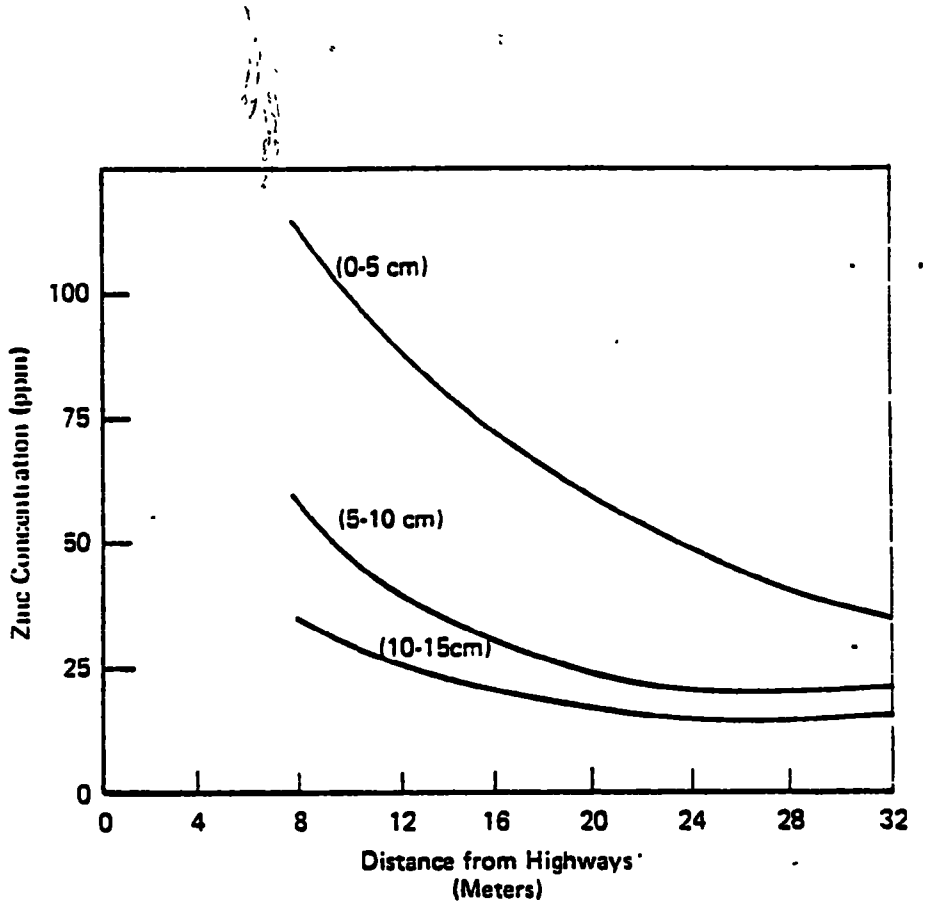
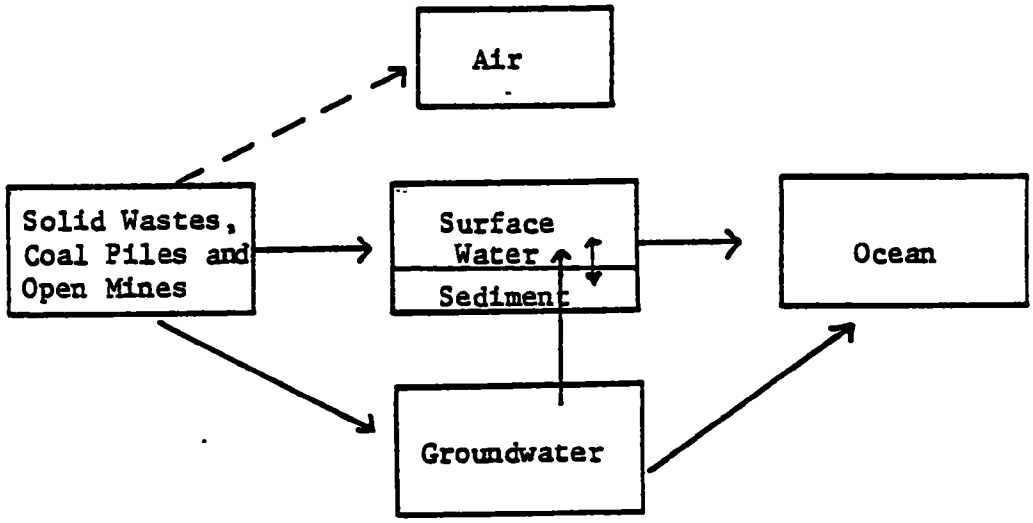


Figure IV-10 AVERAGE Zn CONTENT IN SOILS NEAR HIGHWAYS AT DIFFERENT SOIL DEPTHS

Source: NRC (1978)

Deposition of non-point source atmospheric emissions of zinc in urban environments will contribute to localized pollution of drainage basins due to surface runoff. In streams, the major portion of zinc will partition with the sediments. The remaining zinc in the water column will be associated predominantly with the dissolved solids.

b. Pathway #2 - Solid Wastes Tailings and Coal Piles, etc.



Sources: These materials arise from mineral ore processing, and coal mining. The solid wastes result from the overburden of surface mining, and the low grade portions of mineral ore deposits. The tailings which are highly concentrated in minerals are produced as a final waste product of mineral concentrating operations (Martin and Mills, 1976).

Since 1873, the production of zinc has contributed 883 million metric tons of tailings, and 2208 million metric tons of combined tailings and waste (Martin and Mills, 1976). Disposal of these wastes in the 19th and much of the 20th centuries was without regard to environmental considerations, and thus erosion and weathering contributed to adverse ecological impacts. Currently, however, tailings are left to settle in lagoons, after treatment with lime to raise pH and precipitate heavy metals (NRC. 1979). Backfilling and surface mine reclamation also serve to reduce the amount of zinc found in surface runoff. Zinc in some solid wastes is now recovered.

The nature of the solid wastes and tailings depends upon the nature of the ore. Zinc ores exist as sphalerite (ZnS), hemimorphite (H_2ZnSiO) and smithsonite ($ZnCO_3$), whose host rocks are carbonates, granites, slates and quartizes. Sphalerite, the ore most commonly mined (see Section III) is found concentrated in basaltic rocks, which are of igneous origin. The gangue of this ore often contains pyrite (FeS) and fluorite.

Coal piles and solid wastes from coal cleaning processes may also be considered sources of zinc for the pathway being considered. One survey of 101 coal samples from U.S. coals showed a mean zinc concentration of 272 ppm with a range of 6 to 5350 ppm and a standard deviation of 694 ppm (Mezey, 1976). The mineral sphalerite (ZnS) has been identified in coal and the concentration of zinc has been shown to correlate with that for cadmium. Other data have shown that the zinc is mostly concentrated in the mineral matter in coal and has little affinity for the organic matter (Mezey, 1976). This implies that zinc may be concentrated several fold in the solid wastes from any coal cleaning operation designed to remove pyritic sulfur and/or other mineral matter from coal.

Acid Mine Drainage: Tailings and solid waste from mineral mining aid in the formation of mineralized acid discharge. This is caused by the exposure of fine particulates to air, upon which the oxidation of metal sulfides results in the formation of H_2SO_4. The impact of acid mine drainage (AMD) to local surface waters is largely dependent upon the alkalinity, or buffering capacity, or the waters upstream and downstream of the point of discharge. Zinc sulfide is found associated with igneous rocks and is concentrated in proportion to the concentration of pyrite found in the ore. Pyrite is easily oxided to $Fe(OH)_3$, producing acidic waters as a consequence of the reaction. It may be assumed that sphalerite (ZnS) is also easily oxidized. Igneous rocks are low in calcerous material and therefore the water which passes over the gangue has little opportunity to dissolve carbonates and become a buffered solution. For these reasons, a large potential exists for zinc releases to the local waters of a mined region.

Fate Processes in Streams: Figures IV-11, IV-12, and IV-13 summarize these observations for a stream which receives mine drainage (Martin and Mills, 1976). The mine occurs at kilometer 35 and the confluence of two streams occurs at 0 kilometers. The waters which reach the mine area experience an immediate drop in pH, as well as in bicarbonate concentration. At the same time, the concentration of dissolved zinc increases dramatically.

These figures also give an indication of how the stream recovers as a function of distance. Reduction in the concentration of zinc is caused by precipitation, adsorption and dilution (Martin and Mills, 1976). A literature review of the fate of zinc performed by Versar (1979a) concludes that sorption is the dominant process affecting the reduction of zinc in surface waters. Sorption upon hydrous iron and manganese oxides, organic matter and clays results in enriched sediments and suspended solids so that Zn concentrations in these fractions are in the ppm range, while the water column exhibits concentrations of zinc in the ppb range. Holcombe (1977) found that zinc draining a mined area sorbed preferentially to manganese oxides rather than iron oxides. Iron oxides exhibit a positive surface charge at low pH's, repelling the zinc ions while the opposite is true of manganese oxides.

Perhac (1974) investigated the distribution of zinc within stream bed sediments and the water column of three rivers in Tennessee. The results obtained are summarized in Table IV-4. These rivers do not drain mined areas. The results indicate that the bulk of zinc is transported through surface waters in the dissolved phase, although the highest concentrations exist in the particulate fractions. Although these data show a higher zinc concentration in the coarse particulates than in the colloids, other data have shown an inverse correlation between zinc concentration and sediment grain size (Versar, 1979b).

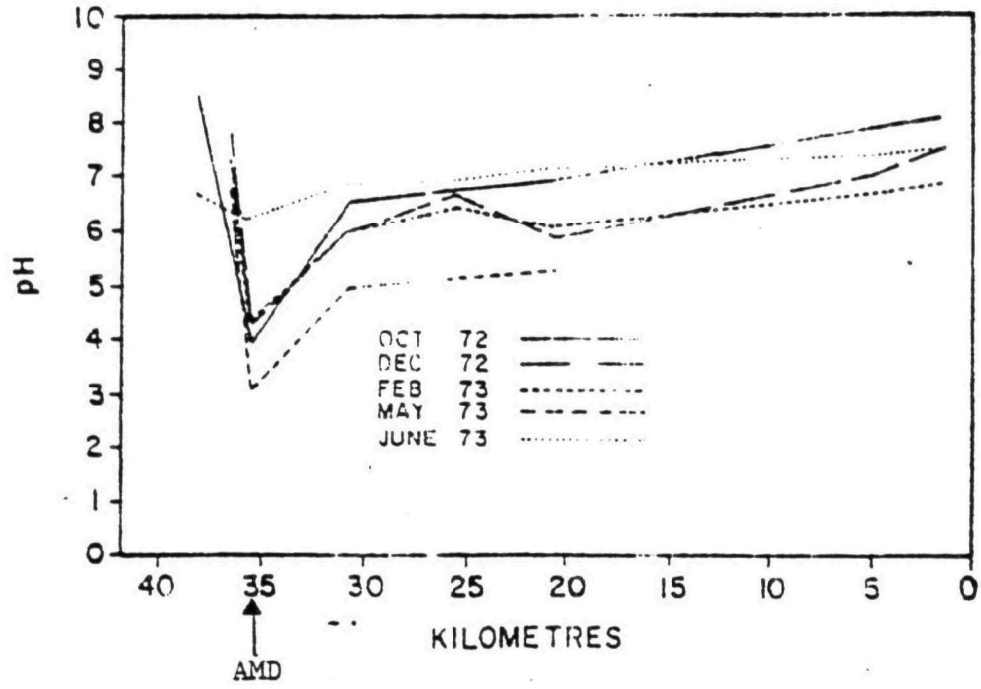


Figure IV-11 The pH in Kerber Creek

Source: Martin and Mills (1976)

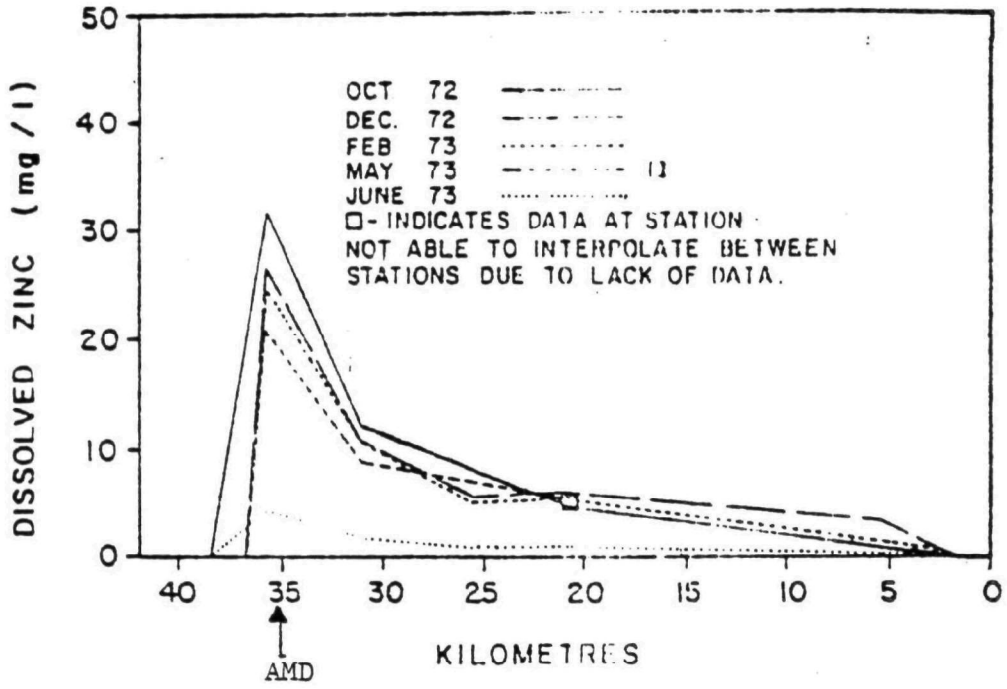


Figure IV-12 Dissolved Zinc Concentrations in Kerber Creek

Source: Martin and Mills(1976)

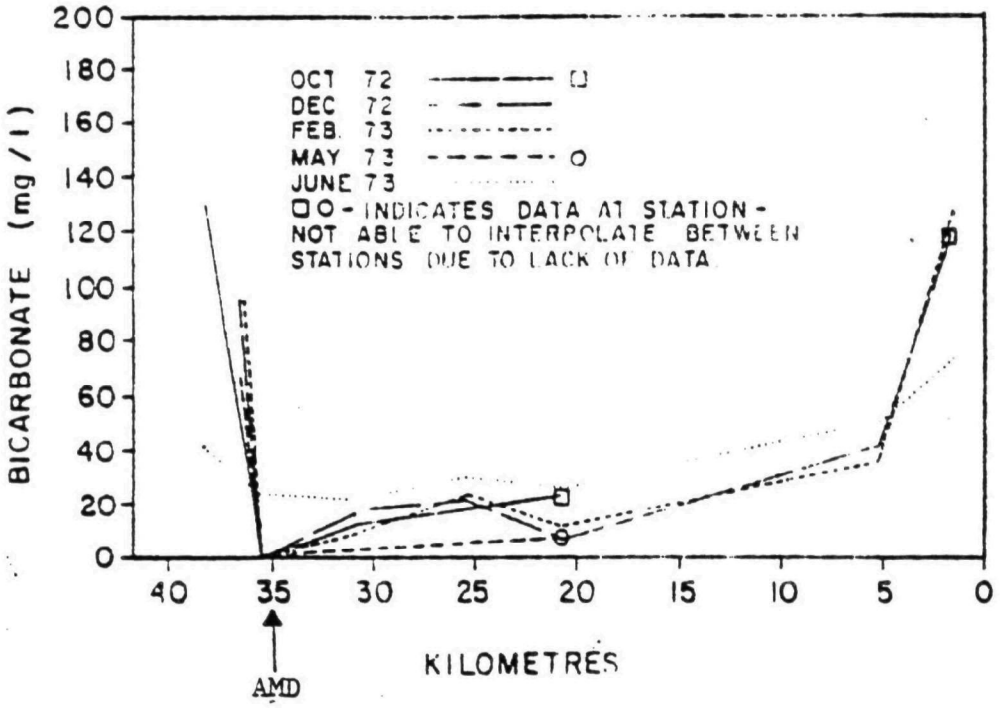


Figure IV-13 Bicarbonate Concentrations in Kerber Creek

Source: Martin and Mills (1976)

Table IV-4

Distribution of Zinc in Stream Waters

	Particle Size μm	% Total Solids	Zinc Concentration (ppm)	% Total Zinc
Water	--	--	0.01-0.033	-
*Dissolved Solids		91.8-98.6	33-185	53-92
Colloids	<0.15	1.3-8.6	50-1840	0.4-2.4
Coarse Particulate	>0.15	0.04-0.35	256-2480	7-46

* Brought to dryness.

Source: Perhac (1974)

The distribution of zinc in rivers, reservoirs and their sediments, and the effect of stream velocity on these concentrations has also been reported by Williams et al. (1973). A detailed study in one river showed no correlation existed between stream flow and the concentration of soluble zinc. However, a strong positive correlation did exist between stream flow and the concentration of zinc in the resuspended bottom sediments, mostly in the form of ooze deposits, present in the water column following increased flow and scour from heavy rain runoff. It should be noted that any event which leads to the resuspension of such polluted sediments (e.g., heavy rains, in-stream concentration, dredging, rapid dam draw-down, etc.) can increase the concentration of zinc in the water column several fold and lead to heavy kills of aquatic life. Synergistic effects of zinc with other pollutants in the sediments (as well as the toxic effects of other pollutants alone) are also important in these cases.

The profile of anthropogenic zinc in freshwater sediments appears to differ from the sharp, initial drop-off profile seen in soils (described previously). One example, shown in Table IV-5, indicates that a well-mixed layer of 15-20 cm may exist for the top sediments; beneath this level concentrations then drop fairly quickly to background levels. The data of McIntosh and Bishop (1976) also indicate such a profile. The distance zinc travels in streams while sorbed to suspended sediments is a function of the stream velocity. Therefore, one can expect greater zinc mobility in April due to rain and snow melt than in August.

Groundwater Contamination: Contamination of groundwaters by metals leaching through tailing piles has been cited by Martin and Mills (1976) and Mike et al. (1972). Leaching of AMD is a function of the tailing pile porosity. Tailings from years ago were higher in porosity, allowing for more active leaching to occur.

Table IV-5

Profile of Zinc in Selected Sediment Cores

from Foundry Cove (Hudson River)

Depth (cm)	Concentration (ppm)		
	Core No. 15	Core No. 6	Core No. 10
0-5	316	358	309
5-10	321	342	307
10-15	305	314	274
15-20	224	235	238
20-25	124	219	168
25-30	97	119	-
30-35	90	79	106
35-40	80		107
40-45	84		
45-50	80		
50-55	82		

Source: Bower et al., 1978

Groundwater contamination from tailing pond waters was researched by Mink et al. (1972). They found that the high levels of zinc in the groundwaters was due to leaching of old mine tailings in contact with a high water table. The waters from the settling pond served only as a recharge basin for the aquifers, thereby keeping the old tailings in constant contact with water. They concluded that the groundwater pollution was not the result of present day mining waste disposal.

Gibb (1976) investigated the extent of groundwater pollution from a surficial toxic waste disposal site at a secondary zinc smelting plant. The site was underlain by low permeability silts and clays and covered by a 1-10 foot thick layer of heavy metal rich cinders and ashes which had been generated by 85 years of smelting. The highest concentrations of zinc were observed directly beneath the cinders, where concentrations exceeded 10,000 ppm. However, analysis of well water samples indicated levels of zinc at less than 1mg/l with very little monthly variation.

Analysis of leachate from a power plant ash pond revealed 26.2 mg/l particulate zinc, and 480mg/l soluble zinc (Theis and Richter, 1979). Within 100 meters of the pond, zinc was available as the ion, and as zinc sulfate. At a distance of 400 meters from the pond, most of the zinc was associated with hydrous iron and manganese oxides. Once in the groundwater, zinc may follow one of the following routes: 1) surface water recharge, or 2) discharge into the ocean. Both of these processes will be slow.

Ultimate Sinks: Lakes or oceans which are fed by streams or groundwater from mined areas may serve as the ultimate sink for zinc. The effect a polluted stream has upon a lake is a function of the volume of pollutants introduced to the lake, and the natural buffering capacity of the lake. Martin and Mills (1976) suggest that the most notable effects of acid mine drainage will result at the mouth of the stream. It is likely that the stream drops its suspended load when its velocity is slowed upon entry into the lake. An example of adverse effects to fish

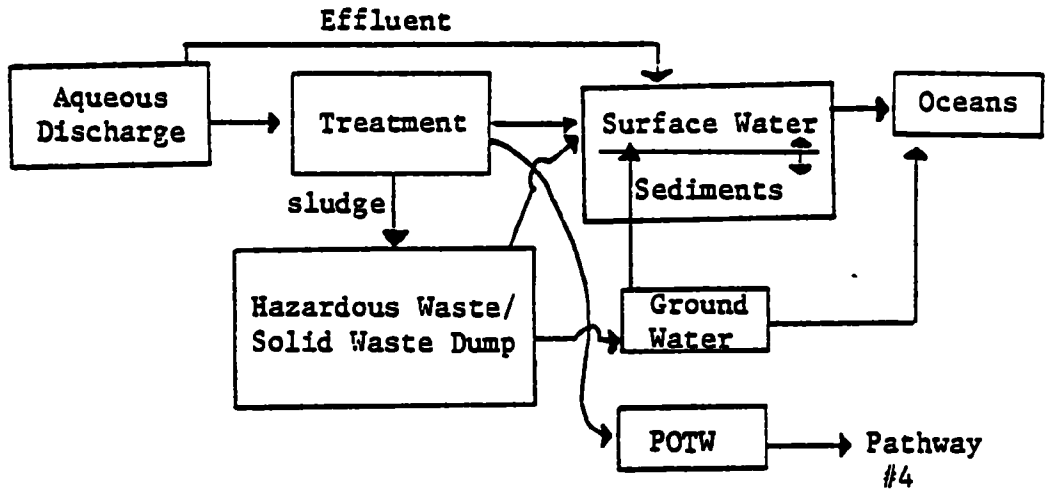
reproduction in a California lake has been cited by Martin and Mills (1976). McIntosh and Bishop (1976) researched the partitioning of zinc among the dissolved solids, suspended solids and sediments of a lake fed by a polluted stream. The dissolved solids were categorized as that fraction of solids which passed through a 45 μ filter; the suspended solids were that fraction which was retained. The results are listed below:

Dissolved Solids - 125 μg/ l Zn
Suspended Solids - 32 μg/ l Zn
Sediments - 7530 ppm Zn (dry wt; top 5 cm)

Summary Statement: Transport of zinc from mine tailings, and solid waste piles will primarily affect surface water quality. The concentration of zinc in stream waters is reduced by precipitation, adsorption, dilution and reduced stream flow, leading to zinc-enriched sediments. The trace quantity of zinc remaining in the water column is associated with the dissolved and suspended solids. The relative importance of these components is variable, although the larger portion of zinc is generally transported in the dissolved form.

Evidence of groundwater contamination has been cited. Transport of zinc from the tailings to the groundwater does not appear to be a fast process, although transport to surface water may be more important, especially from old tailings. Sorption of zinc by soil hydrous iron and manganese oxides retards the movement of zinc in the soil profile.

c. Pathway #3 - Industrial Wastewater Effluents



Sources: Pathway 3 considers the fate of zinc discharged with industrial wastewater effluents. The industries which discharge zinc are numerous. The major ones are involved in the production of brass, zinc oxide, iron and steel, galvinized items, and ore beneficiation. Industrial effluents are discharged with or without treatment into natural waters or municipal wastewater treatment systems. Yost and Masarik (1977) have investigated the efficiency of "chemical-destruct" systems or wastewater treatment systems employed by the metal finishing industries. The results obtained from neutralization and precipitation of zinc oxides for one plant are summarized below.

Zinc Concentration, mg/l			Distribution of Zinc in Aqueous Discharge	
Before Treatment	After Treatment	% Removal	% in Dissolved Solids	% in Suspended Solids
8.6	0.35	96	0.3-7	93-97.7

These results indicate that zinc removal in this case is a very efficient process. The bulk of zinc discharged with industrial effluents is associated with suspended solids.

The effluent of treated industrial process waters will be discharged to municipal sewers or surface waters. The fate of zinc once it reaches a Publicly Owned Treatment Work (POTW) will be discussed in Pathway 4.

In another study, a creek receiving industrial and municipal waste effluents was compared to nearby non-industrial use streams (Mathis et al., 1973). The authors suggested that the extent of urban use is best reflected by the composition of the river sediments. The contaminated creek had a mean concentration of 81 ppm; the non-industrial-use streams contained 30 ppm.

The study by McIntosh and Bishop (1976) reveals the effects of zinc concentrations in the water column when a major metal plating operation discontinued discharging into a small lake in 1975. The results for the average zinc concentrations in the lake are listed below:

	1974	1975
Dissolved Zinc (ppb)	224	30
Zinc in Suspended Solids (ppb)	51	16

The lake sediments acted as a sink for zinc; the average concentration in the top 0-5 cm was 7530 ppm. The authors did not distinguish between 1974 and 1975 deposition. Concentrations were found to decrease significantly below the top 10 cm as shown in Figure IV-14.

The transport of zinc in surface waters has been reviewed in greater detail in Pathway #2. Briefly, the concentration of zinc decreases rapidly downstream from the point of discharge due to sorption, dilution and precipitation. The work of Perhac (1974) revealed that sorption onto particulate matter yielded the largest concentrations of zinc in the water column. However, the bulk of zinc in the water column is transported in association with dissolved solids, since this fraction makes up about 95% of the total zinc.

The sludge generated by effluent treatment contains primarily zinc hydroxide and zinc carbonate in a precipitated form. The sludge is normally disposed of in a solid or hazardous waste dump, or settling pond. A properly designed hazardous waste dump should prevent further translocation of zinc due to leaching. Some sites collect the leachate, and send it to

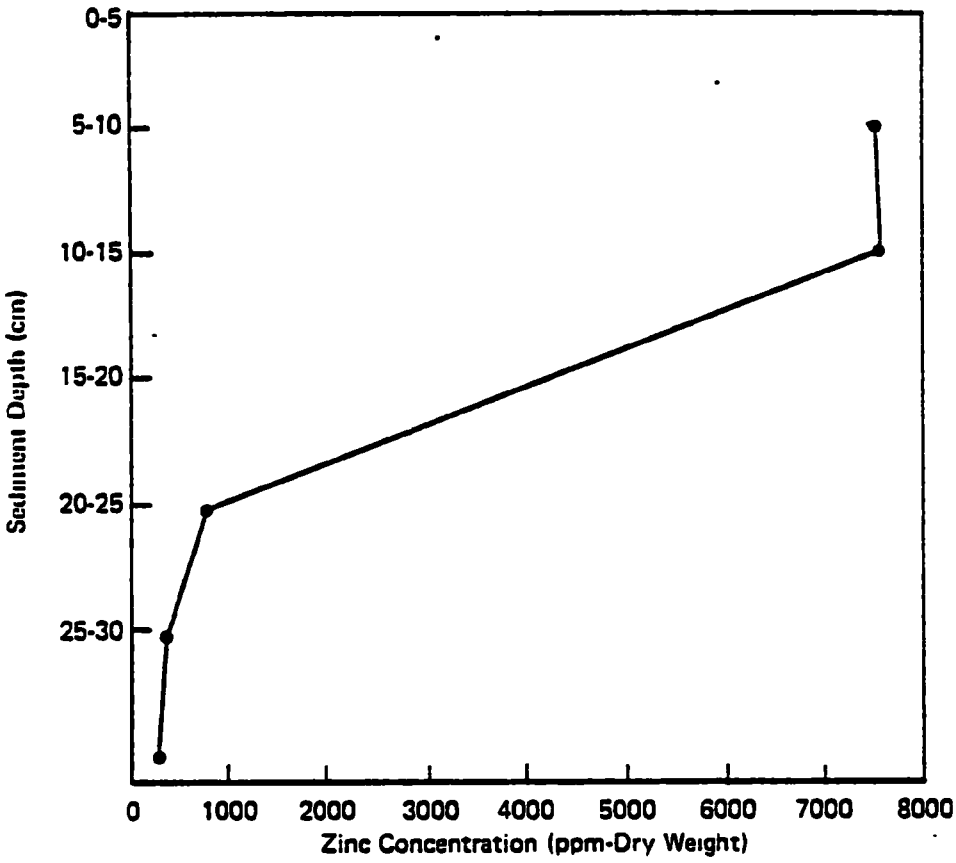


Figure IV-14 CONCENTRATION OF ZINC vs. SEDIMENT DEPTH OF A POLLUTED LAKE

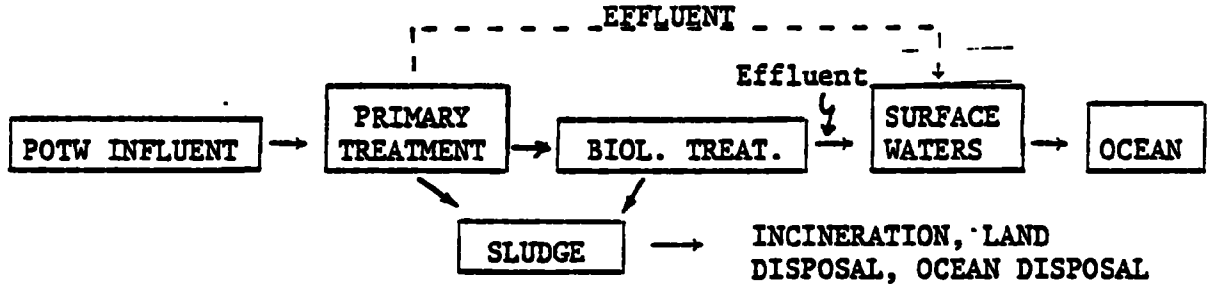
Source: McIntosh and Bishop (1976)

a POTW (with or without further treatment). Groundwater contamination is a possible occurrence in a poorly operated landfill or settling pond. The speed at which zinc is translocated via this pathway is slow. The fate of zinc in solid waste sites was reviewed in Pathway 2.

Ultimate Sinks: The sinks of zinc associated with treated industrial effluents are, in the short-term, hazardous waste dumps, settling ponds, or sites used for the dispoal of sludge generated by POTW's. The long-term sinks, as discussed earlier, are the oceans and lake sediments.

Summary Statement: Industrial effluents may contribute to the concentrations of zinc already present in municipal sewers or surface waters, and if treated, to industrial waste dump sites associated with the waste treatment sludge. Stream and lake sediments best reflect the extent of industrial contributions of zinc over time. Sludge generated from effluent treatment contains zinc primarily as the hydroxide and carbonate. Disposal in a properly designed hazardous waste dump should prevent further translocation of zinc.

d. Pathway #4 - POTW



Pathway #4 describes the fate of zinc in wastewaters which are introduced into a Publicly Owned Treatment Plant (POTW). The inflow to the POTW may consist of combinations of industrial and commercial effluents, domestic wastes and surface runoff. The nature of the influent is consequently quite varied, but typical influent concentrations will be about 1 ppm. Domestic wastes have been estimated to contribute about 31% of the zinc (Davis and Jacknow, 1975).

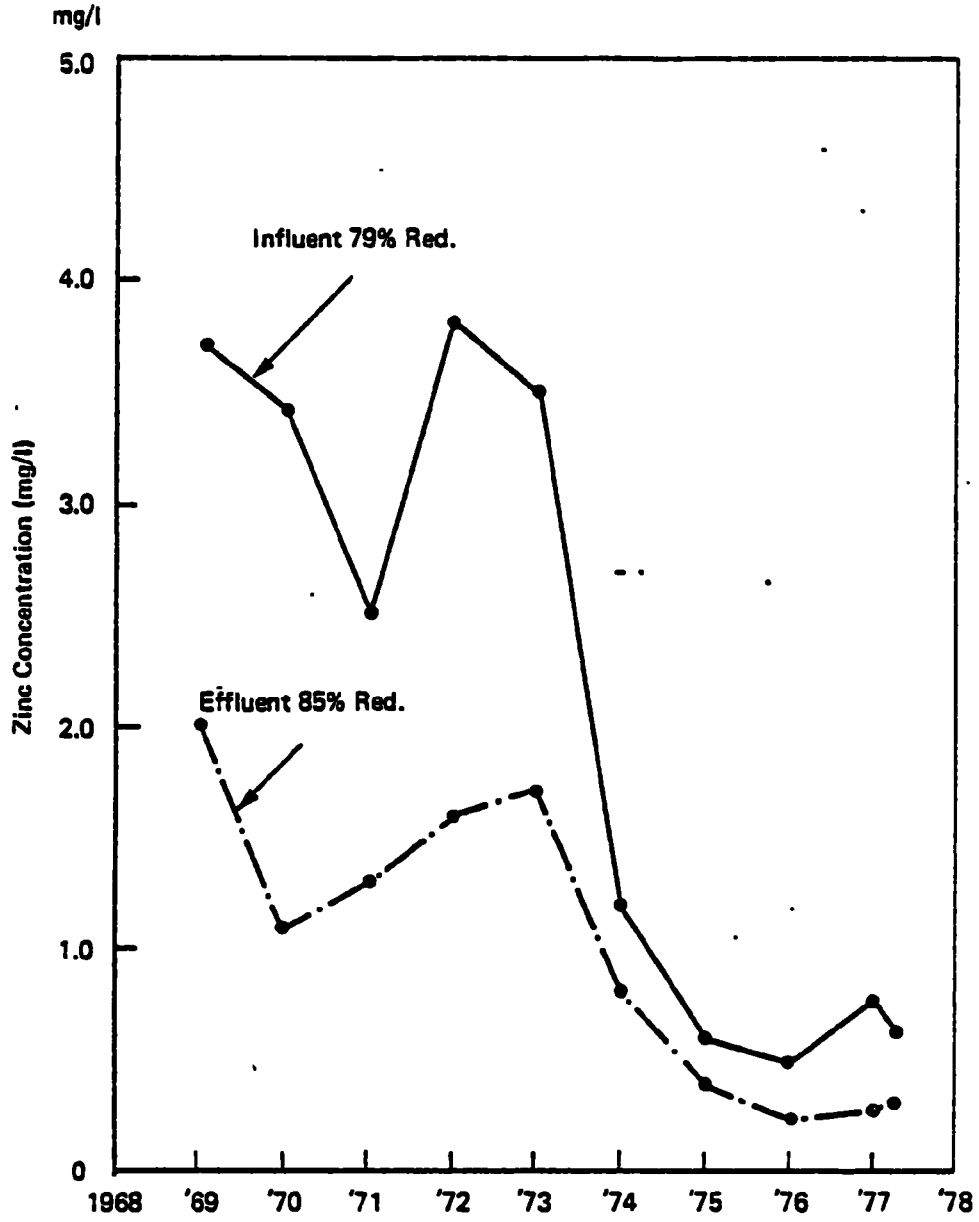
The degree to which zinc is removed from the raw wastewaters, and thus the concentration of zinc in the discharged wastewaters, depends on the type of treatment involved. One report provides a summary of data from various studies including 269 municipal treatment plants in the U.S. using various treatment methods (EPA, 1977). (See Section III.) The data for zinc are summarized below.

	Effluent Data (Means)	
Type of Treatment	% Removal of Zn (%)	Zn Concentration (mg/L)
Primary	31	0.55
Biological (all types)	52	0.28
Activated sludge	58	0.24
Trickling filter	46	0.32
Biological with chemical addition	72	Not Available
Tertiary	63	Not Available

The notion of concentration-dependent removal efficiency for zinc from POTW influents can be demonstrated from the data generated from an activated sludge treatment plant in Grand Rapids, Michigan (Beiner, 1978). In 1968, the metal platers and other industries were forced to pretreat their waste prior to discharge into the sewer. Before this ordinance, 46% of 3.7 ppm zinc in the influent was removed in the municipal treatment plants; after pretreatment enforcement, 63% of 0.78 ppm zinc was removed. Figure IV-15 illustrates the reduction of zinc concentrations in sewage, as well as the effects of inter-disciplinary environmental controls. The "hump" in Figure IV-15 was caused by the discharge into sewers of scrubber waters resulting from newly installed air pollution control devices in brass foundries. By 1973, suitable pretreatment methods were enacted which drastically reduced the concentration of zinc in sewage.

Zinc partitions into the sludge portion of the waste during treatment. The average concentration of zinc in digested sludges has been reported as 2420 ppm (survey of 100 plants) and 6380 ppm (survey of 80 plants) on a dry weight basis (NRC, 1979). As illustrated in the flow diagram above, disposal of the POTW effluent usually consists of discharge into surface waters or oceans. The sludge can be incinerated, dumped into ocean environments, or spread upon land.

Sludge which is disposed of on land may go to a sanitary landfill, or be spread for the purpose of amending the soil. The form of zinc in sludge is not known as revealed by a literature review conducted by Hoover (1978). Sommers et al. (1976) found that zinc sulfides, phosphates, and hydroxides were not detected in sludges containing relatively high concentrations of zinc. They did find a zinc-hydroxy-carbonate complex, and suggested that the chemistry of zinc in sludge is relatively complicated. The same study found that the movement of zinc in sludge-amended soils was unaffected by the soil, pH, or clay content.



Source: Beiner (1978)

FIGURE IV–15 TOTAL ZINC IN SEWAGE, GRAND RAPIDS, MICHIGAN

Minimal movement of heavy metals was observed in the top 7.5 cm of soil and no translocation was detected between 7.5 and 15 cm. Zinc specifically, was found in concentrations of <0.1 ppm in the soil leachate. However, the control sample also had the same concentration. The authors concluded that the application of sludge to soils does not enhance the solubility or movement of zinc.

The zinc in sewage sludges and other wastes disposed of in sanitary landfills may be more mobile than the case described above for soil application. Data on the mobility and concentrations of zinc in leachate from landfills accepting sewage sludges were not available for this study. Data from other landfills, however, do show zinc concentrations ranging from 0.01 to 240 mg/L, with 3 mg/L being a typical value. Zinc was considered to be a significant pollutant in leachate since the concentrations found were significantly higher than those found in nearby (unaffected) groundwaters (EPA, 1977). Sludge which is incinerated will contribute to the concentrations of zinc in the atmosphere. The fate processes will be similar to the same chain of events described in Pathway #1.

The behavior of zinc discharged with POTW effluents into local surface waters will be similar to that already described in other aqueous pathways. The fate of zinc discharged by the Joint Water Pollution Control Project (JWPCP) of the Los Angeles County Sanitation District has been studied in some detail (Morel et al., 1975), and may be generally representative of POTW discharges to the ocean.* While zinc in this study was likely to be found in the fairly insoluble sulfide form in the effluent (∿370 mgd effluent, discharged through submarine outfalls at a depth of 60 meters), the studies indicated that the combined processes of dilution and oxidation resulted in substantial solubilization of zinc (as well as other metals); this increases their residence time in the water and allows them to be transported greater distances where the effects would be

*The wastes, containing both domestic and industrial wastes, contain high levels of zinc (2400 ppm) after primary treatment.

negligible. It was estimated that only about one percent of the metals were deposited in the general area of the outfall. The sediments that do settle near the outfall are likely to be anoxic (Bertline and Goldberg, 1977) and zinc would, thus, be in the sulfide form.

Summary Statement: Wastewater treatment causes zinc to partition into the sludge, such that effluent concentrations range from 0.24-0.55 mg/l. Zinc mobility from sludge spread as a amendment to soil is unaffected by the soil, pH, or clay content. Most of the zinc remains in the top few centimeters of soil. Zinc associated with sludge disposed of in sanitary landfills, may be slightly more mobile, as determined by concentrations found in landfill leachate. Sludge incineration will promote atmospheric emissions of zinc oxide, the fate of which was covered in Pathway #1. Ocean disposal will contribute to slightly enriched sediments in the vicinity of the outfall in which the form of zinc will likely be the insoluble sulfide.

4. Biological Pathways

a. Zinc Uptake from Soils

Zinc Uptake via Aerial Deposition on Soil (Pathway #1) - Zinc uptake by terrestrial plants is a function of the form of zinc, as well as the properties of the soil and plant. The parameters which influence the translocation of zinc into plants are complex and integrated; reviews of these variables have been conducted by Cataldo and Wildung (1978), and NRC (1979).

Cataldo and Wildung (1978) found that the major factor governing the availability of heavy metals to terrestrial plants will be the solubility and thermodynamic activity of the uncomplexed ion. The form of zinc as a result of aerial deposition is primarily the insoluble oxide; dissociation is promoted by small particulate sizes, and low pH of the soil solution.

The soils and grasses near a lead smelting complex in Idaho illustrate the extent of metal uptake by biota in a contaminated area (Ragaini et al., 1977). The results of zinc uptake grasses grown in the top 2 cm of contaminated soils are tabulated below:

	Concentration of Zinc (ppm - dry weight)		
	Soil	Grass	Ave. Conc. in Grass / Ave. Conc. in Soil
Near Smelter	870-13,000	560-11,900	0.9
Background	804-940	420-810	0.7
Ave. Smelter Conc./ Ave. Background Conc.	8	10	

The results indicate that zinc is readily translocated from soil to grasses. The grasses accumulated zinc from contaminated soils as easily as background zinc was translocated.

Sewage Sludge - Zinc uptake from sewage sludge land dispoal may be substantial. Generally, if the pH of the sludge treated soil is maintained above 6.5, the mobility of zinc is limited (Council for Agricultural Science and Technology, 1976). The Council for Agricultural Science and Technology cites several examples of studies involving crop bioaccumulation from sludge amended soils. Giordano and Mays, as reviewed by the Council, found that zinc sulfate was more immediately available to string beans and sweet corn than zinc in sludge. In the long term, however, more zinc was bioaccumulated from the sludge-amended soils. Over time, Tinchtar et al. (Council of Agricultural Science, 1976) discovered that the concentration of zinc in coastal bermuda grass decreased by 50% within three years after sludge application was stopped. Further discussion of plant uptakes is contained in NRC (1979).

Purposeful Application of Zinc - Zinc is used as an agricultural crop and animal trace nutrient. It is applied to the soil as the hydrated sulfate or carbonate at concentrations of about 9-18 kg/acre as the sulfate (NRC, 1979). Approximately 40% of the zinc sulfate produced is used for agricultural purposes.

Zinc is also a component in two popular agricultural fungicides, Zineb and Ziram. Uptake from these uses would be more rapid than from sludge since it is applied in an inorganic form.

b. Bioaccumulation of Zinc in Aquatic Organisms

Bioaccumulation of zinc by both terrestrial and aquatic organisms has been extensively reviewed and the reader is directed to these studies for more detail (Versar, 1979a; NRC, 1979; Phillips and Russo, 1978).

Although zinc is accumulated in the food chain, it does not appear to be biomagnified (Versar, 1979a). Baptist and Lewis, as cited by Versar (1979a) found that the concentration of zinc decreased with each trophic level of a four-tier study. They found that uptake of zinc via water is

the more efficient pathway for zooplankton, while uptake of zinc by fish is more efficient via the food chain. Merlin et al., as cited by Phillips and Russo (1978) found that the diet of the fish determines how the zinc is accumulated. Fish fed synthetic diets containing zinc accumulated zinc much quicker than did fish fed snails containing similar levels. Deposit feeding organisms accumulate zinc more actively from biogenic carbonates (e.g., clamshells), than from other sediment-bound zinc. In turn, the rate of uptake of zinc bound to organic detritus is faster than from hydrous iron and manganese oxides (Versar, 1979a)

Zinc uptake by aquatic organisms is a function of water temperature, pH, salinity and hardness. Uptake increases with temperature due to temperature-related increases in metabolic rate (Phillips and Russo, 1978, NRC, 1979, Namminga and Wilhm, 1977). Zinc uptake and accumulation by aquatic plants increases with increasing pH. Zinc concentrations accumulated by algae were found to be twice as high at pH 8 as at pH 7, and six times as high at pH 9 (NRC, 1979). These data, and other studies, indicate a pH-dependent adsorption exchange by aquatic plants. Although zinc toxicity is inversely proportional to water hardness, uptake of zinc by fish increases as hardness increases. One reason may be that zinc precipitates in calcium-free waters on the mucus of fish body surfaces. Therefore, the zinc is released directly back into water.

Huggett et al. (as cited in NRC, 1979), determined that oysters accumulate more zinc as the salinity level of the water decreases. This correlation was observed for all rivers emptying into the Chesapeake Bay. A similar trend was noted for uptake of zinc by aquatic plants in the absence of "competing" ions. The presence of sodium, potassium, magnesium and calcium ions inhibits uptake of zinc. Competition for available bonding sites on the plants may explain this observation.

Depending on the organism, zinc may or may not be accumulated in one or more organs preferentially. Although the Atlantic oyster has the highest known bioconcentration factor (up to 100,000 times the water

concentration), zinc is not strongly localized in any particular organs or tissues (NRC, 1979). Mussels, on the other hand, tend to concentrate zinc mostly in the visceral mass (including the kidneys) and gonads, and least in the foot and shell. In scallops, zinc is concentrated in the kidneys (NRC, 1979), while in crustacenas the hepatopancreas normally has the highest levels. Most finfish concentrate zinc in the kidneys preferentially over other ograns, although Matthiesson and Brafield (1977) found that the stickleback accumulated zinc primarily in the gills. They also observed that zinc uptake increased with oxygen consumption, suggesting that the gills serve as an important site for zinc absorption in this species.

The biological half-life of zinc in aquatic and freshwater organisms appears to range from three days to 650 days for mollusks, crustaceans and fish (National Academy of Science, 1978). In all cases in which the half-life was determined as a function of temperature, the half-life decreased as the temperature increased. Loss mechanisms for zinc are excretion and molting.

Table IV-6 briefly summarizes the ranges of bioconcentration factors for fresh and saltwater organisms.

Zinc uptake by the biota residing in aqueous bodies which receive industrial and municipal discharges has been investigated. Mathis and Cummings (1973) studied the partitioning of zinc between the sediment, water and biota of the Illinois River. The Illinois River receives domestic and industrial wastes from Chicago and Peoria. Figure IV-16 demonstrates that the highest concentrations of zinc resided with the bottom sediments, closely followed by bioaccumulation by clams, tubificid worms, omnivorous fish, carnivorous fish, and water, respectively.

The results obtained by Namminga and Wilhm (1977) only partially support those of Mathis and Cummings (1973). The creek tested was the discharge basin for municipal and industrial wastes from an oil refinery. Zinc partitioned

Table IV-6

Bioaccumulation of Zinc by Aquatic Organisms

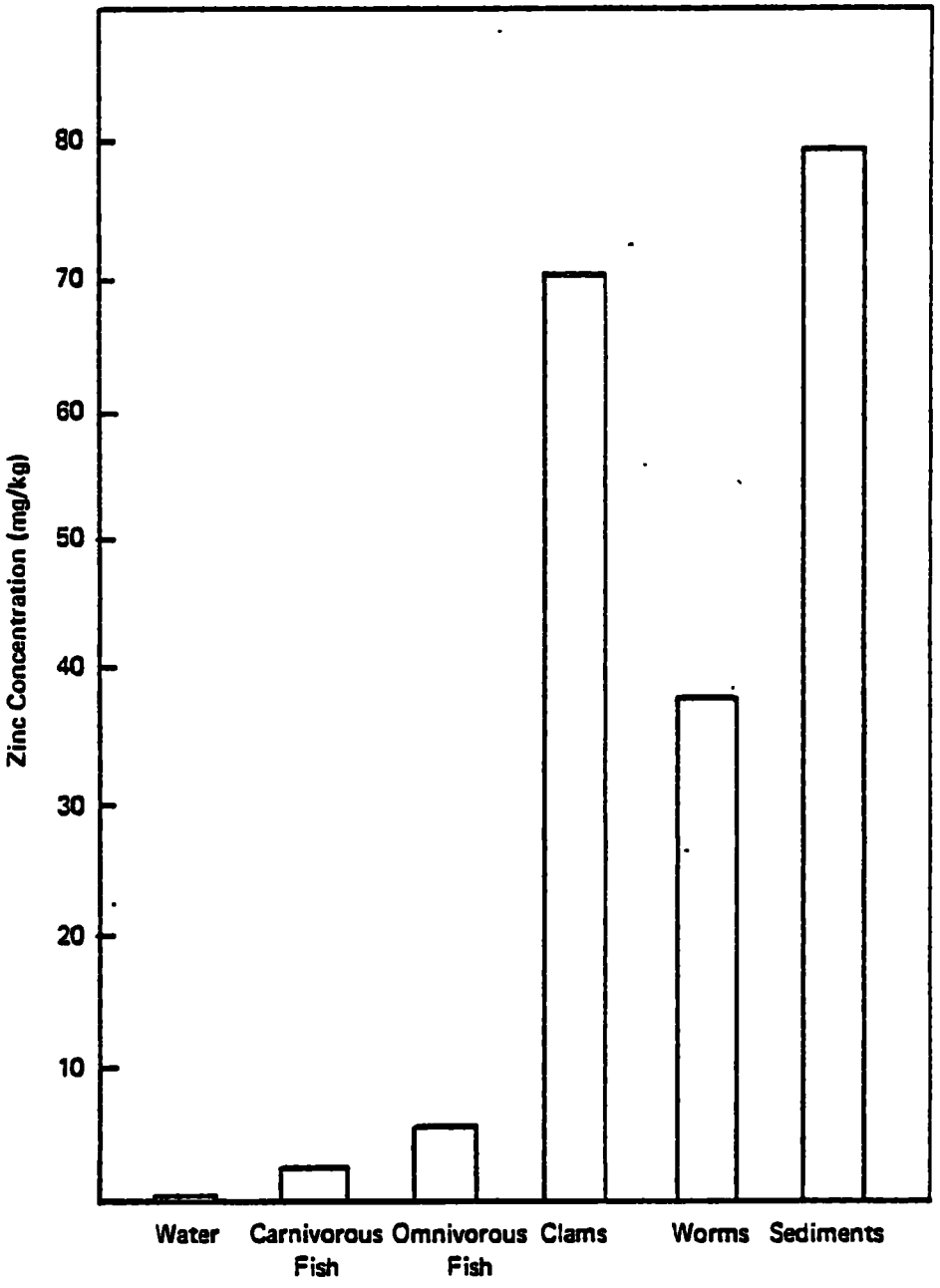
Seawater	Bioconcentration Factor	Concentration in Organisms (ppm - fresh wt.)
Algae	400 - 1,400 (b) 1 - 12,400 (a)	
Decapods	142 - 9,000 (a)	24.9 - 44.5 (b)
Mussels	130 - 310 (a)	
Oysters	450 - 27,000 (c)	100.9 - 271.01 (b)
Clams	43 - 500 (a)	8.0 - 22.5 (b)
Fish	11 - 373 (a)	2.98 - 23.85 (b)
Seaweeds	900 (c)	
Fresh Water		
Plants	4,000 (c)	
Invertebrates	10,000 - 40,000 (c)	
Insects	106 - 1,130 (a)	
Fish	8 - 12 (a)	

References

a. EPA (1979)

b. NRC (1979)

c. Versar (1979a)



Source: Mathis and Cummings (1973).

FIGURE IV–16 PARTITIONING IN BIOTA, SEDIMENTS, AND WATER

as follows: water column, 0.010 mg/K; sediments, 9.2 mg/kg; chironomides (midge larvae), 57.5 mg/kg. The authors hypothesize that zinc accumulates in chironomids primarily due to surface adsorption and cation exchange. The low bioconcentration factor for zinc compared to sediments may be due to frequent molting by the chironomids, thereby eliminating the adsorbed metal.

Zinc bioconcentration in lake waters has been reviewed by McIntosh and Bishop (1978). The history of Little Center Lake was discussed in Pathway #3. The extent of accumulation by fish and periphyton (sessile community of organisms) is summarized as follows:

	Average Zinc Concentration (ppm)	Concentration Factor
Water	.044	
Sediments (0.5 cm)	7,530	171,000
Periphyton	1,230	28,000
Bluegill (Lepomis macrochirus)	180	4,100
Pumpkinseed (Lepomis gibbosus)	142	3,200

These numbers demonstrate that the largest accumulators of zinc, after the sediments, are the periphyton community. Both fish species concentrated similar levels of zinc which were one order of magnitude less than the periphyton, and two orders less than the sediments.

The impact of zinc leached from sewage sludge by seawater of a tropical ecosystem has been studied by Montgomery and Price (1979). The study investigated the uptake of zinc, in addition to other metals, by the inhabitants of a turtle grass mangrove ecosystem. The net loss of zinc from the sewage sludge was paralleled by a net increase of zinc uptake by the "fouling" organisms. Net uptake of zinc was reported for

turtle grass (Thalassia testudinum), an urchin which grazes on turtle grass (Lytechinus variegatus), the sea cucumber (Holothuria mexicana), and the roots of the red mangrove (Rhizophora mangle). After 125 days, the turtle grass leaves contained about 3 µg/g zinc, dry weight; the grazer contained about 17 µg/g. The mean concentration of zinc in the seawater was 20.1 µg/K.

c. Summary Statement

Biological uptake of zinc from soils appears to occur under many different conditions. The rate of uptake is likely to be faster for the more soluble forms of zinc (e.g., $ZnSO_4$) than for zinc encountered as the oxide or in association with sewage sludge. However, all studies report substantial uptake by zinc by terrestrial plants over time.

Uptake by aquatic organisms proceeds via ingestion and sorption. It is unclear which mechanism dominates for fish. Uptake is enhanced by increased temperature, pH and water hardness. Two examples were cited in which uptake decreased as a result of increased salinity and "competing ions."

Bioconcentration is greatest for algae and benthic feeders. No evidence of biomagnification has been observed. The biological half-life of zinc is inversely related to temperature. Loss mechanisms are excretion and molting.

C. REFERENCES

Beiner, J.A. and W.H. Bourma. 1978. Case History City of Grand Rapids, Michigan Program of Industrial Waste Control. In: Pretreatment of Industrial Wastes - Joint Municipal and Industrial Seminar. A.S. Vernick H.D. Feiler, P.D. Lanik, eds. EPA Seminar Handout.

Bertine, K.K, and E.D. Goldberg. 1977. History of heavy metal pollution in Southern California coastal zone-reprise. Environ. Sci. Technol., 11(3):297-299.

Bower, P.M., H.J. Simpson, S.C. Williams, and Y.H. Li. 1978. Heavy metals in the sediments of foundry cove, Cold Spring, New York. Environ. Sci. Technol., 12(6):683-687.

Cataldo, D.A. and R.E. Wilding. 1978. Soil and plant factors influencing the accumulation of heavy metals by plants. Environ. Health Perspectives, 27:149-159.

Christensen, F.R., J. Scherfig, and M. Koide. 1979. Metals from urban runoff in dated sediments of a very shallow estuary. Environ. Sci. Technol., 12(10):1168-1173.

Coles, D.G., R.C. Ragaini, J.H. Ondor, G.L. Fisher, D. Silberman, and B.A. Prentice. 1979. chemical studies of stack fly ash from a coal-fired power plant. Environ. Sci. Technol., 13(4):455-459.

Council for Agricultural Science and Technology. 1976. Application of Sewage Sludge to Cropland: Appraisal and Potential Hazards of the Heavy Metals to Plants and Animals. Prepared for U.S. EPA, Office of Water Program Operations, EPA-430/9-76-013.

Dana, J.A. and J.O. Leckie. 1978. Effects of absorbed complexing ligands on trace metals uptake by hydrous oxides. Environ. Sci. Technol., 12(12): 1309-1315.

Davis, J. and J. Jacknow. 1975. Heavy metals in wastewater in three urban areas. JWPCF, 47(9):2292.

Gibb, J.P. 1976. Field Verification of Hazardous Industrial Waste Migration from Land Disposal Sites, Proceedings of the Hazardous Waste Research Symposium, EPA 600/9-76-015, U.S. EPA, Cincinnati, Ohio.

Hamilton-Taylor, J. 1979. Enrichments of zinc, lead, and copper - recent sediments of Windermere, England. Environ. Sci. Technol., 13(6):693-697.

Hem, J.D. 1972. Chemistry and occurrence of cadmium and zinc in surface water and groundwater. Water Resources Research, 8(3):661-679.

Holcombe, L.A. 1977. Adsorption and Desorption in Mine Drainages. NTIS, #PB-290 614/7WP.

Hoover, T.B. 1978. Inorganic Species in Water: Ecological Significance and Analytical Needs (A Literature Review). U.S. EPA 600/3-78-064, Environmental Research Lab, Athens, Georgia.

Huang, C.P., H.A. Elliott, and R.M. Ashmead. 1977. Interfacial reactions and the fate of heavy metals in soil-water systems. J. Water Pollution Control Fed., 49(5):745-756.

Jacko, R.B., D.W. Nevendorf, and K.J. Yost. 1975. Trace Metal Emissions from a Scrubber Controlled Municipal Incinerator. ASME paper #N75-WA/APC-2.

Jennett, J.C. and S.M. Linnemann. 1977. Disposal of lead and zinc containing wastes on soils. J. Water Pollution Control Fed., 49(8): 1842-1856.

Martin, H.W. and W.R. Mills, Jr. 1976. Water Pollution Caused by Inactive Ore and Mineral Mines - A National Assessment. NTIS #PB-264-956, prepared for the Office of Research and Development, U.S. EPA, Cincinnati, Ohio.

Mathis, E., B.J. and T.F. Cummings. 1973. Selected metals in sediments, water, and biota in the Illinois River. J. Water Pollut. Control Fed., 45(7):1573-1583.

McIntosh, A. and W. Bishop. 1976. Distribution and Effects of Heavy Metals - A Contaminated Lake. Purdue University Water Resources Research Center, #85.

Mezey, E.J., S. Singh, and D.W. Hissong. 1976. Fuel Contaminants, Volume 1: Chemistry. Report by Battelle Columbus Laboratories to U.S. EPA, EPA-600/2-76-177a.

Mink, L.L., R.E. Williams, and A.T. Wallace. 1972. Effect of early day mining operations on present day water quality. Groundwater, 10(1):17-26.

Montgomery, J.R. and N.T. Price. 1979. Release of trace metals by sewage sludge and the subsequent uptake by members of the turtle grass mangrove ecosystem. Environ. Sci Technol., 13(5):546-549.

Morel, F.M.M., J.C. Westall, C.R. O'Melia, and J.J. Morgan. 1975. Fate of trace metals in Los Angeles County wastewater discharge. Environ. Sci. Technol., 9(8):756-761.

Namminga, H. and J. Wilhm. 1977. Heavy metals in water, sediments, and chironomids. J. Water Pollution Control Fed., 49(7):1725-1731.

National Research Council. 1979. Zinc. Prepared by the Subcommittee on Zinc, Committee on Medical and Biological Effects of Environmental Pollutants. National Academy of Sciences, Washington, D.C.

Perhac, R.M. 1974. Water Transport of Heavy Metals in Solution and by Different Sizes of Particulate Solids. NTIS #PB-232 427.

Phillips, E., G.R. and R.C. Russo. 1978. Metal Bioaccumulation in Fishes and Aquatic Invertebrates: A Literature Review. EPA-600/3-78-103, U.S. EPA, Office of Research and Development.

Ragaini, R.C., H.R. Ralston, and N. Roberts. 1977. Environmental trace metal contamination in Kellogg, Idaho, near a lead smelting complex. Environ. Sci. Technol., 11(8):773-784.

Schell, W.R. and A. Nevissi. 1977. Heavy metals from waste disposal in central Puget Sound. Environ. Sci. Technol., 11(9):887-893.

Sommers, L.E., D.W. Nelson, R.E. Terry, and D.J. Silviera. 1976. Nitrogen and Metal Contamination of Natural Waters from Sewage Sludge Disposal on Land. Purdue University, Resources Research Center.

Theis, T.L. and R.O. Richter. 1979. Chemical speciations of heavy metals in power plants ash pond leachate. Environ. Sci. Technol., 13(2): 219-228.

U.S. Environmental Protection Agency. 1977. Information for proposed general pretreatment regulations. 40 CFR 405, Washington, D.C.

U.S. Environmental Protection Agency. 1977. Report of Pollution-Caused Fish Kill. Office of Water Planning and Standards, EPA Form 7500-8.

U.S. Environmental Protection Agency. 1977. The Prevalences of Subsurface Migrations of Hazardous Chemical Substances at Selected Industrial Waste Dispoal Sites.

U.S. Environmental Protection Agency. 1979. Ambient Water Quality Criteria: Zinc.

Versar, Inc. 1979a. Statement of Probable Fate. Draft report to the Monitoring and Data Support Division, EPA.

Versar, Inc. 1979b. Non-Aquatic Fate of Zinc.

Williams, L.G., J.C. Joyce, and S.T. Monk, Jr. 1973. Stream-velocity effects on the heavy metal concentrations. J. Am. Water Works Assoc., pp. 275-279.

Vuceta, J and J.J. Morgan. 1978. Chemical modeling of trace metals in fresh waters: Role of complexation and adsorption. Environ. Sci. Technol., 12(12):1302-1308.

Yost, K.J. and D.R. Masarik. 1977. A Study of Chemical Destruct Waste Treatment Systems in the Electroplating Industry: Plating and Surface Finishing. pp. 35-40.

V. EFFECTS OF ZINC

A. HUMAN TOXICITY

1. Introduction

Zinc is an essential trace element in human and animal nutrition and is present in virtually all mammalian tissues and biological fluids. Estimates of total body zinc for a hypothetical 70 kg man range from 1.4 to 2.3 grams (Burch et al., 1975). The major amount (~90%) of body zinc resides in muscle and bone but the highest concentrations are found in tissues of the eye and the male reproductive tract (NAS, 1978).

Zinc has two known biological functions: (1) as a necessary component of certain metalloenzymes (e.g., carbonic anhydrase, alkaline phosphatase, alcohol dehydrogenase) and (2) for the optimal synthesis of proteins, nucleic acids and lipids. Dietary requirements for zinc are thus related to the needs for growth, tissue repair and compensation for excretion.

Zinc must be supplied in the diet more or less continuously since it is also excreted at a comparable rate. The recommended human dietary allowances for zinc are as follows:

Age	Zinc (mg/day)
0-6 mo	3
6 mo-1 yr	5
1 yr-10 yr	10
adult	15
pregnant female	20
lactating female	25

Many dietary factors influence the utilization of zinc present in food. The formation of insoluble complexes with calcium and phyta e in the alkaline intestinal environment has been shown to markedly decrease the availability of zinc for intestinal absorption by experimental animals. Dietary fiber has also been shown to decrease the availability of zinc for intestinal absorption by man (Reinhold et al., 1976).

Zinc status is also affected by infections, pregnancy, surgical procedures or stressful situations such as myocardial infarction, etc. (Lindeman et al., 1972; Burch et al., 1975).

a. Zinc Deficiency

Due to the essential nature of zinc to human health, attention has focused on marginal or deficient zinc intake rather than zinc toxicity. Growth is impaired in the young of all species in which zinc deficiency has been observed (Horvath, 1976). Insufficient zinc in the diet also results in inhibition of sexual maturation, loss of appetite, inability to gain weight, skeletal abnormalities (bowing of the legs, joint stiffness), parakeratotic esophageal and skin lesions and hair loss (Burch et al., 1975). Mature animals often display no signs of deficiency other than poor appetite and decreased growth (NAS, 1978).

Material zinc deficiency is known to cause marked fetal abnormalities in experimental animals (Hurley and Swenerton, 1966; Hurley et al., 1971). The evidence available at present suggests that congenital malformations in zinc-deficient embryos may be brought about by impaired synthesis of nucleic acids. Swenerton et al. (1969) found DNA synthesis as measured by uptake of tritiated thymidine was much lower than normal in zinc-deficient rat embryos at 12 days of gestation.

Prasad and Oberleas (1974) have shown that the activity of thymidine kinase, an enzyme regarded as essential for DNA synthesis and cell division, is adversely affected in rapidly regenerating connective tissue. In rats, this effect can be demonstrated as early as six days following the institution of a Zn-deficient diet.

A probable human counterpart to animal zinc deficiency has been observed in individuals living in Iran and Egypt and subsisting on diets consisting largely of bread and beans and nearly devoid of animal protein (Halstead et al., 1972; Prasad et al., 1963ab). These individuals have decreased zinc concentrations in plasma, red blood cells and hair

and exhibit short stature owing to retarded growth, testicular atrophy with hypogonadism and geophagia. Their status improves following oral supplementation with zinc for several months.

Since food consumption is also reduced in a zinc deficient state, some of the effects of low dietary zinc may be secondary to decreased food consumption. A more detailed discussion of the various manifestations of zinc deficiency in both man and animals may be found in Burch and Sullivan (1976), F.C.T. (1972), NAS (1978), and Vallee (1959).

2. Metabolism and Bioaccumulation

The average adult ingests 10 to 15 mg of zinc daily. Absorption is rapid but incomplete (about 30%) (Prasad, 1979; Burch et al., 1975). Absorption of zinc seems to take place in the proximal part of the small intestine but the exact mechanism or the sites of absorption in man is not clear. Absorption appears to occur at other portions of the small and large intestine as well (Prasad, 1979; Methfessel and Spencer, 1973).

Zinc is transported throughout the body in the blood, bound mainly to serum proteins (Prasad and Oberleas, 1970; Parisi and Vallee, 1970; Suso and Edwards, 1971; Frazier, 1979).

Orally administered ^{65}Zn appears in the blood of human subjects within 15-20 minutes after ingestion; plasma levels peak 2-4 hours later and decline rapidly thereafter as tissue uptake occurs (NAS, 1978). The biological half-life of ^{65}ZN in man has been calculated to be between 154 and 334 days (Spencer et al., 1965; Richmond et al., 1962; Andrasi and Feher, 1967).

Absorbed zinc is excreted in urine, sweat, semen, hair, nails and desquamated skin (Jameson, 1976). Zinc in feces represents the major zinc loss from the body. Roughly 70-80% of ingested but unabsorbed zinc is found in the stool (~5-10 mg daily) (Tsuchiya and Iwao, 1978; NAS, 1978).

Urinary loss (300-700 µg/24 hr) represents only a small fraction of the daily oral intake, but is fairly constant for a given adult (Burch et al., 1975; Goolamali and Comaish, 1975).

Prasad et al. (1963b) have estimated that 1.15 mg of zinc could be lost per liter of sweat; in hot climates, five or more liters of sweat may be lost per day.

For women of child-bearing age, zinc losses in menstrual fluid are estimated to represent an additional 300 to 600 µg of zinc per menses (Schroeder et al., 1967).

Most of the zinc present in the body is in a state of constant movement with the rate of accumulation and turnover varying greatly from tissue to tissue. For example, zinc is incorporated into the skeleton slowly but is firmly bound for long periods while zinc entering the hair is lost as the hair is shed. The most rapid accumulation and turnover of zinc occurs in liver, spleen and kidneys (NAS, 1978).

Mean serum zinc concentrations in healthy men and women are in the range of 100 µg/dl but drop in pregnant women (97 µg/dl) and in healthy, non-pregnant females taking oral contraceptives (81 µg/dl) (Halsted and Smith, 1970). Patients with liver cirrhosis frequently possess low serum zinc levels and paradoxically, may excrete several milligrams of zinc in their urine each day (Burch et al., 1975). Low serum zinc is also found in patients with uremia, kwashiorkor and sickle-cell anemia (Zazgornik et al., 1971; Smit and Pretorius, 1964; Prasad et al., 1975).

Zinc is also present in human milk but the concentration varies with time after delivery. Zinc concentration in colostrum is 3 to 6 times higher than that present in milk one week after parturition (3-4 mg/l) and gradually falls off over the next 6 months (Prasad, 1966b; NAS, 1978).

Human semen contains 10-35 mg Zn/100 dl; a positive correlation seems to exist between the number of spermatozoa and the level of zinc (NAS, 1978).

Zinc is also present in all human tissues with the highest concentrations present in the choroid (419-562 ppm) of the eye (Galin, 1962), and prostate (850 µg/g) (Underwood, 1971).

Additional information on the metabolism, storage and excretion of zinc by man and experimental animals can be found in the Appendix. None of this information has proved appropriate for inclusion in our risk assessment calculations.

3. Animal Studies

a. Carcinogenesis

(1) Oral

Reports on the effects of dietary zinc on carcinogensis and tumor growth have been varied. Davies et al. (1968) reported decreased levels of serum zinc in patients with cancer of the bronchus and colon, but not in other forms of cancer. Zinc is required for DNA synthesis (Swenerton et al., 1969; Sandstead and Rinaldi, 1969), and since tumors often have a high rate of DNA synthesis, these decreased serum Shiraishi et al. (1969) found that serum zinc decreased in tumor-bearing rats according to the time elapsed since tumor transplantation (i.e., serum zinc values were 166, 153, and 99.8 µg/dl at 10, 20 and 30 days post-transplant, respectively, compared to 143 µg/dl for controls.

On the other hand, increased zinc concentrations, although highly variable from patient to patient, have been found in cancerous lung and breast tissue (Mulay et al., 1971) and in gastric juice of gastric cancer patients (Matsumoto et al., 1969).

Several workers have demonstrated reduced growth in a number of transplanted tumors (Walker 256, Lewis lung, L1210, P388) as well as prolonged survival in rats and mice receiving inadequate dietary zinc (McQuitty et al., 1970; DeWys et al., 1970; DeWys and Pories, 1972; Petering et al., 1967; Poswillo and Cohen, 1971). These studies suggest that tumor inhibition is a general effect of zinc deficiency irrespective of cell type, cell growth rate or site of growth. The results, however, apply only to the rate of growth of tumor cells and not to the process of carcinogenesis.

Elevated levels of dietary zinc may also inhibit tumor growth. Duncan et al. (1974) examined the effect of different levels of zinc (0.4-2500 µg Zn/g ration) on the growth of a transplanted hepatoma (originally induced by 3'-methyl-4-dimethylaminoazobenzene) in female Wistar rats. Growth of the transplanted hepatoma was significantly ($p < 0.1$) reduced in rats maintained on diets either deficient (0.4 µg/g) or high in zinc ($\geq$ 500 µg/g) when compared to control animals (60 µg/g). Greater inhibition of hepatoma growth was not achieved at toxic levels (2500 µg/g) of Zn intake.

Ciapparelli et al. (1972) examined the effects of the administration of drinking water containing 0, 50, 100 or 250 ppm of zinc on DMBA (9, 10-dimethyl-1, 2-benzanthracene)-induced-submandibular salivary gland tumors in 4-5 mo-old Wistar strain albino rats. Zinc concentration in the solid diet was 79 ppm. Tumor growth was retarded during the 3 1/2 month study in the 250 ppm zinc group. Unfortunately, the daily zinc intake for individual animals from solid and liquid sources was not determined. Poswillo and Cohen (1971) have also reported that addition of 100 ppm $ZnSO_4$ to the drinking water of hamsters inhibited the formation of DMBA-induced tumors in the hamster cheek pouch. At 6 months, the tumor incidence in Zn-DMBA-treated hamsters was 11% compared to 87% in hamsters treated with DMBA alone.

On the other hand, increased levels of dietary zinc (500 ppm) did not appear to affect the growth of a grafted Walker 256 carcinosarcoma (a rapidly dividing solid tumor) in rats (McQuitty et al., 1970).

Aside from an increased number of hepatomas above control (see Table V-1) which were not considered to be significantly different from control, the incidences of malignant lymphomas and pulmonary adenomata were comparable to controls in Chester Beatty mice given 5000 ppm Zn (as zinc oleate) in the diet for 3 months followed by 2500 ppm for 3 months and then 1250 ppm zinc for an additional 21 weeks. The dietary concentrations of zinc were reduced due to mortality and severe anemia. Addition of zinc sulfate to drinking water at 5000 and 1000 ppm for the same period of time did not increase the incidence of tumors (Walters and Roe, 1965).

Table V-1

Hepatomas Resulting from Zinc in the Diet of Mice

	Hepatomas		Malignant Lymphomas		Lung Adenomas	
Control	3/24	(12.5%)	3/24	(12.5%)	10/24	(41.1%)
Zn Oleate (diet)	7/23	(30.4%)	2/23	(8.7%)	9/23	(39.1%)
5000 ppm $ZnSO_4$ (water)	3/22	(13.6%)	2/22	(9.1%)	5/22	(22.7%)
1000 ppm $ZnSO_4$ (water)	3/28	(10.7%)	4/28	(14.3%)	9/28	(32.1%)

(2) Intratesticular

Zinc has been shown to induce testicular teratomas in hamsters, rats and roosters when injected directly into the testes (Guthrie and Guthrie, 1974; Riviefe et al., 1966; Guthrie, 1967; Carleton et al., 1953). These tumors developed in only a small percentage of the test animals but spontaneously occurring testicular teratomas are rare.

Injection of 2 mg $ZnCL_2$ into the testes of 49 two-month-old Syrian hamsters during the period of rapid seasonal gonadal growth produced two embryonal carcinomas of the testis (4%) at termination of the study

10 weeks later (Guthrie and Guthrie, 1974). Similarly, injection of 1.25-5 mg $ZnCl_2$ into one or both testis produced six interstitial-cell tumors and one seminoma in ninety 4-8 mo-old Wistar rats by 28 months. A second group of 15 rats injected with 2.5 mg $ZnCl_2$ plus 200 units of gonadotropin developed two interstitial-cell tumors and one malignant teratoma within 24 months (Riviére et al., 1960).

In the cock testes, teratomata developed only if zinc was administered during periods of normally high sexual activity (January-March) or following artificially induced sexual activity by injection of gonadotropins.

Guthrie (1967) produced teratomas within 2-4 months in five of forty-six (10.8%) White Leghorn cockerels injected intratesticularly with 10 mg of $ZnCl_2$ during the period of rapid testicular growth. No tumors (0/45) were found in cockerels injected with 0.2 ml of 1 N hydrochloric acid suggesting that the production of partial necrosis of the testis and the resulting gonadotropic-stimulation do not of themselves produce teratomas.

Similar data were reported by Carleton and co-workers (1953) who noted a tumor incidence of 25.6% (11/43) 3 to 9 months after injection of 10-25 mg $ZnCl_2$ into the testes of 18-month-old White Leghorn roosters.

Except for the ability of zinc to induce testicular teratomas, no experimental evidence exists to suggest that ingestion or parenteral administration of zinc is tumorigenic. Indeed, several studies indicate that the administration of zinc may inhibit the growth of tumor cells. Its effect on the process of carcinogenesis is unclear. The significance of the teratomata following direct injection into the testes during periods of high or artificially induced sexual activity to human risk from ingestion of zinc is questionable.

b. Mutagenesis

No literature was found to suggest that zinc is mutagenic in mammalian or bacterial systems. Van Rosen (1954) did report that an aqueous zinc solution (concentration not stated) had weak chromosome-breaking activity in Pisum rootlets and Herick (1969) noted disturbances in the differentiation of the chromosomes in the broad bean, Vicia fava, exposed to a 0.1% aqueous solution of zinc sulfate.

c. Adverse Reproductive Effects

Little is known concerning the effects of excessive zinc on reproduction in general and embryonic development in particular. Sprague Dawley rats fed 0.4% Zn, as the oxide, in the diet for 37 days continuously beginning 21 days before breeding through day 15 or 16 of pregnancy resorbed all fetuses. Other rats fed the same diet from day 0 to day 15 or 16 of pregnancy produced fetuses of normal appearance but of a smaller body weight than controls. A variable degree (4-29%) of resorption was noted in this group as well. Groups of female rats treated according to both protocols but at 0.2% Zn produced normal fetuses with no effects noted on fetal weight or the degree of resorptions (Schlicker and Cox, 1967, 1968).

In another study, pregnant rats were fed marginal (10%) protein diets (30 ppm Zn) supplemented with a much lower level of zinc (150 ppm) as zinc sulfate. Dams were killed on day 18 of pregnancy. The 13 Zn-treated dams exhibited a higher rate of fetal resorption (9.4%) than the 12 controls (1.9%) fed the basal diet containing 30 ppm Zn. Eight of the 13 dams given excess zinc in the diet had at least one resorption each compared to two of 12 control dams ($p < 0.05$). The actual requirement of zinc during pregnancy in experimental animals is not well established but it appears that a high rate of resorption can occur with moderately high levels of zinc in the diet (Kumar, 1976).

Hedges et al. (1976) saw no adverse effects on reproductive performance in swine fed normal zinc levels (33-83 ppm) during gestation and lactation.

Progeny from sows fed 33 ppm zinc did not appear to have the necessary body stores of zinc for optimum growth, however.

Ferm and Carpenter (1967) provoked a dubious response in a small group of golden hamsters given 2 to 6 mg/kg $ZnSO_4 .7\ H_2O$ intravenously on day 8 of gestation:

Treatment	No. of Dams	No. Embryos Recovered	No. Embryos Resorbed	No. Embryos Malformed
Control	10	116	7	1 (0.8%)
2-6 mg $ZnSO_4 .7\ H_2O$	12	142	4	2 (1.6%)

In a later study Ferm and Carpenter (1968) observed a 4.6% incidence of abnormal embryos (3/65) and 2.8% incidence of resorbed fetuses (2/70) in embryos from 6 hamsters given 2 mg/kg $ZnSO_4 .7\ H_2O$ intravenously on day 8 of gestation. No controls were run. The authors further reported that intravenous doses of 10-25 mg/kg were well tolerated by the dams but induced a fetal resorption rate of 12%. At higher dosage levels (not given but 30 mg/kg was lethal to dams), a few gross malformations (~6%) consisting of exencephaly and rib fusions were seen but no consistent pattern of malformations was discernible. Ferm and Carpenter (1968) have also shown that zinc exerts a remarkable protective effect against cadmium-induced teratogenesis in hamsters provided zinc is administered within 12 hours of cadmium administration.

Thus, there is some evidence to suggest that excessive dietary intake of zinc (0.4% of the diet) prior to conception and throughout gestation or intravenous administration of high doses of zinc (6 mg/kg) during gestation may be linked to increased fetal resorption in rats and hamsters. Confirmational studies should be conducted. However, normal to moderately elevated dietary zinc intakes were neither embryotoxic nor teratogenic in either rats (0.2% diet) or swine (83 ppm).

d. Other Toxicological Effects

Zinc toxicity is an uncommon natural event in mammalian species. The low toxicity of zinc is probably attributable to the homeostatic mechanisms which control its absorption, tissue uptake and excretion. Experimental animals, particularly rats, have tolerated up to 100 times the normal dietary level of zinc without signs of toxicosis (NAS, 1978).

Heller and Burke (1927) fed young rats a basal diet supplemented with 0.25% Zn for 3 generations. No adverse effects were noted. Later studies by Sutton and Nelson (1937), Sadasivan (1951) and Walters and Roe (1965) establish 0.5% zinc in the diet (5000 ppm) to be a toxic dietary level in young rats. Walters and Roe observed severe anemia in mice fed 5000 ppm Zn as zinc sulfate for 3 months while Sutton and Nelson (1937) reported hypochromic microcytic anemia in rats fed 0.5% zinc in the diet for 39 weeks, and Sadasivan noted reduced body weight gain, reduced fat content of the liver, skeletal disorders (lowered calcium/phosphorus ratio in femur) and changes in various clinical chemistry parameters in rats given 0.5% zinc in the diet.

Drinker and co-workers (1927b) reported that the addition of up to 34.4 mg zinc/day to the diet of rats for 35-53 weeks produced no toxicity. They reported similar findings for cats and dogs (Drinker et al., 1927c).

Male cats fed a diet containing 250 to 300 mg of zinc oxide (200-240 mg Zn) for 12 to 16 weeks exhibited marked fibrotic changes in the pancreas, a decrease of 50% in pancreas size and a 25% drop of initial body weight values when compared to control animals (Scott and Fischer, 1938).

No ill effects other than slight growth retardation were noted in male albino rats injected intraperitoneally 6 days per week with 780 µg Zn/kg as zinc sulfate for 66 doses. No growth retardation was seen, however, in rats given 1560 µg Zn/kg for 33 doses. Growth retardation appeared to be dependent on the period of treatment rather than dose (Caujolle et al., 1969).

With respect to acute zinc toxicity, oral LD_{50} values for $ZnCl_2$ range from 200 mg/kg (96 mg Zn/kg) in guinea pigs to 358 mg/kg (168 mg Zn/kg) in the mouse (RTECS, 1977). For zinc sulfate, oral LD_{50} values range from 626 mg/kg (254 mg Zn/kg) in mice to 1396 mg/kg (565 mg Zn/kg) in rats (Caujolle et al., 1969). Acute toxicity is increased when zinc is administered by subcutaneous, intraperitoneal or intravenous routes; representative values are presented in the Appendix. The toxicity of various zinc salts in experimental animals were reviewed in detail by Van Reen (1966) and NAS (1978).

e. Zinc-Cadmium-Copper Interactions

The toxic effects of metals are often complicated by mutual biological antagonism of one metal with another at some functional site. For example, one metal may induce a biological effect by altering the requirements for another metal through competition for the same biochemical sites. The site of competitive interaction of zinc with copper appears to be at the point of absorption, cadmium competes with zinc at cellular binding sites (Task Group on Metal Interaction, 1978).

Studies with experimental animals have shown that cadmium may alter the metabolism of zinc and interfere with a number of zinc-dependent enzymes (NAS, 1978). Zinc, on the other hand, counteracts a number of toxic effects of cadmium. Simultaneous subcutaneous administration of cadmium and zinc protected against severe testicular injury seen in rats given cadmium alone (Elinder and Piscator, 1978; Webb, 1971). Another interaction involves the reduction of cadmium-induced elevation of blood pressure in rats by injections of zinc chelate (Elinder and Piscator, 1978).

A complete discussion of these complex interactions is beyond the scope of this report but has been reviewed in detail by the Task Group on Metal Interactions, 1978; the National Academy of Science, 1978; Elinder and Piscator, 1978; and Nordberg, 1976. There are no available data on the relationship between intakes of zinc, copper and iron and the effects

of cadmium in human populations (Task Group on Metal Interaction, 1978) but there is no question that the toxic effects of zinc are modified to some extent in the presence of other metals.

4. Human Studies

Controlled studies of zinc tolerance as well as toxicosis in humans are sparse. In man, zinc toxicity may occur by 3 routes: ingestion of toxic amounts of zinc, direct skin contact with zinc or zinc salts, or inhalation of fairly high concentrations of freshly formed zinc oxide fumes.

a. Oral

Pécoud et al. (1975) reported that five of six fasting subjects who took 50 mg zinc as zinc sulfate complained of gastric discomfort for a period of 30 to 60 minutes but no other adverse effects were seen. Gastric distress was not reported, however, following ingestion of 25 mg zinc under the same conditions.

In man, ingestion of 1-2 grams $ZnSO_4 .7\ H_2O$ (225-450 mg Zn) results in immediate emesis. Ingestion of zinc in excess of this amount produces nausea, vomiting and diarrhea. Thus, the emetic property of zinc salts results in the rapid removal of a large portion of the ingested zinc before it can be absorbed.

The available data suggest that in most individuals, 150 mg Zn may be ingested on a daily basis without adverse effect. Numerous studies have reported no effect to minimal gastric discomfort and mild diarrhea in humans given 660 mg zinc sulfate (150 mg Zn) per day in 3 divided oral doses for periods up to 6 months (Marshak and Marshak, 1973; Pullen, 1970; Greaves and Skillen, 1970; Flynn et al., 1973; and Czerwinski et al., 1974).

Zinc therapy has been utilized as an adjunct to wound healing in patients with chronic venous leg ulceration. Greaves and Skillen (1970) administered

220 mg of zinc sulfate orally 3 times per day (150 mg Zn/day) to 18 patients for 16 to 26 weeks. Three patients noted mild nausea after swallowing each dose. No hematological or biochemical evidence of zinc toxicosis was detected. In addition, some degree of re-epithelialisation of venous leg ulcers occurred in all 18 subjects with complete healing in 13 of the 18 patients.

Czerwinski et al. (1974) treated 16 geriatric patients diagnosed with senile dementia with 220 mg of zinc sulfate 3 times per day for 24 weeks (150 mg Zn/day). Diarrhea occurred in 37.5% of the patients compared to an incidence of 7% in patients given a placebo in a double-blind study. Treatment was discontinued in two Zn-treated patients because of persistent diarrhea. No other significant changes were noted during the 24 weeks of therapy.

Arakawa and co-workers (1976) observed no adverse effects in two male infants with acrodermatitis enteropathica, a skin disease, following treatment with 35 mg/day oral zinc sulfate. These doses represented 5.9 and 12 mg/kg of zinc per day.

Murphy (1970) reported the survival of a 16-year old boy who ingested 12 grams of metallic zinc (150 mg/kg) mixed with peanut butter. The patient exhibited profound lethargy, light-headedness, slight staggering of gait and experienced difficulty in writing legibly. Eight days after admission, blood zinc levels remained elevated (8.1 mg/kg liter).

b. Intravenous

Brocks et al. (1977) reported fatal zinc intoxication in a 72-year-old woman from an inadvertent intravenous overdose (46 mM or 7.4g $ZnSO_4$ over 60 hours). Her serum zinc concentration was 4184 µg/100 ml compared to a normal range of 75-124 µg/100 ml.

Gallery et al. (1972) reported a case of a patient on home dialysis who suffered acute toxic symptoms (nausea, vomiting and fever) correlating

with markedly raised blood zinc concentration when using water stored in a galvanized tank for dilution of dialysis fluid. Analysis of the tank water revealed 625 µg Zn/100 ml. She subsequently was found to have severe anemia with raised plasma and erythrocyte concentrations of zinc (700 and 3500 µg Zn/100 ml, resp.) 36 hours after dialysis. Normal ranges are: 70-110 µg Zn/100 ml for plasma; 1000-1400 µg Zn/100 ml for erythrocytes.

c. Inhalation

When zinc or its alloys are heated above 930°F, particles of zinc oxide are formed. If inhaled, these particles cause an acute febrile illness several hours after exposure. Commonly referred to as "metal-fume-fever", this disease is typically restricted to foundry workers. Inhalation of zinc oxide at concentrations of 15 mg/m^3 of zinc or above produces fever, malaise, headache, depression, excessive salivation and a cough which may be violent enough to induce vomiting (Kemper and Trautman, 1972; Anseline, 1972; Chmielewski et al., 1974; Jaremin, 1973; Hamdi, 1969).

Drinker et al. (1927a) reported results of 27 experimental inhalations of freshly generated ZnO in 10 subjects. Concentrations of 14 mg of ZnO/m^3 (measured as Zn) produced no reaction after 8 hours; 45 mg/m^3 was tolerated for 20 minutes.

Sturgis et al. (1927) reported on 2 cases of zinc-fume fever in 2 individuals following voluntary inhalation of freshly generated ZnO. Subjects sat in a 45 m^3 gas cabinet for ∿5-12 minutes. The chamber contained an average of 600 mg/m^3 of zinc. Subject A inspired approximately 48 mg of zinc, subject B, 74 mg of zinc. Typical febrile reactions occurred.

Beritic-Stahuljak et al. (1976) found a statistically significant increase in serum albumins and in alkaline phosphatase activity in 42 workers after 5 days exposure to zinc fumes. Schmahl (1974) reported that inhalation of $ZnCl_2$ fumes produced corrosive effects (from HCl

smoke) on the respiratory tract and lungs. Fischer (1974) observed 2 cases of lethal $ZnCl_2$ fume inhalation. The exposed men died 6 and 11 days following exposure. Lungs revealed bronchitis and confluent bronchopneumonia with thrombosis in the vessels, chronic pneumonia and bronchiolitis obliterans.

d. Dermal

Zinc and zinc salts are generally well tolerated by human skin. A contact dermatis, attributable to exposure to zinc chromate has been observed. The chromate, however, is presumed to be the prime offender (Hall, 1944). Belostotskaya et al. (1969) reported that 7 male workers developed allergic dermititis over large portions of their bodies from exposure to $ZnCl_2$.

e. Ocular

Houle and Grant (1973) reported on 2 cases of accidental splashing of $ZnCl_2$ into the eyes. One case involved soldering paste (pH 6.5): the other concentrated $ZnCl_2$ galvanizing solution (pH 3.53). Corneal edema developed and permanent corneal scarring resulted. Both patients developed persistent gray spots beneath the anterior lens capsule. Recovery to best stable visual acuity after injury required 6-28 weeks, but visual acuity needed permanent correction.

5. Overview

Zinc is an essential trace element in human and animal nutrition and is distributed throughout body tissues and fluids. Dietary requirements for zinc (10-15 mg/day) are linked to the needs for growth, tissue repair and compensation for excretion.

Diets grossly deficient in zinc have been associated with growth failure, loss of taste and in the postpubertal male, hypogonadism and decreased fertility. It is likely that factors in addition to zinc may also be involved.

In man, approximately 30% of ingested zinc is absorbed, transported throughout the body in the blood and excreted in urine, perspiration, semen, hair, nails and desquamated skin. Roughly 70-80% of ingested but unabsorbed zinc is found in the stool (~5-10 mg daily).

Except for the ability of zinc to induce testicular tumors when injected directly into the testes, no experimental evidence exists to suggest that ingestion or parenteral administration of zinc is either carcinogenic or mutagenic.

Excessive dietary intake (4000 ppm) of zinc prior to conception and throughout gestation appears to be linked to an increased incidence of fetal resorption in rats. However, rats similarly exposed to 2000 ppm zinc in the diet were unaffected.

Toxicological studies in experimental animals indicate that up to 2500 ppm in the diet has no effect on rats although 5000-10,000 ppm induce severe anemia. Acute oral LD_{50} values in laboratory animals range from 96 to 168 mg/kg of zinc. Possible interactions of zinc with other metals may contribute to toxicity of zinc.

In humans, in those cases in which high concentrations of zinc (150 mg Zn/day) have been ingested, such as for wound healing, no adverse effects other than gastric disturbances and diarrhea have been reported. Gastric discomfort has been reported following ingestion of 50 mg zinc while ingestion of 1-2 grams of zinc salts produces immediate vomiting. Survival following ingestion of 12 grams of zinc has been documented.

B. EFFECTS OF ZINC ON AQUATIC ORGANISMS

1. Introduction

This section provides information about the levels of zinc at which the normal behavior and metabolic processes of aquatic organisms are disrupted. Extensive research has been conducted on the zinc tolerance of many aquatic plant and animal species.

The toxicity of zinc is strongly influenced by a number of factors. Among the most important of these parameters is water hardness, which modified considerably the effects of zinc on aquatic organisms. Less clear are the relationships between zinc toxicity and pH, temperature, dissolved oxygen content, competing ions and ligands and salinity. These parameters cannot always be manipulated as freely as water hardness because of the possible consequences for the test animals, and so have not been as thoroughly studied. Another important variable is the species of plant or animal used in the toxicity experiments, as interspecies tolerances may vary by several orders of magnitude.

Over the past decade, tens of thousands of fish have been reported killed as a result of heavy metal pollution by mining and industrial activities, although most of the kills occurred before 1974 (see Table V-2). While in many cases several metals are implicated, the evidence in many cases has pointed to zinc as a major cause of the fish kills. In the context of these incidents, it is appropriate to attempt an evaluation of the effects of zinc on natural aquatic environments. Despite the failure of most laboratory experiments to duplicate natural conditions, the data summarized here can hopefully provide a basis for understanding the effects of zinc on aquatic life.

2. Freshwater Organisms

a. Chronic/Sublethal

Low concentrations of zinc can cause a wide variety of reactions in aquatic organisms, ranging from behavioral changes to growth inhibition and physical deformity. Although zinc in minute quantities may

Table V-2. Data for Zinc-Related Fish Kills (Since 1971)

Location	Industry Associated with Kill	Chemicals Implicated	Number of Fish Killed
Quinnipiac River Southington, Ct	Chemical	Phenol, Cu, Cr, Zn	1,000
Naugatuck River Tarrington, Ct	Chemical	Cu, Zn, Cr	400
Clark Fork River Missoula, Mt	Mining, Power Generation	Zn, 11.2 ppm, Cu, 4.6, Fe	?
Roaring Brook Glastonbury, Ct	?	Phenol, Cu, Zn	300
Lamberts Pond McCall, Id	Construction* (galvanized material)	Zn, 104 ppb	200
Caney River Bartlesville, Ok	Metals*	Zn ?	2,500
Eliza Creek Bartlesville, Ok	Metals*	Zn ?	123
Mokelumne River Clements, Ca	Mining*	Zn	900
Mokelumne River Clements, Ca	Mining*	Zn	32,823
Big Creek Glover, Mo	Metals	Pb, 984 ppb Zn, 976 ppb Cd, 500 ppb Mn, 445 ppb	?
Mill Creek Kent, Wa	Telephone Co.	Pb, Zn, Cu	100
Housatonic River Stratford, Ct	Metals	Ni, Cu, Zn	8,000
Elkhorn Creek Lexington, Ky	Metals	Cu, Zn	9,602

*Attributed to runoff events.

not be fatal to a fish, it may pose a threat to the species by diminishing reproductive potential or contributing to environmental stress.

Sprague (1968) has observed an avoidance response by rainbow trout (Salmo gairdneri), a particularly sensitive species, at a zinc concentration of 5.6 parts per billion (ppb). Other effects, however, are not evident until much higher levels are attained. In a study by Spehar(1976), 85 ppb zinc caused a significant increase in flagfish larvae (Jordanella floridae) mortality; at 139 ppb, there was no survival. Sinley et al. (1974) found rainbow trout fry to be somewhat more resistant, with an incipient mortality level of 260 ppb in soft water. Brungs (1969) has reported reduced egg production by fathead minnows (Pimephales promelas) at 180 ppb. When the concentration was increased to 2,800 ppb, their growth was stunted, spawning frequency declined, mortality rates rose to 15% (in 96 hours), and hatching was suppressed entirely.

At varying concentrations of zinc (depending on the species of fish and other factors), the gills become clogged with mucous. This apparently occurs in the presence of other heavy metals as well. An often-used indicator of copper or cadmium contamination in water is the frequency of "coughing", where the fish tries to clear the gills of mucous. Sparks et al. (1972) as cited in EPA (1979) has reported this response in the highly tolerant bluegill (Lepomis macrochirus) at a concentration of 40,000 ppb zinc. When the gill clogging reaches an advanced state, tissue hypoxia results, and the fish is gradually asphyxiated..

Bengtsson (1975) has observed several types of sublethal effects in the minnow (Phoxinum phoxinus) at low levels of aqueous zinc. Between 6 and 70 days after the beginning of exposure to 200 ppb and 300 ppb zinc, hemorrhages appeared behind the dorsal fins of several specimens, indicating vertebral damage. Some individuals exhibited pigmentation

disturbances with the appearance of dark verticle stripes in the caudal region, or black pigment in the tail. After 270 days, most of the fish had developed paralysis and muscular atrophy.

A summary of chronic values of freshwater fish is presented in Table V-3.

The only information on chronic toxicity levels for a freshwater invertebrate species if for the cladoceran, Daphnia magna, (EPA, 1979). The "chronic value" given is 84.5 ppb zinc; this indicates a mean level at which growth or reproduction is impaired.

The effects of zinc on freshwater plants range from small reductions in growth rates to mortality. The most sensitive species testes was the alga, Selenastrum capricornutum, for which the incipient growth inhibition concentration was 30 ppb. For a description of the responses of other plant species to different levels of zinc, see Table 5 in EPA (1979).

b. Acute Effects

The acute effects of zinc on freshwater fish have been studied extensively. Among the fish tested for sensitivity to zinc, the cutthroat trout (Salmo clarki) studied by Rabe and Sappington (1970) as cited in EPA (1979) were the least tolerant, with a 96-hour LC_{50} of 90 ppb zinc. For rainbow trout, fathead minnow, and bluegill, there are many experimental acute toxicity values which cover a wide range. In Table V-4, these values have been collected to give only lower and upper bounds because of the abundance of data for numerous species. For a more detailed dissussion of acute toxicity, see EPA (1979).

Invertebrate vulnerability to aqueous zinc extends through approximately the same range as for fish, with the notable exceptions of Daphnia hyalina and D. magna, which are more sensitive. The available data for acute toxicities to freshwater invertebrates are outlined in Table 2 of EPA (1979).

Table V-3

Chronic/Sublethal Effects on Freshwater Fish

Conc. (ppb)	Species	Compound	Hardness (mg/l)	Test Duration	Effects	Source
5.6	Rainbow trout (*Salmo gairdneri*)	$ZnSO_4$	13-15	20 min.	Threshold avoidance level	Sprague (1968)
51	Flagfish adults (females) (*Jordanella floridae*)	$ZnSO_4$	44	100 days	Growth reduced	Spehar (1976)
106	Fathead minnow (*Pimephales promelas*)	Zn++	46	?	Effect on growth, survival or reproduction in life-cycle test**	Benoit and Halcombe*
180	Fathead minnow	$ZnSO_4$	203	10 mo.	83% reduction in egg production	Brungs (1969)
187	Chinook salmon	Zn++	22	?	Effect on growth, survival or reproduction in embryo larval test**	Chapman (1978)*
260	Rainbow trout	$ZnSO_4$	25	42 days	Chronic bioassay 6.4 mortality	Sinley *et al.* (1974)
640			330		6.9 mortality	
852	Brook trout	Zn++	44	?	Effect on growth survival, or reproduction in life-cycle test**	Holcome *et al.* (1978)*

*As cited in EPA (1979).

**The value represents the geometric mean of the levels at which there effects are observed. In the case of embryo-larval tests the geometric mean is divided by 2 to obtain a value comparable to life-cycle studies.

Table V-3. Chronic/Sublethal Effects on Freshwater Fish (Continued)

Conc. (ppb)	Species	Compound	Hardness (mg/l)	Test Duration	Effects	Source
500-1000	Stickleback (Gasterosteus aculeatus)	$^{65}ZnCl_2$	282	3 days	Gill damage	Matthiesson and Brafield (1973)*
1000		$^{65}ZnCl_2$	282	200 hrs.	Increased oxygen uptake	Brafield and Matthiesson (1976)*
1150	Guppy (Poecilia reticulatus)	Zn++	80	30 days	Growth inhibition	Crandall and Goodnight (1962)*
1680	Rainbow trout	Zn++	320	48 hrs.	Increased swimming velocity, increased sensitivity to Zn	Herbert and Shurben (1963)*
8700	Bluegill (Lepomis macrochirus)	Zn++	51	7 days	Increased breathing rate	Cairns and Sparks (1971)*
40,000	Rainbow trout	Zn++	44-55	9 days	Gill tissue damage	Skidmore and Tovell (1972)*

*As cited in EPA (1976).

Table V-4. Acute Toxicities for Freshwater Fish

Concentration (ppb)	Species	Hardness (ppm) as $CaCO_3$	Reference
90 - 420	Cutthroat trout (*Salmo clarki*)	24	Rabe and Sappington (1970)*
97 - 463	Chinook salmon (*Oncorhynchus tshawytscha*)	24	Chapman (1978)*
100 - 6800	Striped bass (*Morone saxcitilis*)	<53 - 157	EPA (1979)
240 - 7210	Rainbow trout (*Salmo gairdneri*)	5 - 500	EPA (1979)
420 - 3130	Atlantic salmon (*Salmo salar*)	14 - 352	EPA (1979)
600 - 33,400	Fathead Minnow (*Pimephales promelas*)	20 - 360	EPA (1979)
749 - 1000	Sockeye salmon (*Oncorhynchus nerka*)	13 - 34	EPA (1979)
905 - 4600	Coho salmon (*Oncorhynchus kisutch*)	25 - 99	EPA (1979)
1270	Guppy (*Poecilia reticulatus*)	20	Pickering and Henderson (1966)*
1500	Flagfish (*Jordanella floridae*)	44	Spehar (1976)
1550 - 6980	Brook trout (*Salvelinus fontinalis*)	44 - 179	Holcombe and Andrew (1978)*
1930 - 23,000	Bluegill (*Lepomis macrochirus*)	20 - 360	
6000 - 11,400	Golden shiner (*Notemigonous crysoleucus*)	24 - 96	Cairns *et al.* (1978)*
6440 - 103,000	Goldfish (*Carassius auratus*)	20 - 50	
7800	Carp (*Cyprinus carpio*)	53 - 55	Rehwoldt *et al.* (1971, 1972)

*As cited in EPA (1979).

Table V-4. Acute Toxicities for Freshwater Fish (Continued)

Concentrations (ppb)	Species	Hardness (ppm) as $CaCO_3$	Reference
12,000	Southern platyfish (*Xiphophorus maculatus*)	166	Rochlir and Perlmutter, 1968
14,300-14,400	White perch (*Morone americana*)	53 - 55	Rehwoldt *et al.* (1971, 1972)
14,500-14,600	American eel (*Anguilla rostrata*)	53 - 55	Rehwoldt *et al.* (1971, 1972)
19,100-19,200	Banded killifish (*Fundulus diaphanus*)	53 - 55	Rehwoldt *et al.* (1971, 1972)
20,000-20,100	Pumpkinseed (*Lepomis gibbosus*)	53 - 55	Rehwoldt *et al.* (1971, 1972)

3. Saltwater Organisms

Relatively little research has been conducted on the toxicity of zinc to marine vertebrates. The mummichog, Fundulus heterroclitus, is the only marine fish that has been tested in sublethal concentrations of zinc. Eisler and Gardner (1973 as cited in EPA 1979) observed histological damage in this species after 24 hours in a 60,000 ppb solution. A 10,000 ppb concentration caused an increase in live ALA-D enzyme activity; the long-term consequences of this effect are unclear.

Acute toxicity data for marine vertebrates are also extremely limited. The mummichog is the only non-anadromous marine fish which tested for acute sensitivity to zinc. The 96-hour LC_{50} for the mummichog is 60,000 ppb zinc, according to Eisler and Hennekey (1977 as cited in EPA 1979), which is considerably higher than for most freshwater fish. Two anadromous fish, the rainbow trout and the Atlantic salmon, showed significantly increased resistance to aqueous zinc in saltwater as compared to fresh. The role of salinity in mitigating the effects of zinc will be discussed in the next section.

Considerably more data are available for invertebrates, and several species are highly sensitive to zinc. Reish and his associates (1976, 1978 as cited in EPA 1979) reported sublethal effects occurring between 220 and 1,250 ppb in four species of polychaetes. Oyster larvae (Crassostrea gigas) are among the most vulnerable marine organisms, according to a study by Nelson (1972 as cited in EPA 1979). At 125 ppb, the larvae exhibited reduced growth rates, and abnormal shell development occurred in 70 ppb after 48 hours. Crab (Rhithropanupeus harrisi) larvae development has been reported delayed after 16 days in a 50 ppb solution (Benijts-Claus and Benijts, 1975). See Table V-5 for more data.

The acute toxicity of zinc to saltwater invertebrates has been extensively studied, and there is much more information from which to draw conclusions. The interspecies tolerance range is extremely wide,

Table V-5 Sublethal Effects of Zinc on Marine Invertebrates*

Concentration (ppb)	Species	Effect	Reference
50	Crab (larva) (Rhithropanopeus harrisi)	Delayed development	Benijts-Calus and Benijts (1975)*
70	Oyster (larva) (Crassostrea gigas)	Abnormal shell development	Nelson (1972)
81	Sea urchin (spermatozoa) (Arbacia punctulata).	Decreased motility	Young and Nelson (1974)
125	Oyster (larva) (Crassostrea gigas)	Growth inhibition, reduced development	Brereton, et al. (1973)
125	Oyster (larva) (Crassostrea gigas)	Substrate attachment inhibition	Boyden, et al. (1975)
200	Mud snail (adult) (Nassarius obsoletus)	Decreased oxygen consumption	McInnes and Thurberg (1973)
250	Sea urchin (egg) (Anthocidaris crassispina)	Retarded development	Kobayoshi (1971)
320	Sea urchin (egg) (Anthocidaris crassispina)	Abnormal development	Okubu and Okubu (1962)
2700	Starfish (adult) (Asterias forbesi)	Equilibrium loss	Galtsoff and Loosanoff (1939)
3000	Polychaete (adult) (Eudistylia vancouveri)	Allantoise enzyme inhibition	May and Brown (1973)

*Taken from EPA (1979)

with hard-shell clam larvae (Mercenaria mercenaria) reported as the most susceptible organisms with a 48-hr. LC_{50} of 166 ppb. Table 9 in EPA (1979) provides a list of these invertebrate species and their median tolerance limits.

The effects of zinc on marine plants consist primarily of growth or photosynthesis inhibition. The most sensitive species studied is the alga, Skeletonema costatum, for which growth was suppressed at a concentration of 50 ppb. Table V-6 lists the other plant species and the toxic zinc concentrations.

4. Factors Affecting the Toxicity of Zinc

There are numerous variables in a natural aquatic environment which may strongly influence the toxicity of zinc to an organism. The hardness of the water, its temperature, dissolved solids content, pH, and the synergy of other substances all modify the toxicity of zinc.

Water hardness is perhaps the most important, and certainly the best documented, of these factors. The negative correlation between zinc toxicity and water hardness (as $CaCO_3$) has been confirmed in the laboratory by many researchers. For a species such as the fathead minnow, the toxicity of zinc varies by a factor of more than 50, largely depending upon the water hardness. Correlations between zinc toxicity and water hardness were computed for the rainbow trout, the bluegill, and the fathead minnow, since data were most complete for these species. The correlation coefficients were .788, .764, and .655, respectively, demonstrating that, despite variations in experimental procedure, the relationship between the two variables is strong.

Although data are limited for freshwater invertebrates, the LC_{50} values for the snail, Physa heterotropha, suggest only a slight influence by water hardness.

Table V-6 Effects of Zinc on Marine Plants*

Concentration (ppb)			
50	Alga (*Skeletonema costatum*)	Growth inhibition	Braek *et al.* (1976)
100	Kelp (*Laminaria digitata*)	Growth inhibition	Bryan (1964)
200	Alga (*Skeletonema costatum*)	Growth inhibition	Braek *et al.* (1976)
250	Kelp (*Laminaria hyperiborea*)	Growth inhibition	Hopkins and Kain (1971)
400	Alga (*Amphidinium carteri*)	Growth inhibition	Braek *et al.* (1976)
400	Alga (*Thalassiosira pseudomona*)	Growth inhibition	Braek *et al.* (1976)
500	Alga (*Thalassiosira pseudomona*)	Growth inhibition	Braek *et al.* (1976)
6500	Alga (*Duraliella tertiolecta*)	Reduction in potassium content	Overnell (1975)
10,000	Kelp (*Macrosystis pyrifera*)	Photosynthesis inhibition	Clendenning and North (1959)
25,000	Alga (*Phaeodactylum tricornutum*)	Growth inhibition	Jensen *et al.* (1974)

*Table taken from EPA (1979)

A study by Judy and Davies (1979) found similar effects on zinc toxicity levels using $Ca(NO_3)_2$. Their results indicated that the Ca^{++} cation is the agent responsible for suppressing the effects of zinc. Matthiesson and Brafield (1977) hypothesize that the base metals (including calcium) "impair the toxic action of zinc, either by reducing the permeability of cell membranes or by directly protecting the biochemical processes with which zinc interferes. They may also facilitate zinc excretion."

There are also external influences on zinc which affect its availability to fish. In the presence of a high concentration of hydroxide or carbonate anions (usually in hard water or high pH), or in a reducing environment, zinc precipitates. Mount (1966) noted an inverse relationship between pH and toxicity levels, such that the lowest LC_{50} value for fathead minnows coincided with the highest pH (8) used. Mortality rates werehigher in the tanks in which the water was "milky" as a result of precipitation. In addition, mucosis of the gills and the accompanying cough response were much more severe among fish in the more basic water.

Hardness and pH factors are probably insignificant for marine fishes, which are adapted to a slightly basic, alkaline medium with a high solute concentration. Because of a lack of data, the effects of salinity on zinc toxicity are not clear. Duke *et al.* (1969) as cited by NRC (1979) found that zinc-65 accumulation in the Atlantic oyster was 10% lower at 30 parts per thousand than at 25 ppt salinity.

Herbert and Wakeford (1964) studied the resistance of Atlantic salmon (*Salmo salar*) smelts and rainbow trout to zinc in varying degrees of salinity. In a 35% seawater solution (simulating estuarine conditions), their tolerance for zinc was, respectively, 13 and 15 times the level in hard fresh water. When the seawater proportion was raised to 72%, their resistances decreased, but were still substantially greater than in fresh water. This study suggests

that the relation between toxicity and salinity is complex. At high salinities, the increased resistance to zinc may be affected by some other mechanism.

Temperature and dissolved oxygen content (D.O.) have been examined for their effects on the toxicity of zinc to freshwater fish. Pickering (1967) observed an inverse relationship between D.O. and zinc toxicity for the bluegill. At D.O. levels of 1.8, 3.2, and 5.6 mg/l, the LC_{50} values were 7.4, 10.6 and 11.4, respectively.

The relationship between temperature and toxicity is not so easily elucidated. Sprague (1968) observed no effects on the avoidance reactions of rainbow trout over a temperature range of 7.5°C. Smith and Heath (1979) observed less resistance to zinc in the goldfish and the bluegill as the temperature of the water was successively raised from 5 to 15 to 30°C. However, they did not find this progression in the two other species tested, the rainbow trout and the shiner. Rehwoldt et al. (1972) observed six species of Hudson River fish in varying temperatures and found only insignificant differences in their sensitivity to zinc.

Any changes in tolerance to zinc that occur as a result of changes in dissolved oxygen content or temperature probably arise from environmental stress. It is difficult for many fish to obtain sufficient oxygen when the D.O. content of the water is 1.8 mg/l. Furthermore, in the temperature experiments it is difficult to control for a single variable because the oxygen saturation point in warm water is lower than in cold water. Consequently, these factors are probably more important for their direct effects upon the fish than for their influence on the toxicity of zinc.

In waters which are polluted with zinc from mining or industrial activities, other heavy metals are often found as well. Lead, mercury, copper, and cadmium are all toxic to varying degrees, and

have some similar effects on aquatic life. The importance of the presence of combinations of heavy metals lies in their synergistic effects. One example of this phenomenon is described in the work of Ozoh and Jacobson (1979) who exposed zebra cichlid (Chichlasoma nigrofasciatum) eggs to concentrations of 0, 16, and 32 ppb of zinc and copper, alone and in combinations. They found that the synergy of copper and zinc interfered more with hatching and normal growth than comparable concentrations of a single metal. Lorz et al. (1978) found that copper in the presence of zinc decreased the appetite of rainbow trout. He hypothesized that copper also increases the metabolic rate, which causes "a reduction in the condition factor" compared to fish exposed only to zinc. Braek et al. (1976) as cited in EPA (1979) has also observed adverse effects on growth in several species of alga as a result of they synergy between copper and zinc.

The interactions of heavy metals are not always synergistic, however. A study by Benijts-Claus and Benijts (1975) gives evidence of mutual suppression by lead and zinc. At concentrations up to 50 ppb, both metals had a significant adverse impact on the development of the mudcrab, Rhithropanopeus harrisi. In certain combinations, on the other hand, zinc apparently suppressed the more toxic lead, and larval growth actually accelerated. Jackim et al. (1977) as cited in EPA (1979) found that zinc in solution decreased the rate of cadmium uptake in the mussel, Mytilus edulis.

Synergy between zinc and other heavy metals depends on absolute and relative concentrations, the age of the solution as it relates to precipitation and dissociation, and environmental factors such as water hardness and pH. In natural environments it may be difficult to gather sufficient or appropriate data in order to determine the zinc exposure and risk to aquatic life. In the cases where a fish kill has already occurred, the same complications hinder efforts to isolate the effect of a single variable such as zinc.

Acclimation to zinc has been shown to occur in several areas. Spehar (1976) has observed that rainbow trout eggs exposed to zinc solution produce adults with a greater resistance to zinc. The data of Sinley et al. (1974) indicate that rainbow trout not exposed to zinc in the egg stage may be up to four times as sensitive to zinc as fish which were exposed to zinc as eggs. These results suggest that even the most susceptible species may adapt, within limits, to higher levels of exposure.

5. Summary of Aquatic Toxicity

According to the literature surveyed, the lowest concentration of zinc at which adverse effects in an aquatic organism have been observed is 30 ppb, a growth inhibition level for the alga, Selenastrum capricornutum. At 51 ppb, growth rates for female flagfish decreased significantly after 100 days of exposure. Acute effects appeared at 90 ppb for cutthroat trout. Hard-shell clam larvae are the most sensitive marine animals tested, for which the lowest 48-hour LC_{50} value found is 166 ppb. The saltwater alga, Skeletonema costatum, has experienced growth inhibition in concentrations as low as 50 ppb.

The toxicity of zinc in freshwater is strongly influenced by several environmental factors. Zinc toxicity is inversely related to water hardness such that the lowest LC_{50} values for a given species are associated with soft water (with low calcium levels). Low dissolved oxygen content places an environmental stress on aerobic organisms, which can increase their sensitivity to zinc. Temperature plays a similar role, as extremes can also create stress for the organism. In some cases, fish may adapt to levels of zinc which are toxic to previously unexposed fish.

The presence of other heavy metals is a complicating factor because their interactions with zinc are not well understood. It appears that copper and nickel act synergistically with zinc so that the overall toxicity exceeds that of a comparable concentration of a single metal.

There is some evidence, on the other hand, that lead and zinc are mutually suppressive, but the relationship should be further studied. Zinc has also been shown to decrease the rate of cadmium uptake in several animal species.

C. REFERENCES

Andrasi, A., J. Feher. 1967. Measurement of the retention and excretion of incorporated 65-Zn. Health Phys. 13:915-16.

Anseline, P. 1972. Zinc-fume fever. Med. J. Aust. 2(6):318-18.

Arakawa, T., T. Tamura, Y. Igarashi, H. Suzuki and H.H. Sandstead. 1976. Zinc deficiency in two infants during total parenteral alimentation for diarrhea. Am. J. Clin. Nutr. 29(2):197-204.

Belostotskaya, E.S., R.E. Reizina. 1969. Occupational dermatoses caused by zinc chloride. Gig. Tr. Prof. Zabol. 13(10):51-2, as cited in CA 72/47138c.

Bengtsson, Bengt-Erik. 1975. Vertebral damage in fish induced by pollutants. In: Sublethal Effects of Toxic Chemicals on Aquatic Animals, ed. J.H. Koeman and J.J.T.W.A. Strik, Elsener Scientific Publishing Company, Amsterdam.

Benijts-Claus C. and F. Benijts. 1975. The effects of low lead and zinc concentrations on the larval development of the mud-crab Rhithropanopeus harrisi Gould. In: Sublethal Effects of Toxic Chemicals on Aquatic Animals, ed. J.H. Koeman and J.J.T.W.A. Strik, Elsener Scientific Publishing Company, Amsterdam.

Beritic-Stahuljak, D., D. Dimov and T. Beritic. 1976. Changes in the blood picture, liver function and protein status in exposure to zinc fumes. Acta Med. Iugosl. 30(3):239-49.

Brocks, A., H. Reid, and G. Glazer. 1977. Acute intravenous zinc poisoning. Brit. Med. J. May:1390-1.

Brown, V.M. 1968. The calculation of acute toxicity of mixtures of poisons to rainbow trout. Water Research 2:273.

Brungs, W.A. 1969. Chronic toxicity of zinc to the fathead minnow, Pimephales promelas Rafinesque. Trans. Amer. Fish. Soc. 2:272.

Bryan, G.W. 1976. Some aspects of heavy metal tolerance in aquatic organisms. In: Effects of Pollutants on Aquatic Organisms, ed. A.P.M. Lockwood, Cambridge: Cambridge University Press.

Burch, R.E. and J.F. Sullivan. 1976. Clinical and nutritional aspects of zinc deficiency and excess. Med. Clin. North Am. 60(4):675-85.

Burch, R.E., H.K. Hahn, and J.F. Sullivan. 1975. Newer aspects of the roles of zinc, manganese, and copper in human nutrition. Clin. Chem. 21(4):501-20.

Cairns, J. Jr. et al. 1975. The effects of lapsed time since feeding upon the toxicity of zinc to fish. Bull. Environ. Contam. Toxicol. 13:269.

Calabrese, A. and D.P. Nelson. 1974. Inhibition of embryonic development of the hard clam, Mercenaria mercenaria by heavy metals. Bull. Environ. Contam. Toxocol. 11:92.

Carleton, R.L., N.B. Friedman, and E.J. Bomze. 1953. Experimental teratomas of the testis. Cancer 6:464-73.

Caujolle, F., P.H. Chanh, N.L.T. Ngoc-Suong and P. Van Tô. 1969. Toxicology of zinc. I. Immediate and deferred toxicity and long-term toxicity. Agressologie 10(4):333-9.

Chapman, G.A. and D.G. Stevens. 1978. Acutely lethal levels of cadmium, copper, and zinc to adult male coho salmon and steelhead. Trans. Am. Fish. Soc. 107:837.

Chmielewski, J., B. Jaremin, C. Bartnicki, and R. Konieczka. 1974. Evolution of occupational exposure to zinc oxide in the marine production shipyard. II. Examination of the state of health of the workers exposed to zinc oxide. Bull. Inst. Mar. Med. Gdansk. 25(1):53-65.

Ciapparelli, L., D.H. Retief, and L.P. Fatti. 1972. The effect of zinc on 9,10-dimethyl-1, 2-benzanthracene (DMBA)-induced salivary gland tumors in the albino rat — A preliminary study. S. Afr. J. Med. Sci. 37(3): 85-90.

Czerwinski, A.W., M.L. Clark, E.A. Serafetinides, C. Perrier, and W. Huber. 1974. Safety and efficacy of zinc sulfate in geriatric patients. Clin. Pharmacol. Ther. 15(4):436-41.

Davies, I.J., M. Musa, T.L. Dormandy. 1968. Measurements of plasma zinc. I. In health and disease. J. Clin. Pathol. 21:363-5.

De Wys, W. and W.J. Pories. 1972. Inhibition of a spectrum of animal tumors by dietary zinc deficiency. J. Natl. Cancer Inst. 48:375-81.

De Wys, W., W.J. Pories, M.C. Richter, and W.H. Strain. 1970. Inhibition of Walker 256 carcinosarcoma growth by dietary zinc deficiency. Proc. Soc. Exp. Biol. Med. 135:17-22.

Drinker, P., R.M. Thompson, and J.L. Finn. 1927a. Metal fume fever: IV. Threshold doses of zinc oxide, preventive measures, and the chronic effects of repeated exposures. J. Ind. Hyg. 9:331-45.

Drinker, K.R., P.K. Thompson, and M. Marsh. 1927b. An investigation of the effect upon rats of long-continued ingestion of zinc compounds, with special reference to the relation of zinc excretion to zinc intake. Amer. J. Physiol. 81:284, cited in Van Reen, 1966.

Drinker, K.R., P.K. Thompson, and M. Marsh. 1927c. An investigation of the effect of long-continued ingestion of zinc, in the form of zinc oxide, by cats and dogs, together with observations upon the excretion and the storage of zinc. Amer. J. Physiol. 80:31, cited in Van Reen, 1966.

Duncan, J.R., I.E. Dreosti, and C.F. Albrecht. 1974. Zinc intake and growth of a transplanted hepatoma induced by 3'-methyl-4-dimethylamino-azobenzene in rats. J. Natl. Cancer Inst. 53(1):227-8.

Elinder, C.G. and M. Piscator. 1978. Cadmium and zinc relationships. Environ. Health Perspect. 25:129-32.

Ferm, V.H. and S.J. Carpenter. 1967. Teratogenic effect of cadmium and its inhibition by zinc. Nature 216(120):1123.

Ferm, V.H. and S.J. Carpenter. 1968. The relationship of cadmium and zinc in experimental mammalian teratogenesis. Lab. Invest. 18:429-32.

Fischer, H. 1974. Morphology of lethal zinc chloride smoke intoxication of the lung (Author's Transl.) Pneumonologie 150(2-4):171-2.

FCT, 1972. The blessings of zinc. Food Cosmet. Toxicol. 10(4):578-83.

Flynn, A., W.J. Pories, W.H. Strain, and O.A. Hill, Jr. 1973. Zinc deficiency with altered adrenocortical function and its relation to delayed healing. Lancet 1(807):789-90.

Food and Nutrition Board. 1974. As cited in Principles of Internal Medicine, T.R. Harrison, ed., Eighth Edition, McGraw-Hill Book Company, 1977, New York, p. 439.

Frazier, J.M. 1979. In vitro binding of cadmium, zinc, and copper to rat and human plasma proteins. Abs. Eighteenth Annual Meeting of Society of Toxicology, New Orleans, LA, p. A75.

Galin, M.A., H.D. Nano, and T. P. Hall. 1962. Ocular zinc concentration. Invest. Opthal. 1:142.

Gallery, E.D., J. Blomfield, and S.R. Dixon. 1972. Acute zinc toxicity in haemodialysis. Br. Med. J. 4(836):331-3.

Greaves, M.W., and A.W. Skillen. 1970. Effects of long-continued ingestion of zinc sulphate in patients with venous leg ulceration. Lancet 2(679): 889-91

Goolamali, S.K. and J.S. Comaish. 1975. Zinc and the skin. Int. J. Dermatol. 14(3):182-7.

Guthrie, J. 1967. Specificity of the metallic ion in the experimental induction of teratomas in fowl. Br. J. Cancer 21(3):619-22.

Guthrie, J. and O.A. Guthrie. 1974. Embryonal carcinomas in Syrian hamsters after intratesticular inoculation of zinc chloride during seasonal testicular growth. Cancer Res. 34(10):2612-4.

Hall, A.F. 1944. Occupational contact dermatitis among aircraft workers. J. Am. Med. Assoc. 125:179, cited in Vallee, 1959.

Halsted, J.A., H.A. Ronaghy, P. Abadi, M. Haghshenass, G.H. Amirhakemi, R.M. Barakat and J.G. Reinhold. 1972. Zinc deficiency in men. The Shiraz experiment. Am. J. Med. 53(3):277-84.

Halsted, J.A., J.C. Smith, Jr., M.I. Irwin. 1974. A conspectus of research on zinc requirements of man. J. Nutr. 104(3):345-78.

Hamdi, E.A. 1969. Chronic exposure to zinc of furnace operators in a brass foundry. Br. J. Ind. Med. 26(2):126-34.

Hardisty, M.W. et al. Dietary habits and heavy metal concentrations in fish from the Severn Estuary and Bristol Channel. Marine Pollution Bulletin...

Hedges, J.D., E.T. Kornegay, and H.R. Thomas. 1976. Comparison of dietary zinc levels for reproducing sows and the effect of dietary zinc and calcium on the subsequent performance of their progeny. J. Animal Sci. 43:453-63.

Heller, V.G. and A.D. Burke. 1927. Toxicity of zinc. J. Biol. Chem. 74:85, cited in Van Reen, 1966.

Herbert, D.W.M. and A.C. Wakeford. 1964. The susceptibility of salmonid fish to poisons under estuarine conditions - I: Zinc sulphate. J. Air Wat. Poll. 8:251

Herick, R. 1969. Effect of zinc on mitosis. Naturwissenschaften 56:286.

Holcombe, G.W. and R.W. Andrew. 1978. The Acute Toxicity of Zinc to Rainbow and Brook Trout: Comparisons in Hard and Soft Water, Duluth: Environmental Research Laboratory.

Horvath, D.J. 1976. Chapter 5, Trace elements and health. In: Trace Substances and Health. A Handbook, Part 1, Paul M. Newberne, ed. Marcel Dekker, Inc., New York, pp. 319-56.

Hoss, D.W. 1964. Accumulation of zinc-65 by flounder of the genus paralichthys. Trans. Am. Fish. J. 93:364.

Houle, R.E. and W.M. Grant. 1973. Zinc chloride and keratopathy and cataracts. Am. J. Ophthalmal. 75(6):992-6.

Hurley, L.S. and H. Swenerton. 1966. Congenital malformations resulting from zinc deficiency in rats. Proc. Soc. Exp. Biol. Med 123:692, cited in Task Group on Metal Interaction, 1978.

Hurley, L.S., J. Gowan, and H. Swenerton. 1971. Teratogenic effects of short-term and transitory zinc deficiency in rats. Teratology 4:199.

Jameson, S. 1976. Zinc deficiency in malabsorption states: A cause of infertility. Acta Med. Scan. Suppl. 593:38-49.

Jaremin, B. 1973. Clinical picture of the "zinc fever" (Observation of 43 patients). Bull. Inst. Mar. Med. Gdansk 24(3):233-42.

Judy, R.D. Jr. and P.H. Davies. 1979. Effects of calcium addition as $Ca(MO_3)_2$ on zinc toxicity to fathead minnows, Pimephales promelas, Rafinesque. Bull. Environ. Contam. Toxicol. 22:88.

Kemper, F. and A. Trautman. 1972. Inhalation of zinc oxide. Dtsch. Med. Wochenschr. 97(8):307.

Kumar, S. 1976. Effect of zinc supplementation on rats during pregnancy. Nutr. Rep. Int. 13:33-36.

Lindeman, R.D., R.G. Bottomly, R.C. Cornelison, and L.A. Jacobs. 1972. Influence of acute tissue injury on zinc metabolism in man. J. Lab. Clin. Med 79:452-60.

Lorz, H.W. et al. 1978. Effects of several metals on smolting of coho salmon. Corvallis: Corvallis Environmental Research Laboratory.

Marshak, A. and G. Marshak. 1973. Zinc sulfate therapy for vocal cord granulomas. J. Laryngol. Otol. 87(6):573-6.

Matsumoto, H., T. Shiraishi, and K. Tsunematsu. 1969. Zinc content in gastric juice in normal subjects and patients with gastric disease. Igaku to Seibutsugaku 78(1):39-40.

Matthiesson, P. and A.E. Brafield. 1977. Uptake and loss of dissolved zinc by the stickleback Gasterosteus aculeatus L. J. Fish Biol. 10:399.

McIntosh, A. and W. Bishop. 1976. Distribution and Effects of Heavy Metals in a Contaminated Lake. Purdue University Water Resources Research Center, West Lafayette, Indiana.

McQuitty, J.T., Jr., W.D. De Wys, L. Monaco, W.H. Strain, C.G. Rob, J. Apgar, and W.J. Pories. 1970. Inhibition of tumor growth by dietary zinc deficiency. Cancer Res. 30:1387-90.

Methfessel, A.H. and H. Spencer. 1973. Zinc metabolism in the rat. I. Intestinal absorption of zinc. J. Appl. Physiol. 34:58-62.

Mount, D.I. 1966. The effects of total hardness and pH on acute toxicity of zinc to fish. Air and Water Pollu.10:49.

Mulay, I.L., R. Roy, B.E. Knox, N.H. Suhr, and W.E. Delaney. 1971. Trace metal analysis of cancerous and non-cancerous human tissues. J. Natl. Cancer Inst. 47:1-13, cited in Schwartz, 1975.

Murphy, J.V. 1970. Intoxication following ingestion of elemental zinc. JAMA. 212(12:2119-20.

Nakatani, R.E. 1966. Biological response of rainbow trout (Salmo gairdneri) ingesting zinc-65. Disposal of Radioactive Wastes in Seas, Oceans and Surface Waters. IAEA.

National Research Council (NRC). 1978. Zinc, prepared by the Subcommittee on Zinc, Committee on Medical and Biologic Effects of Environmental Pollutants. National Academy of Sciences, Washington, D.C.

Nehring, R.B., and J.P. Goettl, Jr. 1974. Acute toxicity of a zinc-polluted stream to four speces of salmonids. Bull. Environ. Contam. Toxicol. 12:464.

Nordberg, G.F., editor. 1976. Effects and Dose-Response Relationships of Toxic Metals. Amsterdam, Elsevier.

Ozoh, P.T.E. and C.O. Jacobson. 1979. Embryotoxicity and hatchability in Cichlasoma nigrofasciatum (Guenther) eggs and larvae briefly exposed to low concentrations of zinc and copper ions. Bull. Environ. Contam. Toxicol. 21:782.

Parisi, A.F. and B.L. Vallee. 1970. Isolation of a zinc α_2-macroglobulin from human serum. Biochemistry 9:2421-6.

Pecoud, A., P. Donzel, and J.L. Schelling. 1975. The effect of food-stuffs on the absorption of zinc sulfate. Clin. Pharmacol. Ther. 17:4 469-74.

Pequenat, J.E. et al. 1969. Estimates of the zinc requirements of marine organisms. J. Fish. Res. Bd. Canada 26:145.

Petering, H.G., H.H. Buskirk, and J.A. Crim. 1967. The effect of dietary mineral supplements of the rat on the antitumor activity of 3-ethoxy-2-oxobutyraldehyde bis (thiosemicarbazone). Cancer Res. 27:1115-21.

Pickering, Q.H. 1968. Some effects of dissolved oxygen concentrations upon the toxicity of zinc to the bluegill (Lepomis macrochirus). Water Res. 2:187.

Poswillo, D.E. and B. Cohen. 1971. Inhibition of carcinogenesis by dietary zinc. Nature 231:447-8.

Prasad, A.S., ed. 1966a. Zinc Metabolism. Charles C. Thomas, Springfield, Ill.

Prasad, A.S. 1966b. Metabolism of zinc and its deficiency in human subjects. Chapter 15 in Prasad, 1966a.

Prasad, A. 1979. Clinical, biochemical, and pharmacological role of zinc. Ann. Rev. Pharmacol. Toxicol. 20:393-426.

Prasad, A.S. and D. Oberleas. 1970. Binding of zinc to amino acids and serum proteins in vitro. J. Lab. Clin. Med. 76:416-25.

Prasad, A.S., A. Miale, Jr., Z. Farid, H.H. Sandstead, and A.R. Schulert. 1963a. Zinc metabolism in patients with the syndrome of iron deficiency anemia, hepatosplenomegaly, dwarfism, and hypogonadism. J. Lab. Clin. Med. 61:537-49.

Prasad, A.S., A.R. Schulert, H.H. Sandstead, A. Miale, Jr., and Z. Farid. 1963b. Zinc, iron and nitrogen content of sweat in normal and deficient subjects. J. Lab. Clin. Med. 62:84-9.

Prasad, A.S., E.B. Schoomaker, J. Ortega, G.J. Brewer, D. Oberleas, and F.J. Oelshlegel, Jr. 1975. The role of zinc in man and its deficiency in sickle cell disease, in: Progress in Clinical and Biological Research, Vol I. Erthyocyte Structure and Function, G.J. Brewer, ed. Alan R. Liss, New York, pp 603-19.

Prasad, A.S. and D. Oberleas. 1974. Thymidine kinase activity and incorporation of thymidine into DNA in zinc-deficient tissue. J. Lab. Clin. Med. 83:634-9.

Pullen, F.W. 1970. Post-intubation tracheal granuloma. A preliminary report on the efficacy of zinc sulfate. Arch. Otolaryngol. 92(4):340-2.

Rachlin, J.W. and A. Perlmutter. 1968. Response of an inbred strain of platyfish and the fathead minnow to zinc. Prog. Fish-Cult. 30:203.

Rehwoldt, R. et al. 1971. Acute toxicity of copper, nickel and zinc ions to some Hudson River fish species. Bull. Environ. Contam. Toxicol. 6:445.

Rehwoldt, R. et al. 1972. The effects of increased temperature upon the acute toxicity of some heavy metal ions. Bull. Environ. Contam. Toxicol. 8:91.

Reinhold, J.G., B. Faradji, P. Abadi, and F. Ismail-Beigi. 1976. Decreased absorption of calcium, magnesium, zinc, and phosphorous by humans due to increased fiber and phosphorous consumption as wheat bread. J. Nutr. 106(4):493-503.

Richmond, C.R., J.E. Furchner, G.A. Trafton, and W.H. Langham. 1962. Comparative metabolism of radionuclides in mammals. I. Uptake and retention of orally administered Zn-65 by four mammalian species. Health Phys. 8:481-9.

Riviere, M.R., I. Chouroulenkov, and M. Guerin. 1960. The production of tumors by means of intratesticular injections of zinc chloride in the rat. Bull. Ass. Fr. Etude Cancer 47:55-87.

RTECS (Registry of Toxic Effects of Chemical Substances), Vol. II. 1977. E.J. Fairchild, R.J. Lewis, Sr., and R.L. Tatken, eds. U.S. DHEW, NIOSH, 78-104-B.

Sadasivan, V. 1951. Studies on the biochemistry of zinc. 1. Effect of feeding zinc on the liver and bones of rats. Biochem. J. 48:527, as cited in Van Reen, 1966.

Sandstead, H.H. 1975. Some trace elements which are essential for human nutrition. Zinc, copper, manganese, and chromium. Prog. Food Nutr. Sci. 1(5):371-91.

Sandstead, H.H. and R.A. Rinaldi. 1969. Impairment of deoxyribonucleic acid synthesis by dietary zinc deficiency in the rat. J. Cell. Physiol. 73:81-4.

Schmahl, K. 1974. Clinical signs in zinc-chloride smoke intoxication (Author's transl.). Pneumonologie 150(2-4):161-9.

Schlicker, S.A. and D.H. Cox. 1967. Maternal dietary Zn in excess, fetal development and Fe and Cu metabolism. Fed. Proc. 26:520.

Schlicker, S.A. and D.H. Cox. 1968. Maternal dietary zinc, and development and zinc, iron and copper content of the rat fetus. J. Nutr. 95:287-94.

Schroeder, H.A., A.P. Nason, I.H. Tipton, and J.J. Balassa. 1967. Essential trace metals in man. Zinc: Relation to environmental cadmium. J. Chron. Dis. 20:179.

Schwartz, M.K. 1975. Role of trace elements in cancer. Cancer Res. 35(11): Part 2, 3481-7.

Scott, D.A., and A.M. Fisher. 1938. Studies on the pancreas and liver of normal and zinc-fed cats. Amer. J. Physiol. 121:253-60.

Shiraishi, T., K. Tsunematsu, and H. Matsumoto. 1969. Changes in serum zinc level in tumor-bearing rats. Med. Biol. (Tokyo) 78(1):31-3.

Sinley, J. R. et al. 1974. The effects of zinc on rainbow trout (Salmo gairdneri) in hard and soft water. Bull Environ. Contam. Toxicol. 12:193.

Skidmore, J.F. 1965. Resistance to zinc sulphate of the zebrafish (Brachydanio rerio Hamilton-Buchanan) at different phases of its life history. Ann. Appl. Biol. 56:47.

Smit, Z.M. and P.J. Pretorius. 1964. Studies in metabolism of zinc. Part 2. Serum zinc levels and urinary zinc excretions in South African Bantu Kwashiorkor patients. J. Trop. Pediatr. 9:105-12.

Smith, M.J. and A.G. Heath. 1979. Acute toxicity of copper chromate, zinc and cyanide to freshwater fish: Effect of different temperatures. Bull. Environ. Contam. Toxicol. 22:113-119.

Spencer, H., B. Rossof, A. Feldstein, S.H. Cohn and E. Gusmano. 1965. Metabolism of zinc 65 in man. Radiation Res. 24:432-45.

Sprague, J.B. 1968. Avoidance reactions of rainbow trout to zinc sulfate solutions. Water Research 2:367.

Sturgis, C.C., P. Drinker, and R.M. Thomson. 1927. Metal fume fever: I. Clinical observations on the effect of the experimental inhalation of zinc oxide by two apparently normal persons. J. Ind. Hyd. 9:88-97.

Suso, F.A. and H.M. Edwards, Jr. 1971. Binding capacity of intestinal mucosa and blood plasma for zinc. Proc. Soc. Exp. Biol. Med. 137:309-09

Sutton, W.R. and V.E. Nelson. 1937. Studies on zinc. Proc. Soc. Exp. Biol. Med. 36:211, cited in Van Reen, 1966.

Swenerton, H., R. Shrader, and L.S. Hurley. 1969. Zinc deficient embryos: Reduced thymidine incorporation. Science 166:1014-15.

Task Group on Metal Interaction, Scientific Committee on the Toxicology of Metals. 1978. Factors influencing metabolism and toxicity of metals: A consensus report. International Association on Occupational Health. Environ. Health Perspect. 25:3-41.

Tsuchiya, K. and S. Iwao. 1978. Interrelationships among zinc, copper, lead, and cadmium in food, feces, and organs of humans. Environ. Health Perspect. 25:119-24.

Underwood, E.J. 1971. Trace Elements in Human and Animal Nutrition, 3rd Edition, Academic Press, New York.

U.S. Environmental Protection Agency. 1979. Zinc: Ambient Water Quality Criteria.

Vallee, B.L. 1959. Biochemistry, physiology, and pathology of zinc. Physiol. Rev. 39:443.

Van Reen, R. 1966. Zinc toxicity in man and experimental species. Chapter 23 in Prasad, 1966a.

Van Rosen, G. 1954. Breaking of chromosomes by the action of elements of the periodical system and by some other principles. Hereditas 40:258-63.

Walters, M. and F.J. Roe. 1965. A study of the effects of zinc and tin administered orally to mice over a prolonged period. Food Cosmet. Toxicol. 3(2):271-6.

Webb, M. 1971. Protection by zinc ions against the toxicity of cadmium ions. Biochem. J. 124(2):17-18.

Zazgornik, J., R. Kotzaurek, and P. Schmidt. 1971. Plasma zinc concentration in uremic patients during hemodialysis. Klin. Wochenschr. 49(5): 278-80.

VI. EXPOSURE

A. HUMAN EXPOSURE

1. Introduction

The previous section on the effect of zinc on humans indicates that it has a very low order of toxicity. In fact, most discussions regarding human exposure to zinc emphasize zinc deficiency, as opposed to effects resulting from large doses. As a result, this section will not go into great detail in estimating zinc exposure to various subpopulations. It will attempt to provide order of magnitude estimates for exposure to zinc through various routes.

2. Ingestion

a. Food

NRC (1979) has reviewed the zinc content of various foods extensively as this subject has been examined in some detail. Briefly, zinc in the diet is primarily supplied with protein. Oysters have the highest reported zinc content (per 100 grams), but other meats, fish, dairy products and grain products are also important sources. FDA (1974), as part of their total diet studies, found that about 40% of the zinc in the diet of a 15-20 year old male was supplied by meat, fish and poultry; dairy products supplied 24% and grain and cereal supplied 19%. The total dietary intake was estimated to be 18.6 mg zinc/day. This level of exposure is well below levels at which any effects are observed, and is just above the recommended daily allowance for zinc of 15 mg.

Certain subpopulations would be exposed to higher levels of zinc in their diet, for example, persons eating large amounts of oysters. In addition zinc may be ingested as a dietary supplement. These persons may ingest up to 75 mg/day (in zinc gluconate), in addition to intake from food.

b. Drinking Water

Zinc in drinking water is generally low, although it may be picked up in the distribution system. The U.S. H.E.W. (1970) reported that a survey

of 2595 distribution samples over the U.S. showed a maximum of 13.0 mg/l, with eight samples exceeding 5 mg/l, the recommended limit for drinking water. The mean concentration was 0.194 mg/l. Using the maximum value for distributed drinking water, intake of zinc can be estimated at 26 mg/day. It is evident that in a few locations, intake of zinc in drinking water may be comparable to intake through food.

3. Inhalation

Zinc concentrations in air have been discussed previously in Section IV-A. Using the maximum 24 hour value near a smelter of 15.7 $\mu g/m^3$ (EPA, 1979), an intake of .314 mg/day can be estimated for residents near smelters. Thus, even for a worst case situation for inhalation, this route results in much lower exposures to zinc than food and drinking water.

Smokers may receive small amounts of zinc in smoke. EPA (1979) has calculated an inhalation of up to 0.02 mg might result from smoking 20 cigarettes. This exposure is insignificant when compared to other routes.

4. Absorption

Due to the low order of toxicity of zinc, absorption would be expected to be an insignificant exposure route compared to food. Ambient waters are generally at concentrations of less than 1 mg/l, resulting in a low potential for exposure.

B. EXPOSURE OF ZINC TO AQUATIC ANIMALS

As one of the most abundant heavy metals in the earth's crust, zinc occurs naturally in small concentrations in virtually all fresh and salt water bodies. Levels of zinc in undisturbed environments are determined largely by the composition of the local substrate, which varies geographically. Human activities such as mining and manufacturing can also influence zinc concentrations in the air, soil and water (see Section III).

Under natural conditions, sea water contains between 1 and 27 ppb zinc, while uncontaminated fresh waters generally have less than 50 ppb (NRC, 1979). Ambient concentrations of zinc, however, are commonly higher. In Table IV-1 are given the most commonly observed ranges of concentrations for aqueous zinc in the major river basins of the U.S. Table IV-2 provides the same data for sediment samples.

In areas near zinc smelters and mines or where zinc is used in manufacturing, ambient water concentrations may be high (see Section IV-A). Waste water discharge streams from smelters may have 50 to 243 ppm zinc (NRC, 1979). Within the mid-Atlantic, Missouri, Arkansas, and Tennessee river basins are many of the major zinc mines in the U.S. The monitoring data presented in Figures IV-2 and IV-3 suggest a moderate association between zinc production and zinc concentrations in water bodies in these areas. In addition, fate considerations (Section IV-B) suggest that heavy rainfalls, dredging, or other physical disturbances to the sediment may result in temporarily high concentrations of zinc.

Hard water has a strongly suppressive effect on the toxicity of zinc to fish (see Section V-B). In setting criteria for zinc toxicity, EPA (1979) determined an inverse logarithmic relationship between toxicity and water hardness as $CaCO_3$. Depending solely on hardness, the LC_{50} for a species may vary by a factor of 100 (EPA, 1979). The river systems in the U.S. which have the softest water are the New England, Pacific Northwest, California, and Southeast watersheds.

Figures VI-1 and VI-2 indicate counties in the U.S. where aqueous zinc levels exceed EPA acute and chronic criteria. This analysis includes data collected for the last ~20 years on zinc levels in the U.S. As an alternative approach we analyzed STORET data for 1978. Table VI-1 shows minor river basins having high concentrations of zinc and very soft water. It is interesting to note that while mean concentrations of greater than 120 ppb are common, in many cases 50% of the observations are not greater than 120 ppb. This distribution indicates that in many

Figure VI-1

Ratio of Observed Zn and Criteria Zn (Acute)

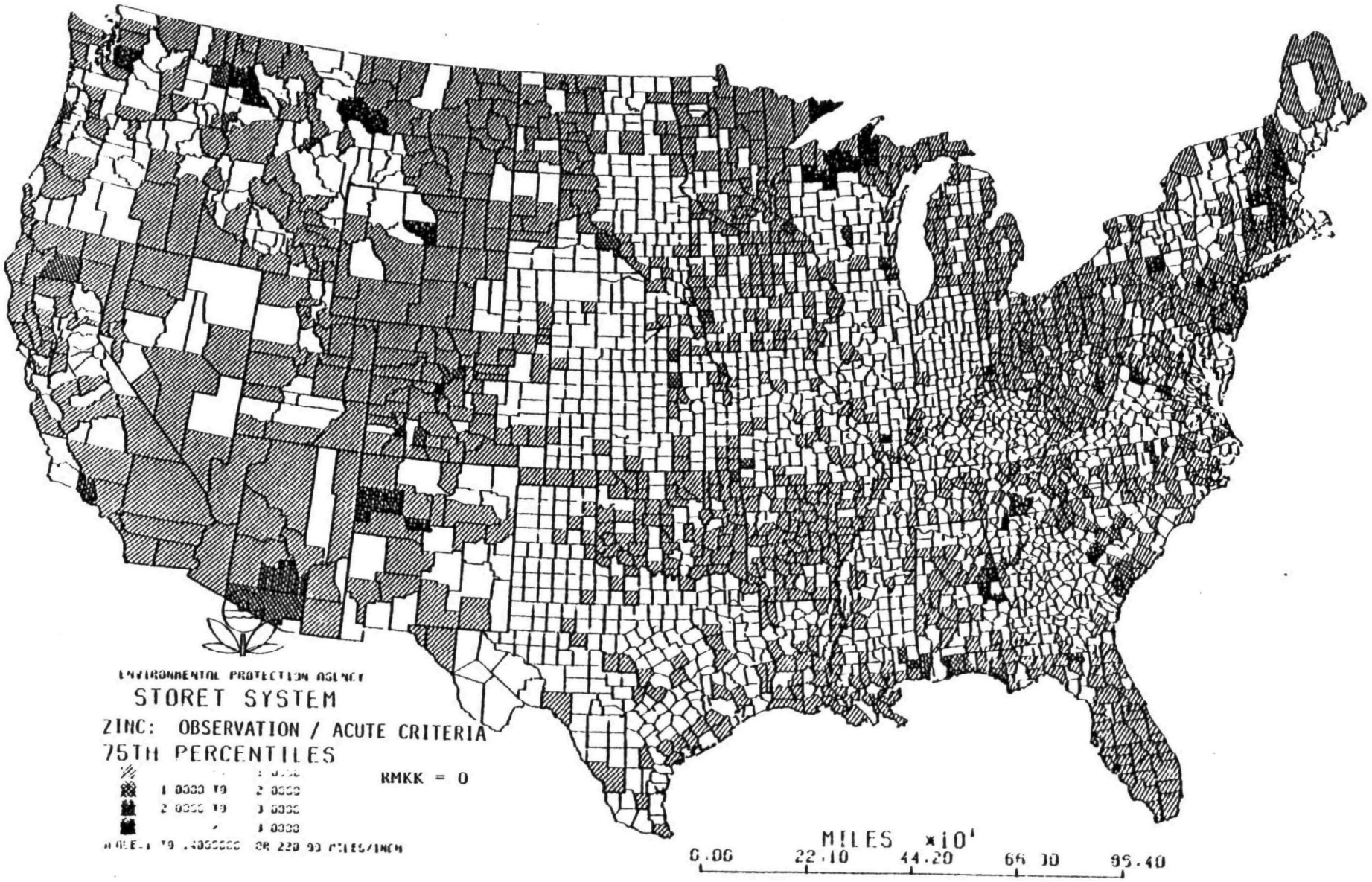


Figure VI-2

Ratio of Observed Zn and Criteria Zn (Chronic)

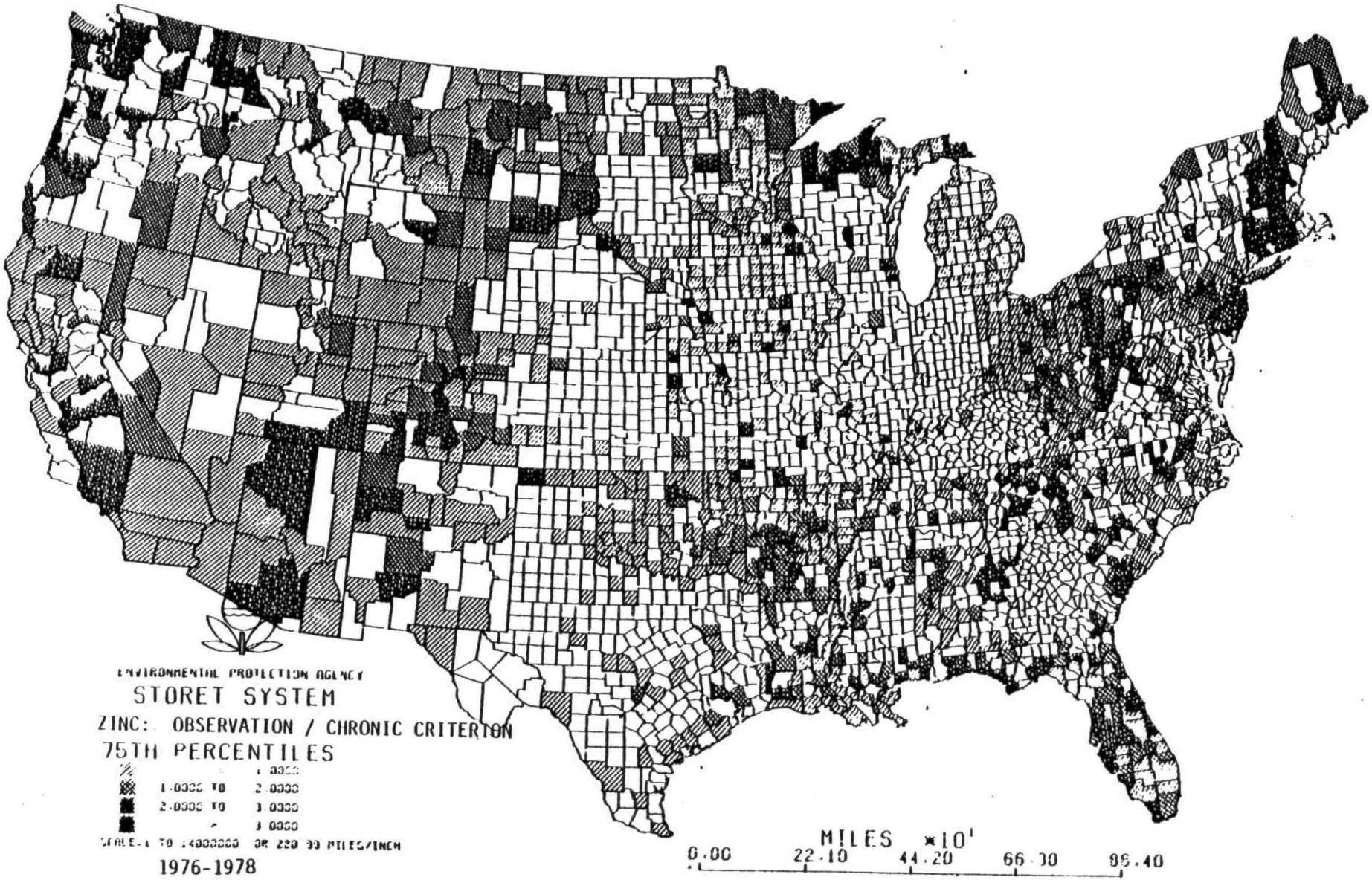


Table VI-1

Zinc Concentrations in U.S. Minor River Basins - 1978

	River Basin Major/Minor Name	Zinc N	Mean Zn >120 ppb	≥50% of ppb observations >120 ppb Zn	≥10% of ppb observations >300 ppb Zn	≥50% of hardness Measurements <50 ppm
1/9	Merrimack R.	126	*			*
1/14	Presumpcot R. & Casco Bay	24	*	*	*	
1/24	Lake Champlain	10	*	*	*	
2/8	Delaware R. - Zone 4	305	*	*	*	
2/15	Rappahannock & York Rivers	296	*			*
5/2	Monongahela R.	331	*			
5/3	Beaver R.	25	*		*	
5/7	Kanawha R	338	*		*	
5/13	Miami R.	86	*			
5/21	Ohio R., main stem & tribs	257	*	*	*	
6/3	Cuyahoga R.	21	*		*	
6/13	Detroit	9	*		*.	
7/2	Hudson Bay, Rainy River (23/02)	5	*	*	*	
7/3	Upper portion, upper Mississippi R.	135	*		*	
7/6	Lower portion, upper Mississippi R.	189	*		*	
7/12	Mississippi, Salt Rivers	9	*		*	
7/16	Fox R.	24	*		*	
7/19	Meramec R.	42	*		*	
8/3	Menominee	50	*		*	
8/24	Green Bay, W. Shore	42	*		*	
8/49	Calumet-Burns Ditch Complex	42	*			
9/14	S. Central Missouri R.	70	*		*	
9/7	Big Sioux R.				*	
9/12	Lower Missouri R.	37	*			

Table VI-1 (Continued)

River Basin Major/Minor Name		Zinc N	Mean Zn >120 ppb	>50% of ppb observations >120 ppb Zn	>10% of ppb observations >300 ppb Zn	>50% of hardness Measurements <50 ppm
11/2	Middle Colorado R.	158	*			
11/4	Gila R.	81	*		*	
11/5	Little Colorado R.	31	*		*	
12/7	Nueces R.	10			*	
13/5	Columbia R. above Yakima R.	34	*			
14/4	Central California Coastal	7			*	
14/5**	Santa Clara R.	5	*		*	
14/6	Los Angeles R.	87	*	*	*	
14/9	Sacramento R.	77	*		*	
15/6	Colorado R. Basin in California	11	*			

**Fewer than 10 measurements at this station.

cases there are a few high observations, but that most of the observations were less than 120 ppb. This premise is confirmed by the fact that numerous river basins show more than 10% of the observations greater than 300 ppb. These data are mapped in Figure IV-2. According to the EPA Water Quality Criteria, at a hardness of 50 ppb, the 24 hr. average criterion would be 27 ug/l Zn, while at a hardness of 250 ppm the criterion would be 79 ug/l Zn.

Exposure Routes

Fish are exposed to zinc in both their diets and the water, and for some species from the sediment. NRC (1979) theorizes that dietary intake is the more important pathway for ingestion because "turnover rates of larger body pools of zinc cannot be sustained by inflow rates of zinc from water alone." NRC further states that the use of short-term exposures of fish to experimental dissolved zinc concentrations in test for acute toxicoses is of dubious value because the fish die before their body pools have equilibrated with the environmental level of zinc.

Zinc levels in sediment are generally about two orders of magnitude higher than in whole water samples (STORET). The availability of sediment-bound zinc to aquatic organisms is uncertain because the relative importance of different exposure routes is not known. Exposure could occur through ingestion or gill uptake, particularly in benthic biota. Absorption into tissues is partially dependent on the composition of the sediment to which the zinc is bound. Luoma and Jenne (1977) reported that zinc was more available to clams when bound to biogenic calcium carbonate than to manganese and iron oxides and organic sediments.

Sediment is also a potential source of aqueous zinc because shifts in pH or hardness could release complexed or precipitated zinc into suspended or dissolved form.

A number of aquatic animals bioaccumulate zinc in their tissues. Unfortunately, existing studies on concentration usually do not give the accompanying aqueous zinc concentration or the zinc levels in the diet.

It is therefore not clear whether zinc is accumulated as a result of dietary intake or absorption from respiration. Bioconcentration in aquatic organisms is discussed in more detail in Section VI-B.

C. CONCLUSIONS

Table VI-1 shows areas of the country where zinc concentrations are high and where waters are very soft. It is evident from this table that aquatic organisms in the following minor river basins are subject to exposures commonly greater than 120 ppb and at least occasionally to greater than 300 ppb:

Presumpcot River and Casco Bay
Lake Champlain
Delaware River - Zone 4
Beaver River
Ohio River
Cuyahoga
Detroit
Hudson Bay, Rainy River
Upper portion - Upper Mississippi River
Lower portion - Upper Mississippi River
Mississippi, Salt Rivers
Fox River
Meramec River
Menominee
Green Bay - West Shore
S. Central Missouri River
Gila River
Little Colorado River
Santa Clara River
Los Angeles River
Sacramento River

D. REFERENCES

Cooke, M. et al. 1979. Biological availability of sediment-bound cadmium to the edible cockle, Cerastoderma edule. Bull. Environ. Contam. Toxicol 23:381-86.

Luoma, S.N. and E.A. Jenne. 1977. Proceedings 15th Annual Hartford Life Sci. Symp., E.R.D.A. Tech. Inf. Center. CONF 750929, as cited in Cooke et al., 1979.

Matthiesson, P. and A.E. Brafield. 1977. Uptake and loss of dissolved zinc by the stickleback Gastrostreus aculeatus. J. Fish. Biol. 10: 399.

National Research Council. 1979. Zinc, Subcommittee on Zinc, Committee on Medical and Biological Effects of Environmental Pollutants.

Pequenat, J.E. et al. 1969. Estimates of the zinc requirements of marine organisms. J. Fish Res. Board Can. 26:145.

U.S. E.P.A. 1979. Zinc Criterion Document.

U.S. F.D.A. 1974. Total Diet Studies. Compliance Program Evaluation.

U.S. H.E.W. 1970. Community Water Supply Study, Analysis of National Survey Findings.

VII. RISK CONSIDERATIONS

A. INTRODUCTION

It is the objective of this section to delineate the population exposed and quantify the risk associated with that exposure. Zinc has not been found to be a carcinogen, teratogen or mutagen. Reproductive effects have been indicated in rats after ingestion of high levels of zinc in the diet. It is questionable whether these effects can be extrapolated to man for two reasons. First, there appears to be little correlation, in general, between reproductive effects in man and reproductive effects in laboratory animals. In addition, it is unclear whether humans could or would accept such a dose due to its undesirable taste and emetic nature. Human data are available on some acute effects and oral lethality. Since levels required for human effects are well above expected exposure, a quantitative discussion of risk is not necessary in this case.

Effects of zinc on aquatic organisms, however, have been observed in the field in the form of fish kills. Therefore, it is likely that some subpopulations of aquatic organisms may be at risk. These subpopulations will be identified by species or class and type of geographic location. Further delineation of aquatic subpopulations at risk is not possible within the time constraints of this project.

B. HUMANS

Table VII-1 summarizes the known adverse effects of zinc on mammals. As discussed in Section V-A, the levels of zinc which result in adverse effects vary from person to person. The 25 mg dose is probably a lower limit for no effect. Gastric discomfort was observed at an acute dose of 50 mg zinc. However, lethality, either via an oral or intravenous exposure requires a dose of at least 1 gram.

TABLE VII-1

Adverse Effects of Zinc on Mammals

Adverse Effect	Species	Lowest Reported Effect Level (% Incidence)	No Apparent Effect Level
Fetal Resorption	Rat	4000 ppm diet days - 21 to + 15 of gestation (100%)	2000 ppm
Fetal Resorption	Rat	180 ppm in marginal protein diet days 0-18 (9.4%)	30 ppm
Severe anemia	Mouse	5000 ppm Zn	1250 ppm Zn
Gastric discomfort *	Man	50 mg Zn (83%)	25 mg
Nausea	Man	1 - 2 grams	--
Lethality	Man	7.4 grams $ZnSO_4$ IV over 60 hours	--
LD_{Lo}** (oral)	Man	3.5 g $ZnSO_4$	--
Lowest lethal dose		3.5 g $ZnCl_2$	Survival reported from ingestion of 12 g
--	Man	--	150 mg Zn/day for 26 weeks

* fasting

** lowest observed lethal dose

Table VII-2 summarizes the exposure levels for zinc. It is obvious from this table that food is generally the largest source of exposure for humans, although drinking water exposure may be as important in a few locations. The expected total intake for an individual would probably not be greater than 50 mg. This level, according to the results in Table VII-1, would result in gastric discomfort in some of the population exposed at this level. In order to achieve this intake, however, an individual would have to be consuming a diet high in zinc, and be residing in an area in which water is supplied at a zinc concentration equivalent to the extreme example of 13 mg/l zinc. Hence, the population exposed at this level is small, and the effect expected would not be serious or irreversible. However, little is known about long term exposures to zinc at these levels.

A subpopulation ingesting zinc supplements is receiving additional exposure, and the total intake may be greater than 100 mg. This population is probably not geographically or demographically identifiable without additional study. This level of exposure is probably not cause for concern, since Table VII-1 indicates that ingestion of 150 mg zinc per day for 26 weeks had no effect. However, acute sub-lethal effects may be observed in some persons at this level.

C. AQUATIC ORGANISMS

There are three major concerns in determining the risk of zinc exposure to aquatic animals. First, regions of the country where zinc concentrations are high (for example, locations where zinc is mined, smelted, or used in manufacturing)face the possibility of industrial discharges or runoff from mines or tailings. Incidences such as these have been the cause of many fish kills in the past. Major river basins which drain areas of high zinc productivity include the mid-Atlantic, Missouri, Arkansas, and Tennessee.

Table VII-2

Zinc Exposure to Humans*

Ingestion	Intake mg/day	Intake mg/kg/day**	Comment
Food	18.6	0.27	Based on average diet of 15-20 year old male.
Dietary Supplement	12-75	0.17 - 1.1	
Drinking Water	26	0.37	Based on maximum reported value in drinking water and consumption 2 liters/day.
	0.38	.005	Based on mean concentration in drinking water.
Inhalation of Ambient Air	0.3	.004	Based on monitoring data near smelters and an inhalation of 20 m^3/day.
Smoking	0.02	.0003	Based on smoking 20 cigarettes, see EPA (1979).

* Does not include occupational exposures.

** Based on 70 kg person.

Secondly, because of the mitigative effect of calcium on zinc toxicity, rivers and lakes with soft water (low in calcium) are more likely to experience fisk kills at lower zinc concentrations. Regions with soft water (<100 ppm as $CaCO_3$) include New England, the Pacific Northwest, northern California and the Southeast, excluding Florida.

Table VII-3 describes the geographic locations where fish populations may be exposed to toxic levels of zinc due to high zinc concentrations and low hardness in general. Specific locations within these large areas were identified in the previous section. The Western Gulf, Southeast, Missouri, Upper Colorado, Rio Grande and Pecos River basins in many instances exceed 100 ppb zinc in their waters. The sediments of the mid-Atlantic, Great Lakes, Tennessee and Pacific Northwest commonly contain 10 to 100 ppm of zinc. The New England watershed poses perhaps the greatest problem, as both its water and sediment fall into these categories. However, detailed analysis has shown that it is primarily the Presumpcot River and Casco Bay that are the problem areas in New England, while concentrations elsewhere are lower.

The final parameter to consider in assessing risk to aquatic life is the sensitivity of various species to zinc. As a group, the salmonids are probably the most sensitive to aqueous zinc. Species such as the rainbow trout which are found in the northeast, Great Lakes, and western states, may be threatened, both in terms of sublethal and acute toxicosis. Warmwater fish are somewhat more resistant to zinc than salmonids, but may also be at risk in some locations. For example, the chronic value for the fathead minnow is 106 ppb, as shown in Table V-3. According to Figure IV-1 over 25% of the water smples taken nationwide had zinc concentrations exceeding 100 ppb.

The cladoceran Daphnia magna experiences acute effects in zinc concentrations as low at 100 ppb. Invertebrate life may therefore be endangered as well in a large portion of the nation's waterways. The growth of certain species of alga such as Selenastrum capricornutum is inhibited

TABLE VII-3

FACTORS CONTRIBUTING TO RISK TO AQUATIC ORGANISMS

Zinc-Producing River Basins	River Basins with Soft Water
Mid-Atlantic	New England
Missouri	Northern California
Arkansas and Red	Pacific Northwest
Tennessee	Southeast (except Florida)

River Basins with High Aqueous Zinc Concentrations*

New England	45%
Western Gulf	31%
Southeast	30%
Rio Grande and Pecos	28%
Upper Colorado	26%
Missouri	24%

River Basins with High Zinc Concentrations in Sediment**

Great Lakes	47%
Mid-Atlantic	36%
New England	33%
Pacific Northwest	33%
Tennessee	31%

* Percentage of samples in the range of 100-1,000 ppb

** Percentage of samples in the range of 10-100 ppm.

completely at 120 ppb (See Section V-B). Although most invertebrate and plant species are not sensitive to zinc in the 100 to 1,000 ppb range, a number of plant and animal species appear to be presently exposed to harmful concentrations.

The premise that these species is threatened so broadly seems somewhat suspicious. There may be a number of reasons that the actual risk is less than the data imply. First, the monitoring data described here are for total zinc. Thus, in most cases at least a portion of this will be in the particulate form, which may be unavailable to aquatic organisms. It has been shown for other metals, i.e., cadmium and copper, that the form of the metal is important in determining toxicity. In general, organic complexes and some inorganic complexes are not available to aquatic organisms. However, fate considerations discussed previously (Section IV-B). indicated that dissolved zinc exists primarily as the free ion in natural waters. Thus the presence of organic and inorganic ligands may not be a determining factor in the toxicity of zinc. Another factor which may redefine risk is acclimation. Studies have shown that adults that were hatched in high levels of zinc are more tolerant than those that were not. The implications and interactions of these factors cannot be determined at this time. However, the potential for aquatic risk to zinc exists in numerous locations.

APPENDIX - HUMAN TOXICITY

Metabolism

Zinc Balance

Zinc balance in mammals is controlled by homeostatic processes that have not yet been clearly elucidated. Factors that affect zinc homeostasis include blood loss, sweating, prolonged intravenous feeding, fasting, burns, infection, nephrosis, chelating agents, myocardial infarction, surgery, cirrhosis, hormones, the presence of other heavy metals (i.e., cadmium, calcium, copper), pregnancy, lactation, and certain dietary components including protein, phytate, fiber, and alcohol (Oberleas and Prasad, 1976; Lindeman, 1972). In a normal 70 kg adult, zinc balance is maintained at equilibrium by an intake of ∿12.5 mg/day (Spencer et al., 1976). This requirement is higher (20-25 mg/day) in pregnant and nursing mothers (NAS, 1978).

Absorption and Distribution

In man, approximately 30% of zinc ingested in the diet is absorbed (Prasad, 1979). Cases of up to 90% absorption have been reported in zinc-deficient individuals (Aamodt et al., 1975; Richmond et al., 1962). The exact mechanism or sites of zinc absorption are not well understood, but absorption in man is believed to occur primarily in the proximal part of the small intestine although absorption may occur at other portions of the small and large intestine as well (Prasad, 1979; Methfessel and Spencer, 1973).

While the precise mechanism by which zinc is transported across the intestinal membrane is unknown, considerable data exist to support the existence of a low-molecular-weight (<100,000 daltons) zinc ligand which facilitates zinc transport across the intestinal epithelia. Studies with laboratory animals have revealed the presence of zinc binding proteins in the intestinal lumen; in the basal and apical plasma membranes and cytosol of the intestinal

epithelia; and in pancreatic secretions (Hahn and Evans, 1973; Evans and Hahn, 1974; Van Campen and Kowalski, 1971; Kowarski et al., 1974; Birnstingl et al., 1956; Montgomery et al., 1943).

Song and Adham (1978) have identified prostaglandin (MW 800 daltons) as an important zinc-binding protein in the small intestine of rats. Their in vitro work with isolated rat small intestine indicates that prostaglandin E_2 facilitates the transport of zinc across the intestinal mucosa.

Zinc is transported throughout the body in the blood, bound mainly to the serum proteins albumin, ceruloplasmin, transferrin, and α-2-macroglobulin (Prasad and Oberleas, 1970; Parisi and Vallee, 1970; Suso and Edwards, 1971; Frazier, 1979).

Spencer et al. (1965) examined the metabolism and tissue distribution of ^{65}Zn chloride in eleven terminal cancer patients. The period between the single intravenous injection of ^{65}Zn (20-53 μCi) and death due to cancer ranged from 1 to 71 days. Tissue samples were obtained and assayed for ^{65}Zn content at the time of autopsy. Urine and blood samples were taken at regular intervals from the time of injection until the time of death.

The authors found that intravenously administered ^{65}Zn was cleared rapidly from the blood. In one typical patient, whole blood and plasma contained 22% and 20%, respectively, of the administered dose 13 minutes after injection. Exponential clearance of zinc from blood lasted for approximately one hour, at which time the blood and plasma levels of ^{65}Zn were less than 5% of the administered dose. The blood levels diminished very slowly after the first hour, with ∿1.0% of the administered dose detectable in the blood 40 days after injection.

In both human subjects and laboratory animals, serum-bound ^{65}Zn exchanges rapidly with zinc in the soft tissues such as liver, spleen, kidneys, prostate, pancreas, adrenals, and thyroid. Zinc uptake and turnover in bone,

hair, muscle, nails, and testis occurs more slowly than that observed in the soft tissues (Gilbert and Taylor, 1956; Heath and Liquier-Milward, 1950; Spencer et al., 1965; Thind and Fischer, 1975). The biological half-life of ^{65}Zn in man has been calculated to be between 154 to 334 days (Spencer et al., 1965; Richmond et al., 1962; Andrasi and Feher, 1967).

Excretion

Studies of the excretion of labelled zinc in terminal cancer patients indicate that the main pathway of ^{65}Zn excretion in man is the gut. In two cancer patients surviving 45 days beyond a single intravenous injection of ^{65}Zn, cumulative fecal elemination of ^{65}Zn was 21.6% and 16.8%, respectively, compared to urinary excretion of 0.87% and 3.4% (Spencer et al., 1965). In man, fecal elimination accounts for the major portion of zinc excretion regardless of the route of administration (NAS, 1978). In addition, when zinc is administered orally, the 70-80% of the ingested zinc which is not absorbed, is also cleared in the feces.

Animal Studies

Zinc metabolism appears to occur in essentially the same manner in all mammals studied, although interspecies differences exist with regard to tissue distribution and retention. In rats, as in man, the relative percent absorption of zinc in the small intestine decreases as the concentration of dietary zinc increases (Bohne, et al., 1967).

Mice injected intravenously with 0.33 of ^{65}Zn chloride eliminated 25% of the administered dose in the feces within the first 24 hours after injection. At 170 hours post-injection, 50% of the dose had been eliminated in the feces, while urinary excretion accounted for only 2.0% of the administered dose (Sheline et al., 1943). In a similar study with dogs, approximately 5.0% of the administered intravenous dose (5.7 γ $^{65}ZnCl_2$) was eliminated in the feces within 24 hours of injection. At two weeks, cumulative fecal elimination accounted for 25.0% of the administered dose. The cumulative urinary excretion of ^{65}Zn at 15 days post-injection was 5.0% and 2.0%, respectively (Sheline et al., 1943).

Male albino rats eliminated 55.0% of a single 100 μCi intraperitoneal dose of $^{65}ZnCl_2$ in the feces within 23 days of injection; less than 2.0% of the administered dose was excreted in the urine during this period. Fecal elimination was reported to have been rapid during the first two or three days after injection, but much slower thereafter. Rapid uptake of ^{65}Zn by soft tissues (particularly the prostate) and the slow turnover in bone and testes were also noted (Wakeley et al., 1960).

TABLE A-1

ACUTE TOXICITY OF ZINC SALTS

Zn Salt	Species	Route	LD_{50} (mg/kg)	Source
$ZnCl_2$	Human	oral	50 LDLo*	RTECS, 1977
	Guinea pig	oral	200	"
	Rat	oral	350	"
	Mouse	oral	350	"
	Human	inhalation	4800 mg/m^3/30 min TCLo**	"
	Rat	intravenous	50 LDLo	"
	Guinea pig	intraperitoneal	173 LDLo	"
ZnO	Human	oral	500 $LDLo_3$	"
	Human	inhalation	600 mg/m^3 TDLo	"
$ZnSO_4$	Human	oral	50 LDLo	"
	Rabbit	oral	2000 LDLo	"
	Rat	oral	2200 LDLo	"
	Mouse	oral	626	Caujolle et al. (1969
	Rat	oral	1396	"
	Rabbit	intravenous	44 LDLo	RTECS, 1977
	Rat	intravenous	50 LDLo	"
	Dog	intravenous	60 LDLo	"
	Mouse	intraperitoneal	29	Bienvenu et al. (1963)
	Dog	subcutaneous	78 LDLo	RTECS, 1977
	Rabbit	subcutaneous	300 LDLo	"
	Rat	subcutaneous	330 LDLo	"
$ZnSO_4 \cdot 7H_2O$	Rabbit	oral	1914 LDLo	"
	Rat	oral	2200 LDLo	"
	Rabbit	intravenous	44 LDLo	"
	Rat	intravenous	49 LDLo	"
	Dog	intravenous	66 LDLo	"
	Dog	subcutaneous	78 LDLo	"
	Rat	subcutaneous	330 LDLo	"
	Guinea pig	subcutaneous	590 LDLo	"

*Lowest published lethal dose.

**Lowest published lethal concentration.

TABLE A-2

HUMAN TISSUE CONCENTRATIONS

Chemical Zinc

Population	Geographic Region	Tissue	Number Sampled	Distribution	Remarks	Reference
Adult	---	Serum or Plasma	---	63-170 µg/100 ml		Halsted et al., 1974
		Red blood cells	---	10-14 µg/ml		
Human controls	Detroit	Plasma	23	112 ± 2.5 µg% ± S.E.		Prasad et al., 1975
		Red blood cells	23	41 ± 1.2 µg/g Hgb ± S.E.		
Sickle-cell anemia patients		Plasma	27	102 ± 2.7 µg% ± S.E.		
		Red blood cells	27	35.2 ± 1.6 µg/g Hgb ± S.E.		
				µg/100 ml ± SD (range)		
Pregnant females	U.S.A.	Plasma	107	63 ± 12 (40-102)	Low plasma zinc levels were associated with elevated copper levels (249 ± 52 µg%) in 70 pregnant women and 10 women on oral contraceptives (300 ± 70 µg%) compared to controls (119 ± 20 µg%)	Halsted and Smith, 1970
Female controls			27	97 ± 11 (76-112)		
Male controls			62	96 ± 13 (72-115)		
Women taking oral contraceptives			30	81 ± 14 (60-110)		

HUMAN TISSUE CONCENTRATIONS

Chemical Zinc

Population	Geographic Region	Tissue	Number Sampled	Distribution	Remarks	Reference
				µg/100 ml ± S.E.		
Women	U.S.A.	Serum	45	90 ± 3		Henkin et al., 1971
Pregnant women		Serum	15	48 ± 3		
		Umbilical cord serum	15	83 ± 3		
		Amniotic fluid	15	32 ± 12		
Patients with chronic renal failure-undergoing renal dialysis	Austria	Plasma	9	261 ± 92 µg%		Zazgornik et al., 1971
Patients with chronic renal failure - no dialysis			--	117 ± 95 µg%		
Controls			--	108 ± 20 µg%		

HUMAN TISSUE CONCENTRATIONS

Chemical

Population	Geographic Region	Tissue	Number Sampled	Distribution	Remarks	Reference
				µg/100 ml ± SD		
Children with Kwashiorkor	S. Africa	Serum	29	62 ± 20.9		Smit and Pretorius, 1964
Controls			--	113 ± 14.7		
Children with Kwashiorkor	India	Plasma	28	41.3 ± 1.5 µg/100 ml ± S.E.		Kumar and Rao, 1973
Controls			--	102 ± 4.7 µg/100 ml ± S.E.		
Females	---	Breast milk	--	3-4 mg/l		Prasad, 1966
		Colostrum	--	20 mg/l		
Females	---	Menstrual fluid	--	308-616 µg/cycle	Assumes menstrual loss of 25-70 ml blood per cycle	Schroeder et al., 1967
Humans	---	Sweat	--	1.15 mg/l	In hot climate, as much as 5 liters of sweat may be lost per day	Prasad et al., 1963b

HUMAN TISSUE CONCENTRATIONS

Chemical Zinc

Population	Geographic Region	Tissue	Number Sampled	Distribution	Remarks	Reference
Humans	Sweden	Urine	96	1.04 ± 0.14 ppm		Wester, 1975
Eight-to ten-year-old children	Poland	Urine	--	0.602 mg/dm^3	Industrial region	Dutkiewicz et al. 1978
			--	0.605 mg/dm^3	Agricultural region	
				µg/g creatine + S.E.		
Sickle-cell anemia patients	Detroit	Urine	12	739 ± 60		Prasad et al., 1975
			10	495 ± 35		
				mg/day ± S.D. (dry feces wgt.)		
Adult males	Japan	Feces				Tsuchiya and Iwao, 1978
(age - > 50 yrs)	- Polluted area		30	21.61 ± 12.48		
	- Control area		30	10.27 ± 4.73		
(age 21-24 yrs)	- Tokyo		19	10.45 ± 6.48		

HUMAN TISSUE CONCENTRATIONS

Chemical Zinc

Population	Geographic Region	Tissue	Number Sampled	Distribution	Remarks	Reference
Humans	---	Skin	--	10 µg/g (dry wgt)		Goolamali and Comaish, 1975
Teenagers undergoing orthodontic procedures	Norway	Teeth	8 1	76-204 ppm 542 ppm		Oehm, et al., 1978
Eight-to-ten-year-old children	Poland	Hair	-- --	177-213 mg/kg 192 mg/kg	Industrial area Agricultural area	Dutkiewicz et al 1978
Human controls	Detroit	Hair	17	193 ± 4.3 µg/g ± S.E.		Prasad et al., 1975
Sickle-cell anemia patients			21	149 ± 10.3		
Healthy pregnant women at time of delivery (Age 16-39 yrs)	U.S.A.	Scalp hair	109	143.3 µg/g		Creason et al., 1976
		Pubic hair	158	110.1 µg/g		

HUMAN TISSUE CONCENTRATIONS

Chemical Zinc

Population	Geographic Region	Tissue	Number Sampled	Distribution	Remarks	Reference
				ppm (dry)		
Human, post-mortem samples	---	Retina	3	385 - 571		Galin, 1962
		Choroid	3	419 - 562		
		Ciliary body	3	189 - 288		
Males	---	Prostate(biopsy)		µg/g dry tissue		
		Normal	12	520 µg/g		Schrodt et al., 1964
		Hyperplasia	9	2300 µg/g		
		Carcinoma	10	285 µg/g		
				µg/g		
Adult males	---	Bone	--	150 - 250		Underwood, 1971
		Prostate	--	850		
		Semen	--	3000		
		Sperm	--	2000		

HUMAN TISSUE CONCENTRATIONS

Chemical: Zinc

Population	Geographic Region	Tissue	Number Sampled	Distribution	Remarks	Reference
				µg/g wet tissue ± S.D.		
Normal human fetuses (obtained by abortion, days 85–185 of pregnancy	Japan	Liver	14	167.7 ± 39.4		Chaube et al., 1973
		Brain	14	5.6 ± 1.2		
		Kidney	14	15.77 ± 2.9		
Normal human embryos (obtained by abortion)	Japan	Whole embryo		µg Zn/g wet tissue (range)		
Age days: 31			4	2.8 (2.6 - 3.0)		
35			10	19.7		
39			3	18.2		
41			10	18.0 (14.5 - 20.9)		
45			6	20.62 (12.3 - 27.08)		
64			1	20.5		
70			1	22.6		
78			1	24.8		

HUMAN TISSUE CONCENTRATIONS

Chemical Zinc

Population	Geographic Region	Tissue	Number Sampled	Distribution			Remarks	Reference
Adults, post-mortem samples	Norway		(% male)	µg/g tissue dry wgt (Range)				Syverson et al., 1976
Age yrs:				Liver	Renal Cortex	Renal Medulla		
16-18			2 (100)	670 (462-878)	503 (289-716)	376 (181-570)		
26-29			3 (100)	215 (64-296)	201 (131-273)	163 (134-206)		
30-33			3 (66)	507 (285-647)	307 (184-463)	251 (118-418)		
43-46			3(100)	242 (74-384)	227 (159-283)	136 (119-149)		
50-59			16 (87)	326 (113-990)	240 (100-312)	201 (67-323)		
60-69			17 (56)	292 (97-915)	229 (105-443)	170 (72-327)		
70-79			17 (50)	273 (146-441)	229 (150-353)	176 (80-350)		
80-89			14 (46)	366 (214-715)	255 (142-564)	184 (76-452)		
91			1(100)	306 (64-990)	236 (100-716)	179 (67-570)		

HUMAN TISSUE CONCENTRATIONS

Chemical Zinc

Population	Geographic Region	Tissue	Number Sampled	Distribution	Remarks	Reference
Adult males, accident victims	St. Louis, Missouri	Liver	10	4570 µg/g tissue ash		Perry et al., 1978
Adults, post-mortem samples	Sweden	Liver	39	79.2 ± 45.1 µg/g wet tissue		Wester, 1975
Adults, accident victims	Japan			µg/g wet tissue ± S.D.		Tsuchiya and Iwao, 1978
Age (yrs)				Liver / Renal Cortex		
<1			6	33.9 ± 1.4 / 17.43 ± 1.19		
1-9			9	65.01 ± 1.53 / 38.78 ± 1.87		
10-19			6	57.20 ± 1.20 / 33.42 ± 1.33		
20-29			16	56.25 ± 1.56 / 47.16 ± 1.65		
30-39			4	62.72 ± 1.26 / 56.99 ± 1.11		
40-49			19	45.82 ± 1.80 / 61.47 ± 1.64		
50-59			4	60.58 ± 1.31 / 55.83 ± 1.19		
60-69			10	56.81 ± 1.54 / 72.43 ± 1.64		
70-			11	60.59 ± 1.21 / 48.00 ± 1.31		

LITERATURE - APPENDIX

Aamodt, R., W. Rumble, S. O'Reilly, G. Johnston, and R. Henkin. 1975. Studies on the metabolism of ^{65}Zn in man. Fed. Proc. 34:922.

Andrasi, A., J. Feher. 1967. Measurement of the retention and excretion of incorporated ^{65}Zn. Health Phys. 13:915-6.

Bienvenu, P., C. Nofre, and A. Cier. 1963. Toxicité générale comparée des ions métalliques. Relations avec la classification périodique. Compt. Rend. Acad. Sci. Paris, 256(4):1043-44.

Birnstingl, M., B. Stone, and V. Richards. 1956. Excretion of radioactive (^{65}Zn) in bile, pancreatic and duodenal secretions of the dog. Am. J. Physiol. 186:377-79.

Bohne, F., N. Nigrovic, and A-E. Harmuth-Hoene. 1967. Metabolismus und toxizitat therapeutischer chelatbildner. 2. Mitteilung: Einflub von DTPA auf die enterale resorption von Zink. Strahlentherapie 134:293-5.

Caujolle, F., P.H. Chanh, N-L. T. Ngoc-Suong and P. Van Tô. 1969. Toxicology of zinc. I. Immediate and deferred toxicity and long-term toxicity. Agressologie 10(4): 333-9.

Chaube, S., H. Nishimura, and C.A. Swinyard. 1973. Zinc and cadmium in normal human embryos and fetuses: analysis by atomic absorption spectrophotometry. Arch. Environ. Health 26(5):237-40.

Creason, J.P., D. Svendsgaard, J. Bumgarnes, C. Pinkerton, and T. Hinners. 1976. Maternal-fetal tissue levels of 16 trace elements in 8 selected continental United States communities. Trace Subst. Environ. Health 10:53-62.

Dutkiewicz, T., E. Kulka, D. Sokolowska, and E. Woyciechowska. 1978. Evaluation of exposure of the human population to the effect of zinc, cadmium, and lead in the environment. Rocz. Panstw. Zakl. Hig. 29(3):299-304, as cited in CA 089/174614F.

Evans, G.W. and C.J. Hahn. 1974. Copper-and zinc-binding components in rat intestine. Advan. Exp. Med. Biol. 48:285-97.

Frazier, J.M. 1979. In vitro binding of cadmium, zinc, and copper to rat and human plasma proteins. Abs. Eighteenth Annual Meeting of Soc. of Toxicol., New Orleans, LA p A75.

Galin, M.A., H.D. Nano, and T. Hall. 1962. Ocular zinc concentration. Invest. Opthal. 1:142.

Gilbert, I.G.F. and D.M. Taylor. 1956. Behavior of zinc and radiozinc in rat. Biochim. Biophys. Acta 21:545.

Goolamali, S.K. and J.S. Comaish. 1975. Zinc and the skin. Int. J. Dermatol. 14(3):182-7.

Hahn, C. and G.W. Evans. 1973. Identification of a low molecular weight ^{65}Zn complex in rat intestine. Proc. Soc. Exp. Biol. Med. 144:793-95.

Halsted, J.A. and J.C. Smith, Jr. 1970. Plasma zinc in health and disease. Lancet 1:322-24.

Halsted, J.A., J.C. Smith, Jr., M.I. Irwin. 1974. A conspectus of research on zinc requirements of man. J. Nutr. 104(3):345-78.

Heath, J.C. and J. Liquier-Milward. 1950. Distribution and function of zinc in normal and malignant tissues. Biochim. Biophys. Acta. 5:404.

Henkin, R.I., J.R. Marshall, S. Meret. 1971. Maternal-fetal metabolism of copper and zinc at term. Am.J. Obstet. Gynecol. 110:131-4.

Kowarski, S., C.S. Balir-Stanek, and D. Schachter. 1974. Active transport of zinc and identification of zinc-binding protein in rat jejeunal mucosa. Amer. J. Physiol. 226:401-07.

Kumar, S. and K.S.J. Rao. 1973. Plasma and erythrocyte zinc levels in protein-calorie malnutrition. Nutr. Metab. 15:364-71.

Lindeman, R.D., R.G. Bottomly, R.C. Cornelison, and L.A. Jacobs. 1972. Influence of acute tissue injury on zinc metabolism in man. J. Lab. Clin. Med. 79:452-60.

Methfessel, A.H. and H. Spencer. 1973. Zinc metabolism in the rat. I. Intestinal absorption of zinc. J. Appl. Physiol. 34:58-62.

Montgomery, M.L., G.E. Sheline, and I.L. Chaikoff. 1943. The elimination of administered zinc in pancreatic juice, duodenal juice, and bile of the dog as measured by its radioactive isotope (^{65}Zn). J. Exptl. Med. 78:151-9.

National Academy of Sciences (NAS). 1978. Zinc, prepared by the Subcommittee on Zinc, Committee on Medical and Biologic Effects of Environmental Pollutants. National Research Council, National Academy of Sciences, Washington, D.C.

Oberleas, D. and A.S. Prasad. 1976. Factors affecting zinc homeostasis. Chapter 10 in: Trace Elements in Human Health and Disease, Vol. 1, A.S. Prasad, ed. Academic Press,New York.

Oehme, M., W. Lund, and J. Jonsen. 1978. The determination of copper, lead, cadmium, and zinc in human teeth by anodic stripping voltammetry. Anal. Chim. Acta. 100:389-98.

Parisi, A.F. and B.L. Vallee. 1970. Isolation of a zinc α_2-macroglobulin from human serum. Biochemistry 9:2421-6.

Perry, N.M., Jr., E.F. Perry, and B.B. Hixon. 1978. Trace-metal concentrations in normal human liver: Methods to cope with marked variability. Sci. Total. Environ. 9(2):125-34.

Prasad, A.S. 1966. Metabolism of zinc and its deficiency in human subjects. Chapter 15 in: Zinc Metabolism, Charles C. Thomas, Springfield, Il.

Prasad, A. 1979. Clinical, biochemical, and pharmacological role of zinc. Ann. Rev. Pharmacol. Toxicol. 20:393-426.

Prasad, A.S. and D. Oberleas. 1970. Binding of zinc to amino acids and serum proteins in vitro. J. Lab. Clin. Med. 76:416-25.

Prasad, A.S., A.R. Schulert, H.H. Sandstead, A. Miale, Jr., and Z. Farid. 1963b. Zinc, iron and nitrogen content of sweat in normal and deficient subjects. J. Lab. Clin. Med. 62:84-9.

Prasad, A.S., E.B. Schoomaker, J. Ortega, G.J. Brewer, O. Oberleas, and F.J. Oelshlegel, Jr. 1975. The role of zinc in man and its deficiency in sickle cell disease, in: Progress in Clinical and Biological Research. Vol. I. Erythrocyte Structure and Function, G.J. Brewer, ed. Alan R. Liss, New York, pp 603-19.

Richmond, C.R., J.E. Furchner, G.A. Trafton, and W.H. Langham. 1962. Comparative metabolism of radionuclides in mammals. I. Uptake and retention of orally administered ^{65}Zn by four mammalian species. Health Phys. 8:481-9.

RTECS, (Registry of Toxic Effects of Chemical Substances), Vol. II. 1977. E.J. Fairchild, R.J. Lewis, Sr., and R.L. Tatken, eds. U.S. DHEW, NIOSH, 78-104-B.

Schroeder, H.A., A.P. Nason, I.H. Tipton, and J.J. Balassa. 1967. Essential trace metals in man. Zinc: Relation to environmental cadmium. J. Chron. Dis. 20:179.

Schrodt, G.R., T. Hall, and W.F. Whitmore, Jr. 1964. The concentration of zinc in diseased human prostate glands. Cancer 17:1555-66.

Sheline, G.E., I.L. Chaikoff, H.B. Jones, and M.L. Montgomery. 1943. Studies on the metabolism of zinc with the aid of its radioactive isotope. I. The excretion of administered zinc in urine and feces. J. Biol. Chem. 147:409-14.

Smit, Z.M. and P.J. Pretorius. 1964. Studies in metabolism of zinc. Part 2. Serum zinc levels and urinary zinc excretions in South African Bantu Kwashiorkor patients. J. Trop. Pediatr. 9:105-12.

Song, M.K. and N.F. Adham. 1978. Role of prostaglandin E_2 in zinc absorption in the rat. Am. J. Physiol. 234:E99-E105.

Spencer, H., B. Rossof, A. Feldstein, S.H. Cohn and E. Gusmano. 1965. Metabolism of ^{65}Zn in man. Radiation Res. 24:432-45.

Spencer, H., D. Osis, L. Kramer, and C. Norris. 1976. Intake, excretion, and retention of zinc in man. Chapter 21 in: Trace Elements in Human Health and Disease, Vol. 1. A.S. Prasad, ed. Academic Press, New York.

Suso, F.A. and H.M. Edwards, Jr. 1971. Binding capacity of intestinal mucosa and blood plasma for zinc. Proc. Soc. Exp. Biol. Med. 137:306-09.

Syverson, T.L.M., T.K. Stray, G.B. Syverson, and J. Ofstad. 1976. Cadmium and zinc in human liver and kidney. Scand. J. Clin. Lab. Invest. 36(3): 251-6.

Thind, G.S. and G.M. Fischer. 1975. Cadmium and zinc distribution in cardiovascular and other tissues of normal and cadmium-treated dogs. Exp. Mol. Pathol. 22(3):326-34.

Tsuchiya, K. and S. Iwao. 1978. Interrelationships among zinc, copper, lead, and cadmium in food, feces, and organs of humans. Environ. Health Perspect. 25:119-24.

Underwood, E.J. 1971. Trace Elements in Human and Animal Nutrition, 3rd Edition, Academic Press, New York.

Van Campen, D.R. and T.J. Kowalski. 1971. Studies on zinc absorption: ^{65}Zn binding by homogenates of rat intestinal mucosa. Proc. Soc. Exp. Biol. Med. 136:294-97.

Wakely, J.C.N., B. Moffatt, A. Crook, and J.R. Mallard. 1960. The distribution and radiation dosimetry of ^{65}Zn in the rat. Intern. J. Appl. Rad. Isotopes. 7:225-32.

Wester, P.O. 1975. Letter: Zinc during diuretic treatment. Lancet 1(7906): 578, March 8.

Zazgornik, J., R. Kotzaurek, and P. Schmidt. 1971. Plasma zinc concentration in uremic patients during hemodialysis. Klin. Wochenschr. 49(5):278-80.