ENVIRONMENTAL ASSESSMENT OF THE DOMESTIC PRIMARY COPPER, LEAD AND ZINC INDUSTRIES

VOLUME II - APPENDICES

PEDCo ENVIRONMENTAL

PEDCo-ENVIRONMENTAL
SUITE 13 • ATKINSON SQUARE
CINCINNATI, OHIO 45246
513/771-4330

ENVIRONMENTAL ASSESSMENT OF THE DOMESTIC PRIMARY COPPER, LEAD AND ZINC INDUSTRIES

VOLUME II - APPENDICES

Prepared by

PEDCo-Environmental Specialists, Inc.
Suite 13, Atkinson Square
Cincinnati, Ohio 45246

Contract No. 68-02-1321
Task No. 38

EPA Project Officer
Margaret J. Stasikowski

Prepared for

U.S. ENVIRONMENTAL PROTECTION AGENCY
Industrial Environmental Research Laboratory
5555 Ridge Avenue
Cincinnati, Ohio 45268

November, 1976

BRANCH OFFICES

Suite 110, Crown Center
Kansas City, Mo. 64108

Suite 107-B Professional Village
Chapel Hill, N.C. 27514

DISCLAIMER

This report has been reviewed by the Industrial Environmental Research Laboratory, U.S. Environmental Protection Agency, and approved for publication. Approval does not signify that the contents 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.

ABSTRACT

This report presents the results of a multi-media (air, water, and solid waste) study of the environmental impacts from the U.S. primary copper, lead and zinc industries. The open literature was surveyed to identify and describe all processes employed by these industries and to characterize pollutant effluents and environmental effects from those processes. Various pollution control systems are described and evaluated for domestic application, and alternate production processes are reviewed.

Principal environmental impacts from the copper industry are air emissions of SO_2 and trace metals from smelting operations. Insufficient markets for by-product sulfur compounds are a disincentive for SO_2 control. Water pollution problems include acid drainage and trace metal contamination of wastewaters from ore beneficiation processes. The fact that many mines and smelters are located in remote arid regions tends to mitigate the severity of these pollution problems. Newer hydrometallurgical processes may produce leach residues that will require further efforts to control.

Lead is produced by six pyrometallurgical smelters in this country. There are large lead deposits in the State of Missouri so that a large percentage of future lead supplies are expected to originate in that state. Mining and concentrating operations produce metal-laden wastewaters that are effectively controlled in the Missouri area by biotic degradation and natural precipitation at high pH. Principal smelter emissions include SO_2 and particulates from sintering and blast furnace operations. Electrostatic precipitators, baghouses, and sulfuric acid plants are used for pollution control.

Zinc smelters are mainly located in populated areas and have largely been forced to clean up their emissions in recent years. Economic prospects have forced several smelters to close in recent years. SO_2 is controlled at all smelters by means of acid plants. Particulates are controlled by electrostatic precipitators and baghouses. There is some concern that volatile metals may pass through these control devices in the vapor phase.

Several programs for further research and development are indicated.

CONTENTS

CONTENTS (Continued)

CONTENTS (Continued)

LIST OF FIGURES

LIST OF TABLES

LIST OF TABLES (continued)

LIST OF TABLES (continued)

LIST OF TABLES (continued)

LIST OF TABLES (continued)

LIST OF TABLES (continued)

ACKNOWLEDGMENT

This report was prepared by PEDCo-Environmental Specialists, Inc., under the direction of Mr. Timothy W. Devitt. Principal authors were Dr. Gerald A. Isaacs, Mr. Thomas K. Corwin, Mr. Hal M. Drake, Mr. Douglas J. Morell, and Mr. Jeffrey A. Smith.

Project officer for the U.S. Environmental Protection Agency was Ms. Margaret J. Stasikowski.

The authors appreciate the efforts and cooperation of everyone who participated in the preparation of this report.

APPENDIX A

HEALTH EFFECTS OF PRIMARY COPPER, LEAD AND ZINC SMELTING

1.0 SELECT REVIEW OF PUBLISHED TOXICOLOGY AND EPIDEMIOLOGY

TOXICOLOGY OF METALS

In order to realistically assess the toxic potential of residual metals emitted from smelters or other sources, several general concepts must be appreciated. Smelter residual metal emissions differ widely in both composition and quantities of individual metals. Although the toxic properties of each individual metal within an emission stream may be known, the combined toxic effect of the stream is not necessarily the simple sum of the effects of the components. Some elements such as Ca and Zn seem to mitigate the toxic effects of other metals such as Cd and Pb, while other combinations of metals probably produce greater than additive adverse effects. Another dilemma is encountered when considering carcinogenic or teratogenic effects of metals. Although occupational exposure limits for carcinogens such as As, Ni and Cr and teratogens such as Cd and Se have been established, no one really knows what concentration of a given carcinogen or teratogen is safe. With these considerations in mind, the toxic potentials of individual metals are discussed, and supposed safe levels of exposure to carcinogens are presented.

Summary of the Toxicity of Metals

Table 1-1 lists of types of effects various metals would be expected to produce. Metals indicated as possible "Factors in Environmental Nonoccupational Disease" are generally those which tend to accumulate in animal tissue. As shown in Table 1-1, most metals which are probable factors in environmental disease are also moderate to severe industrial hazards. Another significant point illustrated by the table is that some of the metals which play a role in environmental disease are also essential elements for mammals. As is generally true for most substances, metal toxicity is dependent upon concentration and length of exposure. Thus, Table 1-1 reflects current knowledge concerning the quantities and concentrations of metals usually encountered.

As discussed in the introduction to this section, typical sources of metal emissions do not emit only one element, and the effects of a combination of metals in an emissions stream can only very rarely be predicted. For this reason, one of the most important aspects of environmental toxicology is to indicate what types of disease may be caused by complex combinations of metals so that epidemiological inves-

Table 1-1. A CLASSIFICATION OF THE EFFECTS OF METALS[1]

Metal	Essential For Mammals	Moderate to Severe Industrial Hazard	Factors in Environmental Nonoccupational Disease	Accidental Poisoning	Limited Industrial Hazard
Aluminum					+
Antimony		+		+	
Arsenic		+	+	+	
Barium					+
Beryllium		+	+		
Bismuth					+
Boranes					
Cadmium		+	+	+	
Cesium					+
Chromium (III)	+				
Chromium (VI)		+			
Cobalt	+		+		
Copper	+			+	+
Gallium					+
Germanium					+
Gold					+
Hafnium					+
Indium					+
Iridium					+
Iron	+		+	+	+
Lanthanons					+
Lead		+	+	+	
Magnesium	+				+
Manganese	+	+			
Mercury		+	+	+	
Metal hydrides and Carbonyls		+			
Molybdenum	+				+
Nickel		+	+		
Niobium					+
Palladium					+
Platinum		+			
Rhenium					+
Rubidium					+
Selenium	+	+	+		
Silver		+			
Strontium	+				+
Tantalum				+	+
Tellurium				+	+
Thallium		+			
Tin (organic)		+			
Titanium					+
Tungsten					?
Uranium		+			
Vanadium	+	+	?		
Zinc	+			?	+
Zirconium					+

tigations will have a starting point. Table 1-2 lists the target organs of individual metals. Knowledge of which organs are affected by which metals is essential for the success of any epidemiological analyses of sources emitting residual metals.

Maximum allowable concentrations for metals in both workroom air and drinking water have been established and are listed in Tables 1-3 and 1-4. It must be emphasized that these values represent probable safe values only in the absence of all other toxic substances. For example, a worker exposed to 500 μg As/m^3, 200 $\mu g/m^3$ Cd dust, 1000 μg Ni/m^3, 500 μg Sb/m^3 and 500 $\mu g/m^3$ V_2O_5 dust simultaneously would probably develop respiratory disease if exposure continued long enough. Each one of those respiratory disease producing metals at its maximum allowable concentration (Table 1-3) would probably be safe (disregarding carcinogenic potentials), but all five present together at their maximum allowable concentrations probably would not. Values listed in Table 1-3 do not apply to 24 hour/day - 7 day/week exposures, and even values lowered proportionately to the increased exposure time may not be sufficient to protect the public health. Although the exposure limits summarized in Tables 1-3 and 1-4 have severe limitations when considering the environmental toxicology of metals, an idea of relative toxicity may be safely extracted. For example, from Table 1-3, it is clear that molybdenum is less toxic than mercury.

In Section 2.0, the associations between Cu, Pb and Zn smelters and increases in mortality from cancers is presented. It is now apparent that As, Ni and Cr cause respiratory cancers in man, but at this point, human cancer of the liver, kidney, bladder and thyroid have not been linked to metals. However, animal studies have linked many other malignant neoplasms besides respiratory cancers to metal exposure, and the associations presented in Section 2.0 link Cu, Pb and Zn smelting to many cancers as well as respiratory ones. For these reasons, the carcinogenic potential of metals upon experimental animals is detailed in Table 1-5. Similarly, the teratogenic potential of metals upon animals is presented in Table 1-6. Even though sufficient statistical data to adequately evaluate the teratogenic associations of smelters is lacking, the large number of associations between cancers and smelters would indicate that metals emitted from Cu, Pb and Zn smelters may be linked with human teratogenesis.

As a final note, it should be kept in mind that environmental exposure to metals from smelters of some other facility is not the sole source of metal intake. Metals are contained in food, water and are normal components of the earth's crust. Table 1-7 lists typical human intake and body burden of metals as well as natural abundance of the various metals.

Table 1-2. TARGET ORGANS OF METALS[1]

Metals	Gastro-Intestinal Tract	Respiratory Tract	CNS	Cardio-Vascular System	Liver	Skin	Blood	Kidney	Bone	Endocrine
Aluminum		+								
Antimony	+	+		+	+	+				
Arsenic	+	+	+		+	+	+			+
Barium	+	+	+	+		+				
Beryllium		+				+			+	
Bismuth					+	+		+		
Boranes		+			+			+		
Boron	+		+							
Cadmium	+	+	+	+				+	+	
Chromium		+	+		+	+		+		
Cobalt	+	+	+	+		+				+
Copper							+			
Gallium			+			+		+	+	
Germanium	+	+		+						
Gold					+	+	+	+		
Hafnium					+	+				
Indium			+		+			+		
Iron	+	+	+		+		+			+
Lanthanons		+			+		+			
Lead	+		+				+	+		
Lithium	+		+	+				+		+
Magnesium			+							
Manganese		+	+							
Mercury		+	+					+		
Metal hydrides							+			
Molybdenum					+		+	+	+	
Nickel		+	+			+				
Niobium					+			+		
Osmium		+								
Palladium							+			
Platinum		+		+		+				
Rhodium			+							
Rubidium			+	+						
Ruthenium		+								
Selenium	+		+		+	+				
Silver		+				+		+		
Strontium				+						
Tantalum		+								
Tellurium			+		+			+		
Thallium	+	+	+		+			+		+
Tin (organic)	+		+							
Titanium		+								
Tungsten			+							
Uranium								+		
Vanadium		+	+			+		+		
Zinc	+						+		+	
Zirconium						+	+			

Table 1-3. ACCEPTABLE AVERAGE CONCENTRATIONS ($\mu g/M^3$) OF OCCUPATIONAL EXPOSURE BASED ON 8-HOUR EXPOSURES*[1]

Antimony and compounds (as Sb)	500	
Stibine (SbH_3)	500	(0.1 ppm)
Arsenic and compounds (as As)	500	
Arsine (AsH_3)	200	(0.05 ppm)
Arsenate, calcium	1,000	
Arsenate, lead	150	
Barium (soluble compounds)	500	
Beryllium and compounds	2	(5†)
Boron oxide	15,000	
Boron trifluoride	3,000†	
Diborane	100	
Pentaborane		(0.005 ppm)
Decaborane (skin)	300	
Cadmium fume	100	(3000†)
Cadmium dust	200	(600†)
Chromic acid and chromates	100†	
Chromium, soluble salts	500	
Chromium, metal and insoluble salts	1,000	
Cobalt, metal fume and dust	100	
Copper fume	100	
Copper, dusts and mists	1,000	
Hafnium	500	
Iron oxide fume	10,000	
Ferban (ferric dimethyldithiocarbamate)	15,000	
Ferrovanadium dust (FeV)	1,000	
Lead and its inorganic compounds	200	
Lead arsenate	150	
Lead, tetraethyl (as Pb-skin)	75	
Lead, tetramethyl (as Pb-skin)	75	
Lithium hydride	25	
Magnesium oxide fume	15,000	
Manganese	5,000†	
Mercury	100†	
Mercury (organo alkyl)	10	(40†)
Molybdenum (soluble compounds)	5,000	
Molybdenum (insoluble compounds)	15,000	
Nickel, metal and soluble compounds as Ni	1,000	
Nickel carbonyl	7	
Osmium tetroxide	2	
Silver, metals and soluble compounds	10	
Tantalum	5,000	
Tellurium	100	
Tellurium hexafluoride	200	(0.02 ppm)
Thallium (soluble compound:-skin as Tl)	100	
Tin (inorganic compounds except oxides)	2,000	
Tin (organic compounds)	100	
Platinum (soluble salts as Pt)	2	
Rhodium, metal fume and dust as Rh	100	
Rhodium (soluble salts)	1	
Selenium compounds as Se	20	
Selenium, hexafluoride	400	(0.05 ppm)
Titanium dioxide	15,000	
Uranium (soluble compounds)	50	
Uranium (insoluble compounds)	250	
Vanadium (V_2O_5 dust)	500†	
Vanadium (V_2O_5 fume)	100†	
Yttrium	1,000	
Zinc chloride fume	1,000	
Zinc oxide fume	5,000	
Zirconium compounds as Zr	5,000	

*From Federal Register, Vol. 36, No. 157; Friday, Aug. 13, 1971.

†Ceilings.

Table 1-4. TOLERANCE LEVELS FOR METALS IN DRINKING WATER AND RESULTS OF SAMPLING OF COMMUNITY WATER SUPPLIES (969) IN 1969*[1]

Element	LIMITS IN mg/liter		Maximum Concentrations Found	NUMBER OF SAMPLES OF A TOTAL OF 2595 EXCEEDING	
	Mandatory Upper	Desirable Upper		Mandatory	Desirable
Arsenic	0.05	0.01	0.10	5	10
Barium	1.0		1.55	2††	
Boron	5.0	1.0	3.28	0	20
Cadmium	0.01		3.94	4	
Chromium (Cr^{6+})	0.05		0.79§	5	
Copper		1.0	8.35		42
Iron		0.3	26.0		223
Lead	0.05		0.64	37	
Manganese		0.05	1.32		211
Selenium	0.01		0.07	10	
Silver	0.05		0.03	0	
Uranium (Uranyl)†		5.0	Not included		
Zinc		5.0	13.0		8

*From U.S. Public Health Service: Community Water Supply Study: Analysis of Nation Survey Findings. U.S. Department of Health, Education, and Welfare, Washington, D.C., 1970.

†Proposed.

††Not measured in all samples.

§Total Chromium measured.

Table 1-5. METAL CARCINOGENESIS IN EXPERIMENTAL ANIMALS*[1]

Metal	Compound	Species	Route	Type of Tumor
Beryllium	$AnBeSiO_3$, BeO	Rabbits, mice, rats	IV	Osteosarcomas
	BeO, $BeSO_4$, $BeHPO_4$	Monkeys, rats	Inhalation	Pulmonary carcinomas
Cadmium	CdS, CdO, $CdCl_2$, $CdSO_4$, Cd powder	Rats, mice	SC, IM	Sarcomas Leydigiomas
	$CdCl_2$	Chicken	Intratesticular	Teratoma
Chromium	Metallic Cr	Rabbits	Intraosséous	Sarcomas
	Roasted chromite ore	Rats, mice	IM, IP, SC	Sarcomas
	$CaCrO_4$, CrO_3, $Na_2Cr_2O_7$ Cr_2O_3		Intrapleural	Squamous Cell carcinomas
	$CaCrO_4$	Rats	Intrabronchial	Squamous cell and adeno-carcinomas
Cobalt	Metallic Co, Co powder CoO, CoS	Rats, rabbits	SC, Im Intraosseous	Sarcomas
Copper	$CuSO_4$	Chickens	Intratesticular	Teratoma
Iron	Iron-carbohydrate complexes	Rats, mice, rabbits	IM, SC	Sarcomas
Lead	$Pb_3(PO_4)_2$, $Pb(C_2H_3O_2)_2$	Rats	SC	Renal adenomas and carcin-omas
	Tetraethyl lead	Mice	SC	Lymphomas
	$Pb(C_2H_3O_2)_2$, $2Pb(OH)_2$	Rats, mice	Dietary	Renal adenomas Renal and testicular carcinomas
Nickel	Ni dust, $Ni(CO)_4$	Guinea pigs, rats	Inhalation	Anaplastic and adeno-carcinomas Squamous cell, anaplastic and adenocarcinomas
	Nickelocene, Ni dust Ni_3S_2 dust, NiO dust Ni pellets	Rats	IM, SC	Sarcomas
	Ni_3S_2 discs	Cats	Implantation in nasal sinuses	Squamous cell and adeno-carcinomas
Selenium	NH_4KSe, grain with Se, Na_2, SeO_4, bis-4-acet-aminophenyl Sehydroxide	Rats	Dietary	Hepatomas Sarcomas Thyroid adenomas
Zinc	$ZnCl_2$	Rats	Intratesticular	Leydigiomas, serminoma
	$ZnSO_4$, $ZnCl_2$	Chickens	Intratesticular	Chorionepithelioma Teratomas
Titanium	Titanocene	Rats, mice	IM	Fibrosarcomas, hepatomas Lymphomas
Aluminum	Al foil	Rats	Implantation	Sarcomas
Silver	Ag foil	Rats	Implantation	Fibrosarcomas
	Ag colloid	Rats	IV	Tumors (?)
Mercury	Liquid mercury	Rats	IP	Spindle cell sarcomas

*Data from Furst and Haro, 1969; Sunderman, 1971.

Table 1-6. EFFECTS OF METALS ON REPRODUCTION*[1]

Metal	Species	Test	Results
Arsenic	Mouse	5 ppm arsenite in drinking water, 3 generations	Increased male to female ratio, reduced litter size
	Hamster, rat	Teratogenic parenteral	Head changes, exencephaly, urogenital abnormalities
Cadmium	Mouse	10 ppm in drinking water, 3 generations	Failure to reproduce 3 generations, congenital abnormality of the tail, runting, death before weaning
	Hamster	Teratogenic parenteral	Abnormalities face and palate
Cobalt	Hamster	Teratogenic	Not teratogenic
Copper	Hamster	Teratogenic	Not teratogenic
Indium	Hamster	Teratogenic parenteral	Abnormalities of limb buds
Lead	Mouse	25 ppm in drinking water, 3 generations	Failure to reproduce 3 generations, runting, death before weaning
	Rat	25 ppm in drinking water, 3 generations	Death before weaning, runting
	Hamster	Teratogenic parenteral	Malformation tail bud
Lithium	Mouse	Teratogenic	Resorption, cleft palate
Manganese	Hamster	Teratogenic	Not teratogenic, embryocidal
Mercury	Human	Epidemiologic	Mental retardaion, neuromuscular effects
	Mouse, rat	Teratogenic (methyl mercury)	Behavior effects, CNS changes
	Hamster	Teratogenic (mercuric acetate and phenylmercuric acetate)	No clear-cut effects
Molybdenum	Mouse	10 ppm (molybdate) in drinking water, 3 generations	Deaths before weaning, runting
	Hamster	Teratogenic	Not teratogenic, embryocidal
Nickel	Rat	5 ppm in drinking water, 3 generations	Death before weaning, runting, reduced litter size, reduced number of males in third generation
	Hamster	Teratogenic parenteral	Embryotoxic, few general malformations
Selenium	Livestock	Epidemiologic	Teratogenic
	Hamster	Teratogenic	Not teratogenic
	Mouse	3 ppm (selenate) in drinking water, 3 generations	Increased male to female ratios, death before weaning, runting
Tellurium	Rat	Teratogenic (dietary 500 to 3500 ppm)	Hydrocephalus
Titanium	Rat	5 ppm (titanate) in drinking water, 3 generations	Runting, death before weaning, male to female ratio reduced
Zinc	Hamster	Teratogenic parenteral	Mild teratogenic effect
	Rat	Dietary administration dam	Increased hydrocephalus

*Data largely from Schroeder and Mitchner, 1971b; Ferm, 1972

Table 1-7. BODY BURDEN AND HUMAN DAILY INTAKE AND CONTENT IN THE EARTH'S CRUST OF SELECTED ELEMENTS*[1]

Element	Human Body Burden (mg/70 kg)	Daily Intake (mg)	Earth's Crust (ppm)
Aluminum	100	36.4	81,000
Antimony	<90		0.2
Arsenic	<100	0.7	2
Barium	16	16	400
Boron	<10	0.01-0.02	16
Cadmium	30	0.018-0.20	0.2
Calcium	1,050,000		36,000
Cesium	<0.01		1
Chromium	<6	0.06	200
Cobalt	1	0.3	23
Copper	100	3.2	45
Germanium	trace	1.5	1
Gold	<1		0.005
Iron	4,100	15	50,000
Lead	120	0.3	15
Lithium	trace	2	30
Magnesium	20,000	500	20,900
Manganese	20	5	1,000
Mercury	trace	0.02	0.5
Molybdenum	9	0.35	1
Nickel	<10	0.45	80
Niobium	100	0.60	24
Potassium	140,000		25,900
Rubidium	1,200	10	120
Selenium	15	0.06-0.15	0.09
Silver	<1		0.1
Sodium	105,000		28,300
Strontium	140	2	450
Tellurium	600	0.6	0.002
Tin	30	17	3
Titanium	<15	0.3	4,400
Uranium	0.02		2
Vanadium	30	2.5	110
Zinc	2,300	12	65
Zirconium	250	3.5	70

*Data largely from Schroeder, 1965a.

Toxicology of the Major Residual Metals Emitted from Primary Cu, Pb and Zn Smelters[1,2,3,4]

Antimony - Antimony compounds are absorbed slowly from the gastrointestinal tract and tend to produce vomiting. Trivalent forms accumulate in the liver and are mainly excreted in the feces, while pentavalent forms tend to concentrate in the liver and spleen and are excreted in the urine. Acute human poisoning produces symptoms similar to those of arsenic poisoning, namely vomiting, watery diarrhea, collapse, irregular respiration and hypothermia. Chronic administration of potassium antimony tartrate (5 ppm) in drinking water to rats increased the mortality rates and decreased serum glucose levels. Toxicity data derived in connection with the therapeutic use of antimonials include cardiac effects, in a few cases atrial fibrillation due to a direct effect on the heart and death, liver toxicity, characterized by jaundice and fatty degeneration, pulmonary congestion and edema, and papular skin eruptions. Indications of industrial antimony poisoning include upper respiratory tract irritation, pneumonitis, dizziness, diarrhea, vomiting, and dermatitis. Antimony may generate stibine (antimony hydride) under reducing conditions. The lethal concentration of stibine in air for mice is about 100 ppm for 1.6 hours, and stibine may be expected like arsine to cause rapid hemolysis, hemoglobinuria, and anuria.

Dernehl and his colleagues (1945) quote Feil who, in 1939, found among workers in antimony smelters the following symptons: conjunctivitis, tracheitis, pharyngitis, anemia, headache, anorexia, and vomiting. Feil ruled out arsenic as the cause since the ore contained less than 10 mg per kilogram. Taylor (1966) described acute antimony trichloride intoxication causing gastrointestinal symptoms with persistent nausea and respiratory tract irritation. In 1953 Renes reported 69 cases of illness among workers in an antimony smelter where they were exposed to dust and fume at high levels (5 to 12 mg/m^3); particle size was less than one micron allowing rapid absorption. Since the arsenic concentrations in this exposure were low, the toxic effect was attributed to antimony. Inflammation of the skin and upper respiratory tract occurred most frequently, and in six cases an acute pneumonitis was demonstrated by chest x-ray and was completely reversed by penicillin and freedom from exposure.[2]

A serious toxic effect of exposure to antimony as the compound Sb_2S_3 was reported by Brieger et al. in 1954. He studied 125 workers who had been employed in the abrasive industry for less than two years. Of these, 6 died suddenly of heart failure, and 37 of 75 examined showed abnormal electrocardiograms (EKG's). The chief abnormality was found in T wave changes. The use of antimony had to be stopped as values of 3 to 5.5 mg/m^3 of antimony were found in the air and up to 9.6 mg/L in urine, in spite of engineering control.[2]

Arsenic - Arsenic compounds may be absorbed by an oral or inhalation route with trivalent compounds usually more toxic than pentavelant ones. Arsenate is the valence form most prevalent in nature and in this form tends to be rapidly excreted by the kidneys, thus, probably not accumulating. Arsenites bind to proteins and are concentrated in leukocytes. In the body, they accumulate primarily in the liver, muscles, hair, nails and skin. Arsenic is both teratogenic and embryotoxic for mice; a level of 10 mg/Kg sodium arsenite was sufficient to produce these effects, while 25 mg/Kg sodium arsenate was without effect.[1] Similarly, no dogs fed arsenic (125 ppm) as arsenite survived a two year study, but only one dog died when fed the same level of arsenic as arsenate.

Symptoms of acute inorganic arsenic poisoning include burning and dryness of the oral and nasal cavities, gastrointestinal disturbance, and muscle spasms; vertigo, delirium and coma may also occur. Chronic arsenic intoxication initially produces malaise and fatigue and may ultimately cause gastrointestinal disturbances, hyperpigmentation, peripheral neuropathy, anemia and leukopenia. Industrial poisoning generally follows the same pattern, although skin changes such as nasal septum ulceration may occur more frequently than the hematologic changes. Epidemiological studies link arsenic by industrial exposures to skin and lung cancer and by oral intake with goitrogenesis and blackfoot disease, a severe form of peripheral arteriosclerosis in Taiwan. Arsine, one of the most toxic arsenic compounds, is the principal source of industrial arsenic poisoning today and has been reported in connection with the refining or processing of tin, lead and zinc. Arsine, a gas with a slight garliclike odor detected only above safe levels, produces massive hemolysis and renal failure; exposures of as low as 10 ppm have been lethal in humans.

Bismuth - Most bismuth compounds commonly encountered are insoluble and poorly absorbed. There have been no reports of industrial poisoning from bismuth, and the bismuth compounds do not present a hazard by dermal application, inhalation or ingestion. Bismuth administered therapeutically is concentrated priamrily in the kidney and to a lesser extent in the liver. Urine is the major route of excretion. Symptoms of chronic bismuth toxicity in man include anorexia, weakness, rheumatic pain, diarrhea, metal line on the gums, foul breath, gingivitis and dermatitis. Although rare, nephritis signaled by albuminuria, jaundice and conjunctival hemorrhage have been reported.

Cadmium - In biologic systems, the metabolism of zinc and cadmium are closely related, so much so that cadmium can be considered an antimetabolite of zinc. About 6 to 10 percent of ingested cadmium is absorbed, and most accumulation occurs in the kidney except in cases of

large exposures where the liver may contain more. Inhaled cadmium dusts and fumes have retention rates from 5 to 50 percent and have a half-life in the lungs of about 5 days. Inhaled cadmium is distributed to other parts of the body probably through the action of the carrier protein metallothionen. Normal cadmium blood levels are below 1 μg/100 ml and normal urinary cadmium values range from 0.5 to 11 μg/l. The urine is the primary route for cadmium excretion, but hair loss and fecal excretion also contribute.

The toxic effects of cadmium are fairly well documented, and some toxic levels of exposure are listed in Table 1-8. Large oral doses of cadmium produce vomiting, while acute inhalation exposures may result in pulmonary edema and death by anoxia within 3 days. The second stage of acute intoxication consists of cellular proliferation in the alveoli as well as hyperplasia of the lining cells sometimes accompanied by intra-alveolar hemorrhage. Delayed lung effects which may occur are perivascular and peribronchial fibrosis along with emphysema. Lethal levels of cadmium exposure are estimated to be 50 mg Cd/m^3 for one hour for cadmium oxide dust and about one-half that for the fume. Cadmium is also teratogenic for hamsters if administered during the proper gestational period, and simultaneous administration of zinc can greatly mitigate these teratogenic effects.[5]

Chronic cadmium inhalation exposure may result in proteinuria and emphysema, differing from chronic bronchial emphysema in that it is not preceded by a history of bronchitis and coughing. Chronic oral cadmium poisoning can also drastically alter human calcium and zinc metabolism as exemplified by the outbreak of Itai-itai (ouch-ouch) disease in the Toyama Bay area of Japan. The disease occurred most frequently in postmenopausal women who has histories of several pregnancies. Lumbar and leg pain were characteristic. Skeletal deformities with markedly decreased body height, susceptibility to multiple fractures after slight physical stress such as coughing, and waddling gait caused by bone deformities may precede complete inability to walk. The abnormal clinical pathologic parameters produced in this disease are proteinuria, glucosuria, reduced urinary phosphate, low serum inorganic phosphorus and calcium, increased serum alkaline phosphatase, urinary amino acids, and decreased urinary and blood zinc levels. Additional symptoms and signs of chronic cadmium poisoning attributed to industrial exposures are anosmia, watery nasal discharges, loss of weight, low hemoglobin values, bone fractures and rickets.

Cadmium (5 ppm) added to the drinking water of rats or mice resulted in a high tumor incidence, shortened life-span, hepatic cirrhosis with fatty degeneration, arteriosclerosis of the kidney and heart, proteinuria, neurologic disorders, severe vestibular disturbances,

Table 1-8. SOME TOXIC LEVELS OF CADMIUM EXPOSURE[3]

Species	Route	Concentration	Time	Effects
Man	Oral (insoluble salts)	> 15 mg	Single dose	Vomiting
Man	Inhalation (CdO fume)	0.5-2.5 mg/m^3	Intermittent 2-3 days	Pneumonitis
Man	Inhalation (occupational exposure) (CdO fume and dust)	Unknown but > 0.2 mg/m^3	Chronic (many years)	Emphysema Mild anemia Proteinuria
Rats	Oral (drinking water)	10-50 ppm	1 year	Anemia and reduced growth rate
Rats	Oral (diet)	135 ppm	6 months	Anemia and poor growth
Dogs	Oral (drinking water)	5-10 ppm	4 years	No significant toxic effects

hypertension and left ventricular hypertrophy. It is interesting to note that hypertension induced in female rats by cadmium exposure can, as can many other effects, be reversed by the administration of zinc. Although the kidney is frequently the target organ in occupational diseases related to cadmium exposure, there is some eivdence that acute industrial exposure can cause myocardial infarction and cor pulmonale without necessarily influencing the kidney.

Cobalt - Cobalt salts are generally well absorbed orally, but large doses usually do not significantly accumulate. Of the ingested cobalt, about 80 percent is excreted in the urine and another 15 percent is eliminated in the feces. Cobalt which does accumulate does so in the liver, heart and hair. The normal levels in human urine and blood are about 98 and 0.18 µg/l, respectively. Ingestion of large amounts of cobalt can produce polycythemia, and goiter when taken chronically. Adverse reported effects from cobalt taken therapeutically include vomiting, diarrhea, and a sensation of warmth, flushing of the face, hypertension, slowed respiration, giddiness, tinnitus and deafness. Cardiomyopathy has been caused by ingestion of beer containing 1 ppm cobalt; why such a small amount of cobalt could produce such effects in the course of only one month is not known. Industrial exposure to cobalt salts leads to respiratory effects, although there is some question as to whether cobalt is the sole agent responsible for these effects. Skin and eye lesions similar to allergic dermatitis have also been reported following industrial exposures.

Copper - Absorption of copper is regulated by the intestinal mucosa; absorbed copper is initially bound to albumin and finally to ceruloplasmin. Excretion of excess copper by the bile usually maintains serum copper at between 120 to 145 µg/l. Excess copper is normally deposited in the liver and in bone. Man is not very sensitive to copper, consequently, it is felt that excessive copper exposure in normal persons does not result in any chronic disease. Industrial exposure to copper appears to only be responsible for metal fume fever. Acute poisoning resulting from ingestion of large amounts of copper salts produce vomiting, hematemesis, hypotension, melena, jaundice, coma and sometimes death. Hemolytic anemias have also been induced by using topical copper - containing compounds for burns and copper - containing dialysis equipment.

Germanium - Sodium germanite is rapidly absorbed from the gastrointestinal tract; and germanium levels of 0.65 and 0.29 µg/ml have been reported as normal values for erythrocytes and serum, respectively. The urine is the major route of germanium excretion (normal range of urinary germanium may be 0.40 to 2.16 µg/ml), but milk and feces are secondary routes. Germanium and its compounds have a low toxicity, and exposure

to germanium is not considered an industrial hazard or a cause of chronic disease in man. The acute effects of high doses of germanium administered to animals are hypothermia, listlessness, diarrhea, respiratory and cardiac depression, edema, and hemorrhage in the lungs and gastrointestinal tract.

Indium - Indium is poorly absorbed from the gastrointestinal tract and is excreted in the urine and feces. No industrial injury has been reported from the use of indium, and human absorption from the use of indium plated silver in utensils is without known toxic effect. Subcutaneous and intravenous injection of indium is followed by hindleg paralysis, convulsions and death. Autopsies of such animals revealed necrotic and hemorrhagic livers and kidneys and muscle degeneration.

Lead - The average daily intake of lead depends upon the source of food, but a good estimate is about 300 µg/day. Children, however, may have a greater relative lead intake due to higher metabolic needs per kilogram of body weight. Absorption of lead from the gastrointestinal tract appears to be regulated to some extent by the mechanism controlling calcium and phosphorous absorption, and under normal conditions, 5 to 10 percent of ingested lead is absorbed. Although calcium antagonizes lead absorption, at levels of ingested lead above 20 mg, significant quantities are not eliminated. Inhalation absorption of lead from the atmosphere is dependent upon normal physiologic parameters, but as an average about 37 percent of inhaled lead is retained within the lungs. From long-term studies, it appears that fecal and urinary excretion play equally important roles in eliminating the body lead burden. Over 90 percent of the lead body burden is stored in bone, but the aorta, liver, kidney, pancreas and lungs have higher lead concentrations than bone. Urinary lead levels of 150 µg/100 ml and blood levels of 80 µg/100 ml are indicative of hazardous exposure.

A great deal is known about the toxic effects of lead on humans, and the amount of lead which needs to be absorbed before such effects occur. The various effects and sequelae of lead intoxication are summarized in Tables 1-9 and 1-10. Symptoms of acute lead poisoning are thirst, a metallic taste, nausea, vomiting, black stools and circulatory collapse. Several days following acute lead poisoning muscular weakness, acute hemolytic crisis and kidney damage may occur preceding death. Colic, one of the most common symptoms, constipation, and weakness in the upper limb, wrists and fingers are the earliest symptoms of chronic lead poisoning. Lead has a devastating effect upon red blood cells. Impairment of the enzymes delta aminolevulinic acid synthetase and dehydrase, coproporphyrinogen oxidase, and heme synthetase along with lead's direct hemolytic action are responsible for the anemia of plumbism. Severe and permanent kidney, central nervous system and

Table 1-9. EFFECTS OF INORGANIC LEAD SALTS IN RELATION TO ABSORPTION[3]

(a) Classification of Levels and Types of Effects

Types of Effects:	1 No demonstrable in vivo effect	2 Minimal Subclinical metabolic effect	3 Compensatory biologic mechanisms invoked	4 Acute lead poisoning		5 Late effects of chronic or recurrent acute lead poisoning
				Mild	Severe	
A. Metabolic: (accumulation and excretion of heme precursors)	normal	slight increase in urinary ALA may be present	progressive increase in ALA, UCP, FEP	increased 5 to 100 fold		increased if excessive exposure recent, but may not be increased if excessive exposure remote
B. Functional Injury:						
1. Hematopoiesis	none	none	shortened RBC life span, reticulocytosis (+) (reversible)	shortened RBC life span and reticulocytosis with or without anemia (reversible)		Anemia (+) (reversible)
2. Kidney (renal tubular function)	none	none	?	aminoaciduria, glycosuria (+) (reversible)	Fanconi syndrome (reversible)	chronic nephropathy (permanent)
3. Central nervous system	none	none	?	mild injury (??? reversible)	severe injury (permanent)	severe injury (permanent)
4. Peripheral nerves	none	none	?	rare	rare	Impaired conduction (wrist, foot drop usually improve slowly, but may be permanent)

Table 1-9 (continued). EFFECTS OF INORGANIC LEAD SALTS IN RELATION TO ABSORPTION[3]

	(a) Classification of Levels and Types of Effects				
	1	2	3	4	
	No demonstrable in vivo effect	Minimal Subclinical metabolic effect	Compensatory biologic mechanisms invoked	Acute lead poisoning	Late effects of chronic or recurrent acute lead poisoning
				Mild / Severe	
Types of Effects:					
C. Clinical Effects:	none	none	Non-specific mild symptoms (may be due in part to co-existing diseases) colic, irritability, vomiting	ataxia, stupor, coma, convulsions	mental deficiency (may be profound) seizure disorder renal insufficiency (gout) (permanent)

	(b) Index of Recent or Current Absorption by Level of Effect				
Blood Level (µg Pb/100 gm whole blood)	< 40 µg Pb	40-60 µg Pb	Level III. 60----100 + µg Pb Level IV with anemia, intercurrent disease. 60----100 + µg Pb Level IV. > 80 µg Pb		may be normal
Urine Lead (adults only) (µg Pb/L)		< 80 µg Pb/L	< 130 µg Pb/L	> 130 µg Pb/L (may be less in severe illness)	endogenous excretion may be normal CaEDTA mobilization test: chronic nephropathy-positive permanent CNS injury ± (see text)

Pb=lead; ALAD=σ-aminolevulinic acid dehydratase; ALA=σ-aminolevulinic acid; UCP=urinary coproporphyrin; FEP=free erythrocyte protoporphyrin; RBC=red blood cell.

Table 1-10. DISTRIBUTION OF SEQUELAE FOLLOWING VARIOUS MODES OF ONSET IN 425 PATIENTS WITH PLUMBISM[3]

Sequelae	Mode of Onset						
	Enceph.	Seizures	Ataxia	G.I.	Febrile	Asympto-matic	Total
None	11(18)	14(33)	7(41)	159(69)	13(81)	53(91)	257(61)
Mental retardation	23(38)	14(33)	5(29)	43(19)	3(19)	5(9)	93(22)
Seizures	32(54)	17(39)	6(35)	30(13)	0	0	85(20)
Cerebral palsy	8(13)	0	1(6)	0	0	0	9(2)
Optic atrophy	4(6)	0	1(6)	0	0	0	5(1)
All cases	59	43	17	232	16	58	425

Data in parentheses are percentages of cases with that mode of onset developing the sequelae. The percentages in any column may total more than 100 because the one patient may develop more than one sequela.

peripheral nerve damage are well documented in chronic lead poisoning. Other reported effects of chronic lead poisoning are a reduction in iodine uptake by the thyroid, a decrease in pulmonary resistance to infectious disease when inhaled, and the induction of renal cancer in rats. Lead has also been associated with sterility, abortion, still births, and neonatal deaths in man. Although the toxicity of organic lead is interesting, it is not relevant to lead emitted from copper, lead and zinc smelters; a good summary discussion of the toxicology of organic lead can be found in Reference 1.

Perhaps the most disturbing aspect of chronic lead poisoning is the frequency with which disabling diseases occur in spite of normal urinary and blood lead levels. Permanent gout, renal insufficiency, mental deficiency and seizure disorders can all occur with normal urinary and blood lead levels. Although Ca EDTA mobilization tests can confirm the etiology of these diseases, normal screening procedures for lead intoxication would not. Thus the above mentioned effects as well as less specific effects of chronic lead poisoning may occur and go unrecognized to a frightening extent.

Magnesium - Calcium and magnesium are competitive with respect to their absorptive sites, and excess calcium may limit the already poor absorption levels of magnesium salts. Serum levels of magnesium are remarkably constant, however, there is an apparent obligatory urinary loss (the primary route of excretion) of about 12 mg/day. Endocrine activities influence magnesium levels, and there is an inverse relationship between the concentration of calcium and magnesium in bone. If exposure is sufficient, magnesium oxide fume can cause metal fume fever. Conjunctivitis, nasal catarrh, and coughs productive of discolored sputum have also resulted from industrial inhalation exposures. Subcutaneous injection of magnesium into animals produces gas gangrene, while oral poisoning of animals results in hypotension, renal impairment and respiratory parlysis.

Manganese - Although the body burden of manganese has been estimated at 20 mg with the liver, kidney, intestine and pancreas having the highest concentrations, no significant changes in tissue concentration occur with age. Manganese has a rapid turnover in the body, and the lungs do not accumulate manganese with age despite significant concentrations in urban air. Manganese is excreted mainly through the gastrointestinal tract via the bile. Orally or dermally administrated manganese is of low systemic toxicity, but industrial toxicity from inhalation exposures may be severe. Acute exposure to manganese dioxide causes pneumonitis, and men working in plants with high concentrations of manganese dust show an incidence of respiratory disease 30 times greater than normal. Chronic inhalation exposure to manganese dioxide,

generally over a period of more than two years, involves the central nervous system. Symptoms of chronic manganese poisoning (manganism) include a masklike face, retropulsion or propulsion and a Parkinson-like syndrome. The pathophysiology of manganism is also remarkable similar to that of Parkinson's disease. Degenerative lesions of the basal ganglia and markedly decreased levels of dopamime and serotonin in the caudate nucleus occur in both disease states. As a final note, L-Dopa, a drug pharmacologically designed to treat Parkinson's disease, has been effective in the treatment of manganism

Mercury - Mercury is a somewhat unique environmental toxicant for two reasons, volatility and biotransformation. In areas containing soil with high mercury levels (10 ppm), atmospheric concentrations of 20 to 200 µg Hg/m^3 result. These levels are sufficient to produce central nervous system effects under conditions of chronic exposure. Biotransformation of mercury into shortchain alkyl mercury compounds by bacteria contributes to the environmental hazard posed by mercury. The high concentration of methyl mercury in fish, the food source containing the most mercury, is probably attributable to this phenomenon.

Absorption of elemental mercury after ingestion is minimal, but elemental mercury undergoes almost complete absorption from inhaled air. Mercury absorbed in pulmonary tissue may be oxidized in situ to inorganic mercury, and in rats, mercury has a half-life in the lungs of about five hours. Elemental mercury may dissolve in blood, and in this form, it may be transported acorss the blood-brain barrier to be oxidized in brain tissue. Brain levels of mercury are higher after exposure to mercury vapor than after ingestion of a similar dose of mercuric salts. Mercury tends to accumulate in the central nervous system and in the kidneys. Elimination of mercury from the dentate nucleus of the cerebellum is particularly slow. In rats, excretion of mercury after acute exposure is characterized by a rapid phase mediated by fecal excretion, and a slow phase where urinary and fecal excretion play equal roles. In the central nervous system, mercury is eliminated more rapidly from the cortex than from the medulla.

Alkyl mercury compounds differ drastically from the other organic mercurials, and methyl mercury which can be environmentally transformed from other forms of mercury is the most serious hazard in this group. Like elemental mercury vapor, methyl mercury is well absorbed through the lungs and skin, but in diametric opposition to elemental mercury, methyl mercury is nearly completely absorbed from the gastrointestinal tract after ingestion. Alkyl mercurials are preferentially accumulated in erythrocytes, and the change to inorganic mercury characteristic of the other organic mercurials is slowest after doses of methyl mercury. In rats, excretion of methyl mercury primarily occurs through the feces

via the bile, and due to rapid reabsorption, enterohepatic recirculation maintains a large pool of methyl mercury within the bile. Perhaps the most insidious aspect of alkyl mercurials is their ability to cross the placental barrier. Levels of methyl mercury can be 20 percent higher in fetal blood than in maternal blood, and much higher levels are found in fetal as compared to maternal brain tissue.

Symptoms of prenatal mercury toxicity due to maternal ingestion of mercury-containing fish as in Minamata include mental disturbance, ataxia, impairment of gait, speech and mastication. Postmorten examination of these cases revealed hypoplasia and atrophy of the brain tissue. Symptoms of postnatal intoxication from short-chain alkyl mercurials usually have a latent period of weeks to months and include gustatory disturbances, sensory deficits of the lip and extremities, ataxia, constriction of the visual field, impairment of hearing, extrapyramidal disturbances, and mental impairment. These types of cases have been reported as a result of excessive intake from fish (Minamata), occupational exposure, and ingestion of seeds treated with organic mercury compounds. The metabolic interrelationship of metals is again illustrated by experimental work which indicates selenium can decrease the toxicity of methyl mercury in birds and rats.

Ingestion of mercuric chloride leads to an ashen-gray appearance of the mouth, vomiting, a metallic taste in the saliva, excessive salivation, gingivitis, and possibly death as a result of damage to the renal tubule epithelium. Acrodynia (pink disease) has occurred in children exposed to inorganic mercury salts and is believed to be a hypersensitivity reaction. Following a latent period of one to two weeks, cardiovascular manifestations, pink color of the extremities, edema, blistering and desquamation of cells, photophobia, apathy, and hypotonia result. Organic or inorganic mercucy produces similar renal toxicity, and in experimental situations, point mutations. Although no good criterion for judging the extent of mercury exposure exists, the lowest whole-blood concentration for toxicity related to methyl mercury intake is 0.2 µg/g, and urinary and blood levels above 0.3 mg/l and 5 µg/100 ml, respectively probably indicate excessive exposure to elemental mercury.

Molybdenum - Soluble hexavalent forms of molybdenum are well absorbed from the gastrointestinal tract. Excretion of molybdenum is rapid, and over half of that excreted is found in the urine. Of the approximately 9 mg molybdenum body burden, most is concentrated in the liver, kidney, adrenal and omentum. Although there are no recorded cases of molybdenum toxicity in man due to industrial exposure, there are some interesting diseases and metabolic interrelationships of molybdenum with other metals, found in animals. Cattle and sheep grazing in pastures containing 20 to 100 ppm molybdenum may develop a disease

called "teart" which may be prevented by copper or sulfate fortified diets. Teart is characterized by anemia, poor growth rate and diarrhea; prolonged exposure has also led to deformities of the joints. Experimental studies have shown molybdenum trioxide to be more toxic than the sulfide. Molybdenum trioxide has induced fatty degeneration of the liver and kidney if given in sufficient quantity to animals.

Nickel - Absorption of nickel salts from the gastrointestinal tract is limited by their astringent and irritant properties. Although dietary nickel is excreted largely in the feces, inhalation of nickel carbonyl results in the appearance of significant increases in urinary nickel. Normal urine values of 2.3 μg Ni/100 ml have been reported, and urinary nickel values of 0.5 mg/l are considered serious. Dermatitis (nickel itch) is the most frequent effect of exposure to nickel. The dermatitis is a hypersensitivity reaction, and in some cases, nickel may produce paroxysmal asthmatic attacks and pulmonary eosinophilia. Nickel carbonyl is the most toxic of nickel compounds, and 30 ppm for 30 minutes is lethal for man. Nickel carbonyl intoxication causes headache and vomiting initially, and after 12 to 36 hours, dyspnea, cyanosis, leukocytosis, fever and chemical pneumonitis. If death is to occur, 4 to 11 days following exposure is the most likely time. Chronic exposure to nickel carbonyl has been implicated epidemiologically in human cancer of the nasal passages and lungs; experimental work with inhalation exposures to animals have confirmed these findings. In addition, inhalation exposure to nickel and nickel oxide have produced malignant pulmonary neoplasms in guinea pigs and rats.

Selenium - Elemental selenium is probably not absorbed from the gastrointestinal tract. Of the 15 mg average body burden of selenium, the greatest concentrations are found in the kidney, liver and hair. Selenium is excreted in the urine, which normally contains about twice as much as the feces. Normal urinary selenium values are 0 to 15 μg/100 ml. Industrial exposure to hydrogen selenide produces garlic breath, nausea, dizziness, lassitude, and eye and nasal irritation. Acute selenium poisoning causes central nervous system effects such as drowsiness, nervousness and sometimes convulsions. Pallor, coated tongue, gastrointestinal disorders, nervousness, garlic breath, liver and spleen damage, anemia, mucosal irritation and lumbar pain are all symptoms of chronic inhalation exposure to selenium. Selenium is considered embryo toxic and teratogenic in animals, and some reports claim these effects have also been observed in humans. Selenite is more toxic than selenate in animals and has been shown to increase the number of aortic plaques in rats. Many of the symptoms of selenium toxicity can be prevented by arsenic, by high-protein diets, and by methionine in the presence of Vitamin E.

The complexity of the interrelationships of the metals has been further illustrated in an experimental investigation of selenium metabolism in rats. In this study arsenic, mercury, and thallium inhibited the pulmonary excretion of volatile selenium compounds. Mercury increased the blood, kidney and spleen concentrations. Thallium increased renal and hepatic retention. Arsenic increased biliary excretion, lowered selenium tissue levels, and prevented selenium induced liver damage. Lead was without apparent effect.

Tellurium - Tellurium is stored in greatest quantity in bone, while the kidney has the largest soft tissue concentration. Soluble tetravalent tellurites absorbed into the body after oral administration are reduced to tellurides, partly methylated, and then exhaled as dimethyl telluride (responsible for the garlic odor in persons exposed to tellurium). The urine is the principal route of excretion, while sweat and milk are secondary routes. Tellurates and tellurium are of low toxicity, but tellurites are usually more toxic. Acute inhalation exposure can cause sleeplessness, anorexia, nausea, a metallic taste, and a garlic odor of the breath and perspiration. In rats, chronic exposure to tellurium dioxide has produced decreased growth and necrosis of the liver and kidney. One of the more toxic tellurium compounds, tellurium hexafluoride, in concentrations of 5 ppm is lethal to rats exposed for four hours. Five hundred ppm tellurite diets fed to pregnant rats have also produced hydrocephalus in the offspring.

Thallium - Thallium is not normally present in animal tissues, but it can be easily absorbed through the skin and gastrointestinal tract. After poisoning, the highest concentrations of thallium are found in the kidneys and urine, while the intestines, thyroid, testes, pancreas, skin, bone and spleen have lesser amounts. Acute exposure induces large amounts of thallium to be excreted in the urine during the first 24 hours. Following the initial period after exposure, fecal excretion may play the greatest role in elimination of thallium from the body. Acute thallium poisoning is characterized by gastrointestinal irritation, acute ascending paralysis and pyschic disturbances. The estimated lethal dose of thallium for humans is 8 to 12 mg/kg body weight. In man, fatty infiltration and necrosis of the liver, nephritis, gastroenteritis, pulmonary edema, degeneration of adrenals, peripheral and central nervous systems, alopecia, and in some cases death have occurred from chronic systemic thallium intake.

Tin - At most, only 10 percent of ingested soluble tin salts are absorbed through the gastrointestinal tract, but the majority of inhaled tin or its salts remains in the lungs, most extracellularly. Of the average U.S. daily intake of 17 mg tin, most is excreted by the kidneys, and the normal urinary tin level is about 14 µg/100 ml. Tin which does

accumulate after ingestion is found in the liver and kidneys. Chronic inhalation of tin dusts or fumes usually only cause a benign pneumoconiosis, but tin hydride is more toxic than arsine and produces central nervous system effects. Orally, inorganic tin or its compounds require massive doses to be toxic. On the other hand, organic tin compounds are considerably more toxic. Excessive industrial exposure to triethyltin has caused headaches, visual defects, and EEG changes which were only very slowly reversed. Acute burns or sub-acute dermal irritation can result from tributyltin exposure, and triphenyl tin has been shown to be a portent immunosuppressant.

Vanadium - The average body burden of vanadium is about 30 mg with most being stored in adipose tissue. In most forms, vanadium is moderately absorbed from the gastrointestinal tract, and urine is the principal means of vanadium excretion. Normal serum levels of vanadium are considered to be 35 to 48 µg/100 ml. The toxic action of vanadium is largely directed toward the respiratory system as attested to by the unusually high incidence of bronchitis and bronchopneumonia in workers exposed to vanadium compounds. Symptoms also linked with industrial vanadium exposure include eye and skin irritation, gastrointestinal distress, nausea, vomiting, abdominal pain, cardiac palpitation, tremor, nervous depression and kidney damage. Acute vanadium poisoning also causes hemorrhage, paralysis, convulsions and respiratory depression in animals. Epidemiological analyses suggest that heart disease is related to vanadium air pollution and vanadium may act with cadmium to produce these effects.

Zinc - Large concentrations of zinc are found in muscle, liver, kidney, pancreas, epididymis, prostate, testes and eyes. Excretion of zinc is through the gastrointestinal tract; pancreatic fluid contains significant amounts, while the bile contains somewhat less. The urine contains only 20 percent as much zinc as is found in the feces. Accidental oral poisoning with large doses of zinc has produced fever, vomiting, stomach cramps and diarrhea. The main consequence of industrial exposure to zinc compounds is the metal fume fever produced by inhaling freshly formed fumes of zinc oxide. $ZnCl_2$ fumes may produce cyanosis, dermatitis and ulceration of the nasal passages. As zinc is an essential element, there is a homeostatic mechanism for regulating body zinc levels. Thus, orally administered zinc is not toxic to rats unless more than 0.25 percent of their diet is composed of zinc.

SELECT REVIEW OF PERTINENT PUBLISHED EPIDEMIOLOGICAL STUDIES

Many good epidemiological studies dealing with the health effects of metals emitted from Cu, Pb and Zn smelters have been published. In fact, so many have been published that a detailed synopsis of each would

be both redundant and nearly impossible to read. For this reason, only about a dozen of the most relevant and applicable studies are summarized in this section. It is realized that some important work has been omitted, but the included works cover most of the well documented health effects of metals epidemiologically associated with Cu, Pb and Zn smelting. Many other good epidemiological studies with some relevance to the question at hand are included in the list of general references at the end of this section.

Arsenic

Human Arsenic Exposure in Relation to a Copper Smelter[6] - Children living near a copper smelter in Tacoma, Washington, were shown to have increased levels of arsenic in hair and urine. The urinary arsenic level decreased with distance of residence from the smelter stack. House vacuum-cleaner dust showed a similar distance relationship. Urine arsenic levels in children varied synchronously over a 5-week period, indicating that inhalation was the most likely exposure route. In children urinary arsenic level showed an inverse relationship to age with younger children showing consistently higher urine arsenic levels.

A death-record analysis indicated an increased respiratory cancer incidence in men working at this smelter. Since published urinary arsenic levels for men working at this smelter were similar to those seen in people residing near the smelter, it was felt that the community surrounding the smelter might be exposed to an increased respiratory cancer risk. Accordingly, action was taken to reduce arsenic emissions from the smelter.

Respiratory Cancer and Occupational Exposure to Arsenicals[7] - The proportionate mortality experience of 173 decedents exposed primarily to lead arsenate and calcium arsenate was compared with that of 1809 decedents not exposed to those compounds. A significant increase in respiratory cancer was found among the exposed employees. The relationship between cumulative arsenic exposure and the ratio of observed to expected respiratory malignancy deaths was estimated using a least squares approach. The predicted/expected ratio exceeded six-to-one for individuals exposed to compounds containing an equivalent level of 1 mg/m^3 arsenic for a period of more than eight years. For the more heavily exposed individuals, an excess of respiratory cancer was observed 35+ years after the initial exposure. Observations based on the proportionate study were supported by an analysis of the same target population, employing the prospective study in a retrospect approach.

Prevalence of Skin Cancer in an Endemic Area of Chronic Arsenicism in Taiwan[8] - In a limited area on the southwest coast of Taiwan where artesian well water with a high concentration of arsenic has been used

for more than 45 years, a high prevalence of chronic poisoning has been observed in recent years. The total population of this endemic area, which is defined by the presence of both patients and contaminated wells, is approximately 100,000. A general survey of 40,421 inhabitants (19,269 males and 21,152 females) of the area was made. The overall prevalence rates for skin cancer, hyperpigmentation, and keratosis were 10.6, 183.5, and 71.0/1000, respectively. The male-to-female ratio was 2.9:1 for skin cancer and 1.1:1 for hyperpigmentation and keratosis, respectively. Generally speaking, the prevalence increased steadily with age in all three conditions, though there was a decline in age groups above 69 in females with cancer or hyperpigmentation. The prevalence rate for skin cancer showed an ascending gradient according to the arsenic content of the well water, i.e., the higher the arsenic content, the more patients with skin cancer. The results were the same for hyperpigmentation and keratosis. Blackfoot disease, so-termed locally, a peripheral vascular disorder resulting in gangrene of the extremities, especially of the feet, had an overall prevalence rate of 8.9/1000. A dose-response relationship between this disease and the amount of arsenic in the well water was similar to that observed for skin cancer. The association of Blackfoot disease with hyperpigmentation, keratosis, and skin cancer was significantly higher than expected. The causal relationship between Blackfoot disease and chronic arsenicism is discussed.

Cadmium

Some Facts and Documents Relating to the Health Aspects of Cadmium Pollution in Japan[9] - An investigation near mines and smelters in several prefectures in Japan prompted by discovery of cadmium induced Itai-itai (ouch-ouch) disease in the Jinzu River Valley, and the resultant promulgation of regulations is presented. Investigations showed that ingestion of rice and water, polluted by cadmium through mine drainage and/or smelter particulate fallout, was the main source of intake. Rice tended to concentrate the cadmium which had buil. up in the soil. Cadmium toxicity was greatest in women who had given birth to several children and been a resident longer than 30 years.

Seven separate areas, under the influence of three (3) mines and four (4) smelters were observed. Conclusions indicated that: (1) farmers in the areas should not eat rice containing 1.0 ppm Cd (wet weight); (2) no case of Itai-itai disease or cadmium poisoning were found other than that mentioned in the Jinzu River Valley; and (3) local pollution of ambient air by cadmium and lead was found at Annaka (smelting plant).

Due to the results obtained in these areas, and the publicity that was generated, regulations were promulgated giving acceptable standards and the grounds for these standards. The standards are: (1) 0.01 ppm

cadmium for water; (2) 0.1 ppm Cd in liquid effluent (assuming dilution factor of 10); (3) 1.0 ppm Cd in unpolished rice; 0.9 ppm Cd in polished rice; (4) 1.0 mg/nm^3 Cd atmospheric emission standard; and (5) soil pollution control for agricultural land on which the rice contains levels of cadmium greater than 1.0 ppm. In addition, where the above designated levels are exceeded, the cadmium concentration in urine of not less than 30 adult persons in the area should be examined; urinalysis diagnosis is to include protein, and cadmium tests, if possible. Measurement of blood pressure was also advocated. Guidelines for "areas requiring observation" produced standards of 9 µg/l Cd in the urine as a primary screening test and 30 µg/l Cd, based on the average concentration in food of 0.99 ppm as a secondary screening level. Total diseased patients from 1967-1972 totaled 123, including 40 dead. Control of damage, both physical and agricultural is discussed.

Lead

The Consequences of Chronic Lead Poisoning[10] - Workers in various professions exposed to lead vapors or dust in concentrations exceeding 0.01 mg/cubic meter were examined and working capability was evaluated on the basis of clinical symptoms and general condition. In the earlier stages of poisoning, temporary transfer to working places without exposure to lead is recommended. Where the symptoms do not disappear, then permanent retirement must be recommended. The average exposure time before retirement due to chronic poisoning was 11.6 years for men and 12 years for women. Retirement of women was less frequent than for men, which is attributed to the less hazardous conditions for women.

EPA's Position on the Health Effects of Airborne Lead[11] - Clinical manifestations of mild lead intoxication (loss of appetite, irritability, drowsiness, apathy and abdominal pain) are mentioned, citing specific cases, especially with regard to children. Levels of lead concentration below that considered toxic may contribute to minimal brain damage. The metabolic effects of low levels of lead (below 40 µg/100 g blood) which include enzyme inhibitation may impact the central nervous system or cause genetic damage. Chromosomal abnormalities were found in 13 of 15 randomly selected individuals located near a lead smelter. Lead blood levels of 40 µg/100 g blood for adults are acceptable evidence that excessive exposure to lead has occurred, and the possibility that lower levels in children may cause significant damage is discussed.

Sources of lead exposure among the general population include paint, dirt, and dust. Ninety percent of airborne lead emissions comes from leaded gasoline which in turn contributes to lead levels in dust and dirt. Airborne lead exposures were more relevant in adults than small children, whose primary exposure comes from ingestion of house dirt and dust as well as leaded paint.

The report indicates that: (1) levels of 30 µg/100 g blood are considered excessive for pregnant women; (2) a safe blood level for children should not be above 40 µg/100 g; (3) blood levels of 30 µg/100 g obtained from umbilical cord blood is excessive; (4) airborne lead levels of 2 µg/m have been demonstrated to contribute to high blood lead levels; (5) individuals within groups may be excessively exposed to lead even though the average exposure is well within normal limits; and (6) every effort should be made to reduce all preventable lead exposure.

Occupational and Enyironmental Lead Poisoning in Association with a Secondary Lead Smelter[12] - Employees at a secondary lead smelter, household occupants of neighboring residences, cows, horses, dogs and cats quartered nearby, and plants, soil and water from the adjacent area were surveyed to determine the public health significance of lead pollution originating from this factory. Eighty-six percent of company laborers were found to have blood lead levels at or greater than 80 µg/100 ml, and in two years since the installation of a blast furnace by the company, at least nine employees had been hospitalized for treatment of lead poisoning, primarily but not exclusively lead colic. Illnesses and deaths from lead poisoning have been demonstrated in cows, horses, dogs and cats quartered in the vicinity of the smelter. A case of chronic plumbism in a survivor of bovine lead poisoning has been described. Cows foraging on land within 400 yards downwind of the smelter furnace have been determined to be in danger of dying from lead poisoning. Lead poisoning has also been found to present a significant threat to the health of children residing within 100 yards downwind. Apparent deviations from good practice are a major obstacle to controlling lead pollution by the company and in instituting proper medical supervision of its workers. Blood protoporphyrin has been suggested as a better measure of cumulative lead-induced physiologic damage than blood lead. Normal blood protoporphyrin values for cattle and a correlation between elevated blood lead and elevated blood protoporphrin in these animals have been established for the first time. Normal values for hair lead of cattle, horses and dogs have also been determined.

Neuropsychologic Dysfunction in Children with Chronic Low-Level Lead Absorption[13] - The relationship between low-level lead absorption and neuropsychologic function is not clearly defined. To investigate it further, neurologic and psychologic evaluations were undertaken of 50 asymptomatic children, ages 3 to 15 years, with blood lead concentrations of 40 to 60 µg/100 ml (mean, 49 µg/100 ml) and of 81 controls with levels less than 40 µg/100 ml (mean, 27 µg/100 ml). Groups were matched by age, sex, ethnic background, and socioeconomic status. All children lived within 4 miles of a large smelter, and smelter emissions were a major source of absorbed lead. Lead exposure was chronic (mean, 6.6 years) and, in many instances, lifelong.

Performance I.Q. was found to be significantly lower in the children with lead levels greater than or equal to 40 µg/100 ml (mean scores 94 versus 102, $p < 0.01$). Children in this group also showed subclinical weakness of wrist muscles as compared to controls. Verbal I.Q., activity and behavior ratings, and other neurologic functions did not differ significantly. These findings suggest that low-level lead absorption, at least when it is of sufficiently early onset and long duration, may produce definite neuropsychologic impairment. Whether the impairment noted in this study will be reversible remains to be determined.

Non-Professional Saturnism: A Pollution Epidemic from Industrial Emission[14] - An epidemic of lead poisoning occurred among residents of a small Italian community, Stanghella, in the proximity of a plant for the fusion of lead from used automobile batteries. Of the 179 persons examined, 30.7 percent had urinary lead levels between 30 to 60 micrograms/l and 37.2 percent had significantly high levels. Such levels are not generally encountered in individuals without occupational exposure to lead. High levels of erythrocytic protoporphyrins proved the existence of very high lead concentrations in the tissues of about two-thirds of the population. Only one-third of the cases showed a definite increase in urinary coproporphyrins; however, the majority of the subjects were examined 3 months after the plant was closed and urinary coproporphyrins are known to decrease rapidly following cessation of lead exposure. Pronounced iron anemia was registered in 12 cases. Most subjects lived 300 to 700 feet downwind from the plant, suggesting that the lead poisoning was brought about by direct contamination of their houses and front yard vegetable gardens.

Chromosome Aberrations Observed in Male Workers Occupationally Exposed to Lead[15] - Chromosome observations were performed on peripheral blood lymphocytes of 14 workers from a zinc industry who have presented signs of lead poisoning of different degrees. According to type and duration of exposure, workers examined were divided into three groups: those exposed to a high level of zinc and low levels of lead and cadmium; those exposed to high levels of the three minerals; and those exposed to high levels of lead and cadmium in the absence of zinc. The aberrations were dicentrics, rings, chromatid exchanges, as well as gaps and fragments. Exposure to zinc and cadmium does not seem to increase the number of cells with severe chromosome anomalies, and lead intoxication can be considered to be responsible for the chromosome aberrations.

Manganese

Industrial Hygenic Studies on Manganese[16] - Forty-one workers in the manganese railing manufacturing process were examined to see the effects of traceable manganese dusts on their health. Their medical

histories, conscious symptoms, chest x-rays, manganese excretion in urine, and other nervous and physiological symptoms were examined. Air samplings were taken by a hi-volume sampler, particle size was measured by the Andersen air sampler, and the Mn content in dusts and urine was measured by atomic absorption spectrophotometry. The total amount of dusts in three areas were 9550, 4058, and 2834 mg/cu m and the dust particle sizes were generally large (only 13.3% were less than 2 micron). The Mn content in dust was 0.963 percent: by individual samplers, Mn in dust was 1.467 mg/cu m. It was far below the maximum allowable concentration in the working environment which is 5 mg/cu m. The workers average age was 43.2 years, the average duration of work in the same environment was 15.6 years, 65.9 percent were older than 40 years, and 75.6 percent had worked more than 10 years. The chest x-ray revealed 12 workers (29.3%) with pneumoconiosis. They were all older than 40 years old and had worked more than 15 years in the same environment. Significant differences from the control group were noted in the complaints of hip pains (39.0%) and the average on the total number of complaints (12.6%). Other symptoms with high rates of complaints were increased sputum (41.5%), wheezing (7.3%), chest pain (9.8%), fatigue (29.3%), and pain in the joints (34.2%).

Nickel

Some Statistical Materials on Carcinogenic Hazards in the Production of Nickel[17] - The increased mortality rates due to carcinoma and sarcoma among workers of a nickel combine were statistically investigated and compared with similar rates for the city population and environs. Lung cancer accounted for the highest mortality rate at a factor of 2.8 greater than that expected among males 40 years old and older working at the roasting-reduction and cobalt shops and exposed to dusts containing nickel sulfides, nickel oxides, cobalt and arsenic compounds. The average service period for workers employed in nickel shops who died of pulmonary carcinoma was 13 years. Femoral and pulmonary sarcomas were most often encountered among the men at the combine.

Copper, Lead, or Zinc Smelters

Occupational Lung Cancer Among Copper Smelters[18] - Because a high lung cancer mortality rate was noted among males in a town, a case control study was carried out on the basis of mortality cards. The case group consisted of 19 males who died from lung cancer and a control group of 19 males who died of other causes. The only significant difference found is that 11 of the 19 subjects with lung cancer had been employed as smelters in a local copper refinery, and exposed to arsenic and other compounds. In all 11 cases, the disease became manifest after the men had stopped working in the refinery.

An Environmental Impact Study of a Zinc Smelter[19] - It is the conclusion of this report that the Asarco zinc smelter in Amarillo, Texas is emitting particulate and heavy metal contaminants to the atmosphere. This report finds that:

a. Zinc, cadmium, lead and mercury are being emitted by the plant.

b. There is sufficient information to suggest a direct connection between the plant emissions and blood cadmium levels in children of the area.

c. Concentrations of zinc, lead and cadmium are building up in the soils adjacent to the plant.

d. Zinc emissions from the plant contribute about 20 percent of the atmospheric lead burden in the area.

e. Lead emissions from the plant contribute about 20 percent of the atmospheric lead burden in the area.

f. Stack emissions appear to be in compliance with Air Control Board regulations.

g. It is undisputed by the staff and Asarco that fugitive emissions are in violation of present Air Control Board regulations and will also be in violation of new regulations to be enforced after December 31, 1973.

The following conclusions are drawn by this report:

a. No significant toxic effects should result from zinc oxide emissions by the plant.

b. Lead levels in the Amarillo area are not above normal urban levels.

c. If retort emissions contain 50 percent by weight zinc, it is technologically and economically feasible for the plant to install abatement equipment.

Specific References for Section 1.0

1. Casarett, L.J. and J. Doull. Toxicology, The Basic Science of Poisons. Macmillan Publishing Co., Inc. New York, 1975.

2. Hardy, H.L. and Alice Hamilton. Industrial Toxicology. Publishing Sciences Group, Inc. Acton, Mass., 1974.

3. Lee, Douglas H.K. Metallic Contaminants and Human Health. Sponsored by NIH. Academic Press. New York, 1972.

4. Documentation of the Threshold Limit Values for Substances in Workroon Air. American Conference of Governmental Industrial Hygienists. Cincinnati, Ohio, 1971.

5. Ferm, V.H. and S.J. Carpenter. The Relationship of Cadmium and Zinc in Experimental Mannaliam Teratogenesis. Laboratory Investigation. 18 (4):429-432, 1968.

6. Milham, Samuel Jr. and Terrence Strong. Human Arsenic Exposure in Relation to a Copper Smelter. Reprint: Environmental Research. 7:176-182, 1974.

7. Ott, M.G., et al. Respiratory Cancer and Occupational Exposure to Arsenicals. The Dow Chemical Co. Midland, Mich., 1974.

8. Tseng, W.P., et al. Prevalence of Skin Cancer in an Endemic Area of Chronic Arsenicism in Taiwan. Journal National Cancer Institute. 40:453-463, 1968.

9. Hasegawa, Dr. G. Some Facts and Documents Relating to the Health Aspects of Cadmium Pollution in Japan. Env. 45(73):69-78, 1973.

10. Grigor'ev, Z.E. The Consequences of Chronic Lead Poisoning. (O Posledestviyakh Kdhronicheskio svintsovoi intoksikatsii.) Text in Russian. Vrachebnoe Delo. 11:117-118, November 1967.

11. EPA's Position on the Health Effects of Airborne Lead. Health Effects Branch, Environmental Protection Agency. November 29, 1972.

12. Levine, R.J. and R.M. Moore, Jr. Occupational and Environmental Lead Poisoning in Association with the Sanders Lead Company, Inc., A Secondary Lead Smelter in Troy, Alabama. HEW-CDC. Atlanta, 1974.

13. Landrigan, P.J., M.D., et al. Neuropsychologic Dysfunction in Children with Chronic Low-Level Lead Absorption. HEW. Atlanta, 1974.

14. De Rosa, E. and F. Gobbato. Non-Professional Saturnism: A Pollution Epidemic from Industrial Emission. (Epidemia di saturnismo non professionale per inquinamento da effluenti industriali.) Translated from Italian. Igiene Mod. (Parma), 63(1/10):472-484, 1970.

15. Deknudt, Gh., A. Leonard, and B. Ivanov. Chromosome Aberrations Observed in Male Workers Occupationally Exposed to Lead. Environ. Physiol. Biochem. 3(3):132-138, 1973.

16. Kobayashi, Y., Y. Sugiyama, S. Ushisome, Y. Niitani, S. Sarai, and A. Ochial. The Effects of Exposure to Traceable Manganese on the Human Body - Industrial Hygienic Studies on Manganese Poisoning (2). (Biryo mangan bakuro ni yoru jintai eidyo ni tsuite - Mangan chudoku ni kansuru rodo eiseigakutekikkenkyu.) Text in Japanese. (Presented at the Japan Society of Industrial Hygiene, Annual Meeting. April 1973).

17. Saknyn, A.V. and N.K. Shabynina. Some Statistical Materials on Carcinogenic Hazards in the Production of Nickel on an Ore Oxide Base. (Nekotoryye statisticheskiye materialy o kantserogennoi opasnosti v proizvodstve nikelya na baze okislennykh rud.) Gigiena Truda Prof. Zabolevaniya. 14 10-13, Nov. 1970. Translated from Russian. Leo Kanner Assoc. Redwood City, Calif., 7 p., Dec. 1971.

18. Kuratsune, N., S. Tokudome, T. Shirakusa, M. Yoshida, Y. Tokumitsu, T. Hayano, and M. Seita. Occupational Lung Cancer Among Copper Smelters. International Journal of Cancer. 13(4):552-558, 1974.

19. An Environmental Impact Study of The American Smelting and Refining Company's Zinc Smelter. Air Pollution Control Services, Texas State Department of Health. Amarillo, Texas. 1974.

General References for Section 1.0

1. Anderson, D. Emission Factors for Trace Substances. Office of Air Quality Planning and Standards, Research Triangle Park. December 1973.

2. A Report of Typical Element Emissions from Texas Smelters. Texas Air Control Board. Austin, Texas. April 1974.

3. Athanassiadis, Y. C. Preliminary Air Pollution Survey of Cadmium and Its Compounds, A Literature Review. U.S. Department of Health, Education and Welfare, Public Service. Raleigh, North Carolina. October 1969.

4. Background Information for New Source Performance Standards: Primary Copper, Zinc and Lead Smelters, Volume I: Proposed Standards. EPA-450/2-74-002a. Environmental Protection Agency, Research Triangle Park, North Carolina. October 1974.

5. Barltrop, D., et al. The Significance of High Soil Lead Concentrations for Childhood Lead Burdens. (Presented at EPA-NIEHS Conference on Low Level Toxicity. Raleigh, North Carolina. October 1973.)

6. Baum, E. J. and R. L. Pitter. Airborne Lead from Reentrained Street Dusts. PNWIS-APCA paper 74-AP-30. (Presented in Boise. November 1974.)

7. Beson, N. R. et al. Reestablishing Apple Orchards in the Chelan Mason Area. U.S. Bureau of Reclamation. October 1968.

8. Birmingham, Donald J., et al. An Outbreak of Arsenical Dermatoses in a Mining Community. Arch. Derm. Volume 91, May 1965.

9. Blejer, H. P., P. E. Caplan, and A. E. Alcocer. Acute Cadmium Fume Poisoning in Welders - A Fatal and a Nonfatal Case in California. Calif. Med. 105(4):290-296, October 1976.

10. Bohn, H. L. Soil Absorption of Air Pollutants. J. Environ. Quality. 1(4), 1972.

11. Bonnel, J. A. Lead Smelting at Avonmouth. British Journal of Industrial Medicine. 30, 100, 1973.

12. Bothner, M. H. Personal Correspondence to Mr. John Roberts, PSAPCA. University of Washington, October 25, 1975.

13. Bourasset, A. and G. Galland. Cancer of the Respiratory Tract and Exposure to Nickel Salts. (Cancer Des Voies Respiratoires Et Exposition Aux Sels De Nickel). Translated from the French. Arch. Maladies Profess. Med. Trav. Securite Sociale (Paris). 27:227-229. Jan.-Feb. 1966.

14. Buechley, R. W. Epidemiological Consequences of an Arsenic-Lung Cancer Theory. American Journal of Public Health. 53(8):1229-1232.

15. Butlin, H. T. Tar and Paraffin Cancer. British Medical Journal. p. 66-71.

16. Campbell, A. M. G., et al. Lead in Relation to Disseminated Sclerosis. Brain. 73:52-71, 1950.

17. Carpenter, R. Geochemistries of Mercury, Arsenic and Other Metals in Puget Sound: Report of Progress, 1 May 1972 - 31 December 1973. ASF-RANN Giant GI 33325X. Seattle. January 4, 1974.

18. Cooper, Hal B. H., et al. Measurement of Mercury Vapor Concentrations in Urban Atmospheres. (Presented at Annual Meeting of Instrument Society of America. Houston, Texas. October 15, 1973).

19. Cooper, W. C. and W. R. Gaffey. Mortality in Lead Workers. Journal of Occupational Medicine. 17(2): 100-107.

20. Copenhaver, E. D., et al. Cadmium in the Environment: Annotated Bibliography. Oak Ridge National Laboratory. Oak Ridge, Tennessee. April 1973.

21. Cralley, L. V. and P. R. Atkins. Industrial and Environmental Health, the Worker and the Community. Academic Press. New York. 1975.

22. Crecelius, E. A., et al. Contamination of Soils Near a Copper Smelter by Arsenic, Antimony and Lead. Water, Air, and Soil Pollution. Dordrecht-Holland. 3:337-342, 1974.

23. Crecelius, E. A. The Geochemistry of Arsenic and Antimony in Puget Sound and Lake Washington. (Ph.D dissertation, University of Washington, Seattle). 1974.

24. Crecelius, E. A. and R. Carpenter. Arsenic Distribution in Waters and Sediments of the Puget Sound Region. (Presented at the First Annual NSF Trace Contaminants Conference. Oak Ridge National Laboratory. August 1973.)

25. Dencker, L. Possible Mechnisms of Cadmium Fetotoxicity in Golden Hamsters and Mice: Uptake by the Embryo, Placenta and Ovary. University of Uppsala, Uppsala, Sweden. p. 461-471, 1975.

26. Djuric, Dusan, L. Graovac-Leposavic, S. Milic, and L. Senicar. Lead Contamination of Meza Valley, Lead Mobilization with Ca-Na_2-EDTA from the Body of Inhabitants. Pracovni Lekar. Prague. 24(2-3):49-50, 1972.

27. Djuric D., Z. Kerin, L. Graovac-Leposavic, L. Novak, and M. Kop. Environmental Contamination by Lead From a Mine and Smelter: A Preliminary Report. Archives of Environmental Health. 23:275-279.

28. Eatough, D. J. Determination and Possible Public Health Impact of Transition Metal Sulfite Aerosol Species. Brigham Young University. Provo, Utah. December 1975.

29. Agricultural Land Soil Pollution Prevention and Other Laws. Environmental Agency. Japan. 1971.

30. Main Policies for Agricultural Land Soil Pollution. Environmental Agency. Japan. August 1974.

31. Quality of the Environment in Japan. Environmental Agency. Japan. 1973.

32. Air Pollution Aspects of Emission Sources; Primary Lead Production, A Bibliography with Abstracts. Environmental Protection Agency. June 1973.

33. Mercury in the Environment. Environmental Protection Agency. December 1973.

34. National Emission Standards for Hazardous Air Pollutants, Asbestos, Beryllium and Mercury. Environmental Protection Agency. Federal Register. 38(66), Part II. April 6, 1973.

35. Ferens, M. Catherine. A Review of the Physiological Impact of Mercurials. University of Georgia. EPA-660/3-73-002. February 1974.

36. Fishburn, C. W. and Carl Zenz. Metal Fume Fever, A Report of a Case. J. Occup. Med. 11:142-144, March 1969.

37. Franklin, K. S. More About Cadmium Poisoning. Refrigeration Service Contracting. 34:28-30, October 1966.

38. Fulkerson, W. and H. E. Goeller, Editor. Cadmium - The Dissipated Element. Oak Ridge National Laboratory, Oak Ridge, Tennessee. January 1973.

39. Ganje, T. J. and A. L. Page. Lead Contaminations of Plants, Soil, and Air Near Highways. Calif. Agr. 26:7-9, April 1972.

40. Geological Survey. Mercury in the Environment. Professional Paper 713, U.S. Government Printing Office. 1970.

41. Ghelberg, N. W., Ingrid Ruckert, and H. Straus. The Lead Content of Milk in an Area with Nonferrous Metallurgical Industry. (Continutul de Plumb in Lapte, Intro zona cu Industrie Metalurgica Neferoasa.) Text in Rumanian. Igiena. Bucharest. 21:17-22, January 1972.

42. Greve, L. H., P. E. Joosting, and B. F. Visser. Influence of Dust Exposure Upon the Distribution of Alveolar Ventilation. (Nederlandse Organisatie Voor Toegepast Natuurwetenschappelijk Onderzoek.) Forschungsinstitut fuer Gesundheitstechnik. Delft. 244:601-603, February 1968.

43. Guja, Antoni, Jr. Gastric Disorders and the Uropepsin Level in Persons Exposed to the Action of Zinc Oxides. (Dolegliwosci gastryczne a Poziom uropepsyny u osob narazonych na dzialanie tlenkow cynku.) Translated from Polish. Wiadomosci Lekar. Warsaw. 26(2):141-143, 1973.

44. Hammer, D. I., et al. Cadmium and Lead in Autopsy Tissues. (Presented at the AMA Annual Convention. June 1973.)

45. Hammer, D. I., et al. Hair Trace Metal Levels and Environmental Exposure. American Journal Epidemiology. 93(2):84, 1971.

46. Hammer, D. I., et al. Trace Metals in Human Hair as a Simple Epidemiologic Monitor of Environmental Exposure. Trace Substance in Environment Health-V. University of Missouri, Columbia. 1972.

47. Hanslian, L., J. Navratil, J. Jurak, and M. Kotrie. Upper Respiratory Tract Lesions from Chromic Acid Aerosol. (Poskozeni hornlich cest dychacich aerosolem kyseliny chromove.) Text in Czech. Pracovni Lekar. 19(7):294-298, September 1967.

48. Heilman, P. Arsenic Content of Soils in the Vicinity of the Ruston Smelter. Western Washington Research and Extension Center, Washington State University. June 1973.

49. Hematological Changes in Workers in the Production of Hard Metals. (Hematologicke zmeny u pracujicich Pri vyrobe tvrdych kovu.) Text in Czech. Pracovni Lekar. 19:11-15, January 1968.

50. Hemphill, D. D., et al. Toxic Heavy Metals in Vegetables and Forage Grasses in Missouri's Lead Belt. (Presented at the Association of Official Analytical Chemists Annual Meeting. Washington, D.C. October 9-12, 1972.) J. Assoc. Offic. Anal. Chemists. 56(5):994-998, 1973.

51. Holmberg, R. E. and V. H. Ferm. Interrelationships of Selenium, Cadmium, and Arsenic in Mammalian Teratogenesis. Archives of Envir. Health. 18:872-877, June 1969.

52. Huntamer, D. D. and W. R. Schell. Environmental Sampling and Analysis for Trace Metals in Puget Sound. (Presented at the 24th Regional Meeting of the American Chemical Society. Cheney, Washington. June 13-14, 1974.)

53. Ilic, V. and K. Popovic. Metal Fume Fever. (Cinkova groznica.) Translated from Serbo-Croatian. Arhiv Hig. Rada. Toksikol. (Yugoslavia.) 22:359-364, 1971.

54. Ito, Yoko, M. Nagata, and T. Tsuji. Diagnosis of Respiratory Sumptoms of Inhabitants Around a Certain Refinery by Oral Communication. (Bo seirensho shuhen jumin no kokyuki shojo monshin seiseki.) Text in Japanese. Nippon Koshu Eisei Zasshi. (Japan) J. Public Health. 18(10):426, 1971.

55. Hokawa, Dr. Y., T. Abe, S. Tanaka, M.D. Bone Changes in Experimental Chronic Cadmium Poisoning, Radiological and Biological Approaches. Kyoto Japan University. Arch. Environ. Health. 26:241-244, May 1973.

56. Hokawa, Dr. Y., T. Abe, S. Tanaka, M.D. Renal and Skeletal Lesions in Experimental Cadmium Poisoning Histological and Biochemical Approach. 28:149-154, March 1974.

57. Johnson, C. J. Environmental Contamination with Lead and Arsenic From a Copper Smelter. PNWIS-APCA Paper #73-AP-37. Seattle. November 1973.

58. Kangas, J., et al. Smelter Gases Yield Mercury. Chemical Engr. pp 55-57, September 6, 1971.

59. King, P. G. Maximum Daily Intake of Lead Without Excessive Body Lead-Burden in Children. American Journal Dis. Children J.V. 122:337, October 1971.

60. Kitamura, S., K. Hayakawa, and T. Shibata. The Absorption, Excretion, and Toxicity of Antimony. (Anchimon Sb, no kyushu, haisetsu narabini dokusei ni tsuite.) Text in Japanese. (Presented at the 46th of the Annual Meeting of the Japan Society of Industrial Hygiene. April 1973.) p. 180-181, 1973.

61. Klauder, D. S., L. Murthy, and H. G. Petering. Effect of Dietary Intake of Lead Acetate on Copper Metabolism in Male Rats. University of Cincinnati, Ohio. Trace Substances in Environmental Health-VI. p. 131-136, 1973.

62. Klein, D. H. Mercury and Other Metals in Urban Soils. Dept. of Chemistry, Hope College, Holland, MI. Environmental Science and Technology, 1972.

63. Klein, D. H. and P. Russell. Heavy Metals: Fallout Around a Power Plant. Environmental Science and Technology. Vol. 7, April 1973.

64. Kobayashi, J., et al. Effect of Air and Water Pollution on Agricultural Products by Cd, Pb, Zn Attributed to Mine Refinery in Annada City, Gunma Prefecture. Jap. J. Hyg. 24(4):364-375, October 1970.

65. Kreuger, H. W. Lead Content of Dirt from Urban Households. Kreuger Enterprises, Inc. Cambridge, Mass. March 1972.

66. Landrigan, P. J., M.D., et al. Epidemic Lead Absorption Near an Ore Smelter. HEW-CDC. Atlanta. 1974.

67. Larsson, Sven-Erik and M. Piscatèer. Effect of Cadmium on Skeletal Tissue in Normal and Calcium Deficient Rats. Israel Journal of Med. Sciences. 7:495-497, March 1971.

68. Lillis, E. J. Lead and Cadmium in House Dust, Street Dust and Dustfall in Areas Close to Major Roadways. Masters Thesis, University of Washington, Seattle. 1974.

69. Lob, M., M. Guillemin, and M. N. Valloton. Use of Modern Parameters to Evaluate the Biological Effect of Lead on Subjects with Occupational and Non-Occupational Exposure. (Interet des parametres modernes pour l evaluation de l action biologigue du plomb chez les sujets exposes et non exposes professionnellement.) Text in French. Arch. Maladies Profess. Med. Trav. Securite Sociale. (Paris) 33:621-626, December 1972.

70. Lutz, G., A. A. Levin, S. G. Bloom, K. L. Nielson, J. L. Cross, and D. L. Morrison. Technical Intelligence, and Project Information System for the Environmental Health Service (Volume III): Lead Model Case Study. Battelle Memorial Inst., Columbus Ohio. EHS Contract CPS-69-005, Rept. CPS-69-005-3. 140 p. June 29, 1970.

71. McCallum, R. I., M. J. Day, J. Underhill, and E. G. A. Aird. Measurement of Antimony Oxide Dust in Human Lungs in Vivo by X-Ray Spectrophotometry. British Occupational Hygiene Society. (London) 2:611-619, 1971.

72. McCallum, R. I. Respiratory Disease in Foundrymen. British Journal of Industrial Medicine. 29:341, 1972.

73. McCaull, J. Building a Shorter Life. Environment. 13(7):2-15, 38-41, September 1971.

74. McClannan, H. A Study of Metallic Air Pollution in the Vicinity of Copper Smelter. Masters Thesis, University of Washington, Seattle. December 1974.

75. Manton, W. I. Significance of Lead Isotope Composition in Blood. Nature. (London) 244(5412):165-167, July 20, 1973.

76. Mason, T. J., et al. Atlas of Cancer Mortality for U.S. Counties: 1950-1969. DHEW Publication #(NIH)75-780. U.S. HEW.

77. Mason, T. J., et al. U.S. Cancer Mortality by County: 1950-1969. DHEW Publication #(NIH)74-615.

78. Mastromatteo, Ernest. Nickel: A Review of Its Occupational Health Aspects. J. Occupational Med. 9:128-136, March 1968.

79. Mehani, S. Lead Retention by the Lungs of Lead-Exposed Workers. Annual Occupational Hyg. (London) 9:165-171, July 1966.

80. Metals Focus Shifts to Cadmium. Environ. Sci. Tech. 5:754-755, September 1971.

81. Mikahaylov, V. A., N. V. Tyushnyakova, A. V. Saknyn, L. N. Yeinichnykh, and A. S. Vorontsova. Generally Toxic Action of Nickel-Containing Dusts. (K voprosu ob obshchetoksi-cheskom deystvil nikel soderzhashchikh pyley.) Translated from Russian. Vop. Gig. Prof. Parol. Tsvet. Dhern. Met. 1971:200-206, 1971.

82. Ministry of Foreign Affairs, Japan. Development of Environmental Protection in Japan. Japan. 1973.

83. Ministry of Health and Welfare (Japan). Environmental Pollution Control Dept. Materials for Countermeasures Concerning Environmental Pollution by Cadmium. (Kado myumu kankyo osen taisaku shiryo.) Translated from Japanese. Countermeasures Against Environmental Pollution by Cadmium. p. 385-393, March 1972.

84. Ministry of Health and Welfare (Japan). Environmental Pollution Control Dept. Opinion of the Welfare Ministry with Regard to Ouch-Ouch Disease and Its Causes (Annexed Materials). (Itai-itaibyi to sono gen in ni kansuru koseisho no kenkai (fuzoku shiryo).) Translated from Japanese. Countermeasures Against Environmental Pollution by Cadmium. p. 125-148, March 1972.

85. Ministry of Health and Welfare (Japan). Public Nuisance Section. Opinion of the Ministry of Health and Welfare and the Countermeasure to the Environmental Hazard By Cadmium at the District Around Mikkaichi Refinery in Kurobe City, Toyama Prefecture. (Toyama-ken Kurobe-shi Mikkaichi Seirenjo shuhen ni okeru kadomiumu kankyo osen ni kansuru koseisho no kenkai to kongo no taisake.) 7 p. May 1970.

86. Mirando, E. H. and M. Gomez. Lead Poisoning in Childhood in Ceylon. Arch. Disease Childhood. 42(226): 579-582, December 1967.

87. Misiaszek, M. S. and D. Van Wyck. The Effect of Air Pollution and Weather on Lung Function in Exercising Children and Adolescents. American Review of Respiratory Disease. 109(2):262-273.

88. Missouri University. Study of Lead, Copper, Zinc and Cadmium Contamination of Food Chains of Man. National Technical Information Service, U.S. Dept. of Commerce, Publisher. December 1972.

89. Mottet, N. K. and R. L. Body. The Mercury Burden of Human Autopsy Organs and Tissues. Archives of Environmental Health. 1973.

90. Munshower, F. F. Cadmium Compartmentation and Cycling in a Grassland Ecosystem in the Deer Lodge Valley, Montana. Ph.D. Thesis, Botany Dept., Univ. of Montana, Missoula, Montana. 106 p., 1972.

91. National Institute for Occupational Safety and Health. Criteria for a Recommended Standard - Occupational Exposure to Inorganic Arsenic. HEW Public Service, 1973.

92. Nelson, K. W. Statement of K. W. Nelson, Director, Department of Environmental Sciences, American Smelting and Refining Company. Puget Sound Air Pollution Control Agency Hearing. February 14, 1973.

93. Nodiya, P. I. Study of the Body Cobalt and Nickel Balance in Students of Technical Trade Schools. (izucheniye balansa kobal ta i nikelya v organizme uchashcikhysa PTU.) Translated from Russian. Gigiena i Sanit. 37(5):108-109, 1972.

94. Moniyama, Kazunari. Kidney Damage in Cadmium Poisoning Japan Public Health Assoc., Proc. Sci. Res. Meet. Itai-itai Disease Cadmium Poisoning. Japan. August 28-29, 1971.

95. Noweir, M. H. and E. A. Pfitzer. Evaluation of Coproporphyrin in Urine from Workers Exposed to Lead. (Presented at the American Industrial Hygiene Association Conference. Denver, Colo. May 11-16, 1969.) Am. Ind. Hyg. Assoc. J. 31(4):492-500, July-Aug., 1970.

96. Occupational Health and Safety Administration. OSHA Proposes .004 Milligram Standard for Arsenic Exposure. Occupational Health and Safety Letter, January 22, 1975.

97. Okutani, H. and A. Harada. The Effects of Lead Pollution on Residents Around a Steel Factory. (Bo seiko kojo shuhen chiiki jumin no en osen.) Text in Japanese. Nippon Eisei Zasshi (Japan J. Hyg.). 28(1):91, April 1973.

98. Oregon State University, Experimental Station. Selenium: Maddening Mineral. Oregon's Agricultural Progress. Oregon State University, Corvallis. 21(1):21-24, Summer 1974.

99. Pinkerton, C., et al. Human Milk as a Dietary Source of Cadmium and Lead. Reprinted from Trace Substances in Environmental Health - VL 1973. A symposium, D. D. Hemphill, Ed., University of Missouri, Columbia.

100. Poetzl, K. and R. Reiter. Study of Retention of Chemical Components of Industrial Aerosols. (Zur Retinierbarkeit der chemischen Komponenten von Industrieaerosolen.) Text in German. Zentr. Arbeitsmed. Arbeitsschutz. 19(9):1-8, 1969.

101. Posner, E. Diagnosis and Management of Industrial Chest Disease (Respiratory Tract Disease). British Med. J. 1(5486):525-529, February 26, 1966.

102. Puget Sound Air Pollution Control Agency. The Concentration of Lead, Arsenic, Mercury, Cadmium, SO_2 and Suspended Sulfates Downwind from the Tacoma Smelter, the Impact and Control Status, and Benefits from Reduction. (Presented to the ASARCO Impact Task Force. January 1, 1975.)

103. Rajner, V. Effect of Vanadium on the Respiratory Tract. (Vliv Vanadu na Dychaci Cesty.) Translated from Czech. (Presented at the National ORL (Otolaryngolocical) Conference. Karlovy Vary, Czechoslovakia. October 8, 1959.) Cesk. Otolaryngol. 9:202-204, August 1960.

104. Reiter, R., K. Poetzl, and W. Carnuth. An Automatic Retention Simulator. (Ein automatischer Retentionssimulator.) Text in German. Staub, Reinhaltung Luft. 32(10):392-399, October 1972.

105. Roschin, A. V. Industrial Aerosols of Rare Metals and Their Hygienic Significance. British Occupational Hygiene Society. (London) 2:811-817, 1971.

106. Roschin, I. V. Hygienic Aspects of Industrial Vanadium Aerosol. Translated from Russian. Literature on Air Pollution and Related Occupational Disease. 4:109-114, August 1960.

107. Rossano, A. T. and H. McClannan. Concentrations of Arsenic and Lead in Airborne Particulates and Household Dust in The Vicinity of a Copper Smelter - A Preliminary Report. PNWIS-APCA Paper 74-AP-31. Boise, Idaho. November 18, 1974.

108. Sahagian, B. M., et al. Transmural Movements of Zinc, Managanese, Cadmium and Mercury by Rat Small Intestine. Dept. of Internal Medicine, Washington Univ. School of Medicine and Cochran Veterans Admin. Hospital, St. Louis, Missouri. May 1967.

109. Saito, K., T. Sato, Y. Sato, and K. Niizuma. Effect of Low Lead Concentration on Human Body. Ind. Health (Japan). 11(3):85-93, Sept. 1973. (Presented at the 31st Japanese Society of Public Health. Sapporo, Japan. October 25027, 1972.)

110. Sawinsky A., J. Durszt, and G. Pasztor. Leukocyte Damage Through Exposure to Lead. (Leukozytenschaedigung bei Bleiexposition.) Text in German. Z. Ges. Hyg. Grenzg. (Berlin) 17(4):239-240, 1971.

111. Sayre, J. W., M.D., et al. House and Hand Dust as a Potential Source of Childhood Lead Exposure. Am J. Dis Child. Vol 127, February 1974.

112. Schegel, H., G. Kufner, and H. Leinberger. The Practice of Lead-Damage Prevention in the Metal-Working Industry. Translated from German. Lead and Environment 1972. West German Federal Ministry of Health, Commission for Environmental Dangers Lead Working Group. p. 111-130, 1972.

113. Schroeder, H. A. and D. K. Darrow. Relation of Trace Metals to Human Health. Environmental Affairs. 11(1):222-236.

114. Schuetzle, D. Arsenic in the Air in Puget Sound Area. Air Resources Division, University of Washington, Seattle. 1971.

115. Schulz, E. J. Arsenic as a Cause of Skin Cancer, with Notes on Its Occurrence in Pretoria. S.A. Medical Journal. (Presented at the Conference on Chemical Carcenogenesis. Johannesburg. July 1966.) p. 819-822.

116. Secchi, G. C., L. Alessio, and G. Cambiaghi. Studies on Ala-Dehydratase Activity of Erythrocytes in Subjects Not Occupationally Exposed to Lead, Living in Urban and Rural Areas. (Ricerche sull attivita ALA-deidrasica eritrocitaria di soggetti non exposti a contatto professionale con piombo ed abitanti in zone rurali ed urbane.) Text in Italian. Med. Lavoro. (Milan) 62:435-450, October 1971.

117. Selikoff, I. J., Ed. Hazards of Mercury. Academic Press. 4(1), March 1971.

118. Selyankina, K. P. and L. N. Alekseeva. Selenium and Tellurium in the Atmosphere Around Electrolyte Copper Plants. Hygiene and Sanitation. EPA and National Science Foundation. Washington, D.C. 35(3):431-432.

119. Statnick, R. M. Measurement of Sulfur Dioxide, Particulate, and Trace Elements in a Copper Smelter (Tacoma) and Roaster Reverberatory Gas Streams. EPA, NERC, Research Triangle Park, N.C. July 1974.

120. Sullivan, R. J. Preliminary Air Pollution Survey of Arsenic and Its Compounds. HEW. Raleigh, N.C. October 1969.

121. Sullivan, R. J. Preliminary Air Pollution Survey of Nickel and Its Compounds. HEW. Raleigh, N.C. October 1969.

122. Symon, K. and V. Bencko. Health Aspects of Air Pollution by Arsenic. (Presented at the 3rd Int. Clean Air Congress. Duesseldorf, West Germany. 1973.) p. A53-A55.

123. Tammes, P. M. and M. M. DeLint. Leaching of Arsenic from Soil. Neth. J. Agric. Sci. 17:128-132, 1969.

124. Tatarskaya, A. A. Cancer of the Respiratory Tract in Persons Working in the Nickel Industry. (Rak dykhatelynyky putei u rabot ayushchikh s nikelom.) Text in Russian. Vopr. Onkol. 13(6):58-60, 1967.

125. The Spector of Cancer. Environmental Science and Technology. 9(13):1116-1122, December 1975.

126. Tomita, K., G. Matsui, and M. Hirota. An Investigation on the Health Conditions of the Workers in a Cadmium Smelter. J. Ind. Health. (Tokyo) 5:700-708, Nov. 1963.

127. Trace Pollutant Emissions from the Processing of Metallic Ores. ORD, EPA. EPA-650/2-74-115. Oct. 1974.

128. Trakhtenberg, I. M., I. V. Savitskii and R. Ya. The Effect of Simultaneous Effects of Toxic and Temperature Factors. (OVliyanii Na Organizm Malykh Kontsentratsii Rtuti, K Volrosu o Sovmestnom Deistvii Taksicheskogo I Temperaturiogo Fakorov.) Text in Russian. Gigiena Truda I Prof. Zabolevaniya. 9:7-12, December 1965.

129. Tsuchiya, K. Results of a Long-Term Observation of Cadmium Workers. Japan Public Health Assoc. (Presented at the Sci. Res. Meeting. Tokyo. 1971.) Translated from Japanese. Redwood City, Calif. p. 170-185, April 1973.

130. U.S. Environmental Protection Agency. Helena Valley, Montana, Area Environmental Pollution Study. June 1971.

131. Wada, O., K. Toyokawa, T. Suzuki, S. Suzuki, Y. Yano, and K. Nakao. Response to a Low Concentration of Mercury Vapor. Arch. Environ. 19:485-488, Oct. 1969.

132. Wagner, W. L. Environmental Condition in U.S. Copper Smelters. NIOSH. Salt Lake City, Utah. HEW Publication # 75-158. April 1975.

133. Wixson, B. G. and J. C. Jennett. The New Lead Belt in the Forested Ozarks of Missouri. Environmental Science and Technology. 9(13):1128-1133, December 1975.

134. Worker, N. A. and B. B. Migicovsky. Effect of Vitamin D on the Utilization of Zinc, Cadmium and Mercury in The Chick. Canada Dept. of Agriculture. Ottawa, Ontario. 75(61):222-224.

135. World Health Organization. Who Technical Report Series No. 505. New York. 1972.

136. Yamada, Shin-ya. The Use of Metal Resources and Environmental Pollution - The Connection Between Occupational Disease and Pollution. (Kinzoku shigen no riyo to kankyo osen - shokugyobyo to kogai no tsunagari.) Text in Japanese. Science. 41:530-544, October 1971.

137. Yamaga, G., K. Omori, T. Tozawa, and Kazunori. The Effects of Low-Concentration Lead on the Body. (Teinodoen baduro no seitai eikyo.) Text in Japanese. (Presented at the 46th Annual Meeting of the Japan Society of Industrial Hygiene. April 1973.)

138. Yukio, S., N. Keitaro, M. Yutaka, O. Masahiro, K. Shigeru, T. Yuji, T. Tamotsu, O. Minoru, and M. Michito. Studies on Selenium Poisoning, Part I. Survey of Mineral Pollution Caused by Selenium Refining Plant. (Seren Chudoku ni Kan-suru Kenkyu. Kaiipo. Bo Seren Seiren Koho no Kogai ni Kan-suru Chosa Kenkyu.) Translated from Japanese. Tokushima Univ, Dept. of Hygiene. p. 846-854, Feb. 10, 1958.

2.0 AN EPIDEMIOLOGICAL ANALYSIS OF DISEASE SPECIFIC MORTALITY ASSOCIATED WITH PRIMARY Cu, Pb AND Zn SMELTING

As illustrated in Section 1.0, the health effects which could be produced as a result of copper, lead and zinc smelting are both varied and numerous. A rough estimate of the composition and quantities of residual metal emissions from existing smelters is available but it is only extensive enough to provide a general idea of what types of disease may be produced. In order to obtain a realistic idea of what diseases are occurring as a result of smelting, mortality data for 16 diseases has been examined for all counties containing and those surrounding primary Cu, Pb or Zn smelters which have been operational for at least 35 years. This approach was taken so that mortality profiles for counties containing smelters could be constructed. By generating a mortality profile, an overview of the mortality characteristics and potential health problems of the exposed population can be obtained.

Several cautionary notes must be made in relation to the analysis which follows. As in all statistical studies with wide scopes, many variables which could influence the associations discovered could not be investigated due to a lack of both available data and knowledge concerning all the pertinent variables. Precise quantification has not been attempted in this study because of the wide scope and limited accuracy of data concerning smelter characteristics employed. For example, the data utilized for smelter ore concentrate compositions is only considered to be of order of magnitude accuracy and is used accordingly. Also, it was not possible to adjust county mortality rates for such factors as population, population density, income, and other factors known to influence disease rates. Thus, the nature of this study does not lend itself to the computation of confidence intervals or other classical statistical methods for determining significance. This analysis was designed to determine what disease specific mortality is linked with primary Cu, Pb, and Zn smelting. In depth epidemiological analyses involving clinical evaluations of the affected members of the exposed populations will be required to substantiate the validity of the associations presented.

DATA AND METHODS

The basic strategy of this analysis was to generate mortality profiles for counties containing smelters as compared to all surrounding counties. A justification for this type of approach rests with the fact

that a smelter can be considered a point emissions source within a given county. Thus, if smelter emissions were producing adverse health effects, the county containing the smelter would be expected to bear the largest adverse effect as compared to all surrounding counties. All primary Cu, Pb or Zn smelters which have been operational for at least 35 years have been examined by comparing smelter-county mortality data for sixteen diseases. The sixteen diseases used to construct smelter-county mortality profiles are listed in Table 2-1 along with their ICD (International Classification of Diseases) Code. These diseases were chosen upon the basis of availability of data and known target organs of metal toxicity (Table 1-2). A 35 year period of continuous smelter operation was included as a necessary condition to allow sufficient time for the scope of the mortality data, and the latency periods for chronic diseases and cancer.

Mortality data for this analysis were taken from two sources which employed two different revisions of the ICD Code, but the procedures used for data reduction were completely analogous for both data sets. The most specific county mortality data were taken from the HEW publication, U.S. Cancer Mortality By County: 1950-1969.[1] This data source contained average annual age-adjusted cancer mortality rates and yielded information about the magnitude of death rates. The other source of data employed, Vital Statistics of the U.S. 1971 Volume II - Mortality, Part B,[2] had the advantage of not being limited to only cancer mortality. However, only the actual number of deaths which occurred in the counties were reported. Consequently, average annual age-adjusted mortality rates were not available, and no accurate estimate of the absolute magnitude of mortality rates could be made. As an alternative, percentages of total deaths in a county which were caused by specific diseases were calculated and substituted for absolute mortality rates for non-cancer causes of death. Although the latter mortality parameter is not as desirable or sensitive as mortality rates, it can still indicate excess disease specific mortality within a given county. For convenience, the summarized mortality data are presented in two parts with each part corresponding to the sources from which the data were taken. Preceding the summarized cancer mortality data, a detailed example of the data reduction procedure is given. No example of the reduction procedures used in data taken from the second source is presented because the two procedures were totally analogous except for a substitution of percentages of total deaths for mortality rates.

The following series of tables will demonstrate the procedure used in working up published mortality data. Table 2-2 was prepared by first listing the county containing the Bunker Hill lead smelter and then all counties contiguous to that county. All cancer mortality rates in Table 2-2 were taken from the reference footnoted in the table. Tables

Table 2-1. CAUSES OF DEATH USED TO GENERATE MORTALITY PROFILES FOR COUNTIES CONTAINING Cu, Pb OR Zn SMELTERS

Causes of Noncancer Deaths	International Classification of Disease (ICD) Code 8th Revision
Major cardiovascular disease	390 - 448
Hypertensive heart disease	402 & 404
Ischemic heart disease	410 - 413
Other forms of heart disease	420 - 429
Hypertension	400, 401, 403
Cerebrovascular disease	430 - 438
Arteriosclerosis	440
Other small vessel disease	441 - 448
Influenza and pneumonia	470 - 474, 480 - 486
Bronchitis, emphysema and asthma	490 - 493

Causes of Cancer Deaths	ICD Code 6th revision
All cancers combined	140 - 205
Cancer of the liver and biliary passages	155
Cancer of the trachea, lung and bronchus	162, 163
Cancer of the kidney	180
Cancer of the bladder	181
Cancer of the thyroid	144

Table 2-2. CANCER MORTALITY RATES FROM 1950-1969 FOR COUNTIES IN THE AREA OF THE BUNKER HILL LEAD SMELTER**

County, State	Average Annual Age-Adjusted Cancer Mortality Rates Per 100,000 Population (Male/Female)					
	Liver and Biliary Passages (ICD-155)	Trachea, Lung and Bronchus (ICD-162,163)	Kidney (ICD-180)	Bladder (ICD-181)	Thyroid (ICD-144)	All Combined
Shoshone, Idaho*	5.7/6.8	37.6/8.2	7.6/1.8	6.0/1.8	1.5/1.8	191.5/121.2
Latah, Idaho	3.9/4.1	16.5/2.1	3.2/3.1	7.7/2.1	.5/1.5	145.3/107.5
Kootenai, Idaho	4.0/5.0	26.4/4.9	.9/.7	4.5/1.8	1.3/2.1	139.0/120.0
Benewah, Idaho	4.6/5.2	27.6/5.5	3.9/3.5	1.2/0	0/1.8	143.9/134.5
Clearwater, Idaho	5.0/3.4	21.6/0	2.9/1.2	3.0/0	1.0/0	124.4/94.1
Bonner, Idaho	1.6/6.2	24.5/4.5	6.6/1.2	3.4/.6	.5/.6	134.1/115.6
Mineral, Montana	3.5/0	33.4/9.7	0/0	9.7/1.9	0/0	163.4/78.3
Sanders, Montana	2.7/7.0	21.5/4.0	3.9/1.4	2.2/1.0	0/0	127.8/100.3

*County contains Bunker Hill Pb smelter.

**All cancer rates were taken from the HEW publication: U.S. Cancer Mortality by County: 1950-1969. This source used the 6th revision of the ICD Code.

2-3A through 2-3F are simply a ranking of the average male and female cancer mortality rates for individual cancers and are based upon Table 2-2; "1" indicates the county with the lowest observed rate while "8" implies the highest cancer rate. For ease in comparison of the cancer mortality rankings of Shoshone County with that of other smelter containing counties, the numerical rankings in Table 2-3A through 2-3F can be converted to normalized percentile rankings. Thus Shoshone County was ranked sixth in bladder cancer out of 8 counties in the area (Table 2-3D) so its percentile ranking is $6/8 \cdot 100 - 0.5\ (1/8)$ or 69. Similarly, Shoshone County ranked eighth out of 8 counties in liver, lung, kidney and all cancers so its percentile ranking for these categories is $8/8 \cdot 100 - 0.5\ (1/8)$ or 94. The percentile cancer mortality rankings for Shoshone County have been summarized in the first entry of Table 2.4 denoted simply "Bunker Hill-Kellog, Idaho." Tables 2-4, 2-5 and 2-6 list only the percentile cancer mortality rankings for all Pb, Zn and Cu smelters examined, however, analogous procedures were used to obtain each table entry. Similarly, Tables 2-7, 2-8 and 2-9 list only the percentile non-cancer mortality rankings for all Pb, Zn and Cu smelters examined. For some diseases, there were an insufficient number of deaths in a particular cluster of counties to compute meaningful percentile mortality rankings; these cases are denoted with a dash (-) in Tables 2-4 through 2-9.

Table 2-3A. LIVER AND BILIARY PASSAGES CANCER MORTALITY RANKINGS FOR COUNTIES IN THE AREA OF THE BUNKER HILL LEAD SMELTER**

County, State	Average Annual Age-Adjusted Cancer Mortality Rates Per 100,000 Population (Male Rate + Female Rate)/2	Ranking
Shoshone, Idaho*	6.25	8
Latah, Idaho	4.0	3
Kootenai, Idaho	4.5	5
Benewah, Idaho	4.9	7
Clearwater, Idaho	4.2	4
Bonner, Idaho	3.9	2
Mineral, Montana	1.75	1
Sanders, Montana	4.85	6

*County contains Bunker Hill Pb smelter.

**All cancer rates were taken from the HEW publication: U.S. Cancer Mortality by County: 1950-1969. This source used the 6th revision of the ICD Code.

Table 2-3B. TRACHEA, LUNG AND BRONCHUS CANCER MORTALITY RANKINGS FOR COUNTIES IN THE AREA OF THE BUNKER HILL LEAD SMELTER**

County, State	Average Annual Age-Adjusted Cancer Mortality Rates Per 100,000 Population (Male Rate + Female Rate)/2	Ranking
Shoshone, Idaho*	22.9	8
Latah, Idaho	9.3	1
Kootenai, Idaho	15.65	5
Benewah, Idaho	16.55	6
Clearwater, Idaho	10.80	2
Bonner, Idaho	14.5	4
Mineral, Montana	21.55	7
Sanders, Montana	12.75	3

*County contains Bunker Hill Pb smelter

**All cancer rates were taken from the HEW publication: U.S. Cancer Mortality by County: 1950-1969. This source used the 6th revision of the ICD Code.

Table 2-3C. KIDNEY CANCER MORTALITY RANKINGS FOR COUNTIES IN THE AREA OF THE BUNKER HILL LEAD SMELTER**

County, State	Average Annual Age-Adjusted Cancer Mortality Rates Per 100,000 Population (Male Rate + Female Rate)/2	Ranking
Shoshone, Idaho*	4.7	8
Latah, Idaho	3.15	5
Kootenai, Idaho	0.8	2
Benewah, Idaho	3.7	6
Clearwater, Idaho	2.05	3
Bonner, Idaho	3.9	7
Mineral, Montana	0.0	1
Sanders, Montana	2.6	4

*County contains Bunker Hill Pb smelter.

**All cancer rates were taken from the HEW publication: U.S. Cancer Mortality by County: 1950-1969. This source used the 6th revision of the ICD Code.

Table 2-3D. BLADDER CANCER MORTALITY RANKINGS FOR COUNTIES IN THE AREA OF THE BUNKER HILL LEAD SMELTER**

County, State	Average Annual Age-Adjusted Cancer Mortality Rates Per 100,000 Population (Male Rate + Female Rate)/2	Ranking
Shoshone, Idaho*	3.9	6
Latah, Idaho	4.9	7
Kootenai, Idaho	3.15	5
Benewah, Idaho	0.6	1
Clearwater, Idaho	1.5	2
Bonner, Idaho	2.0	4
Mineral, Montana	5.8	8
Sanders, Montana	1.6	3

*County contains Bunker Hill Pb smelter.

**All cancer rates were taken from the HEW publication: U.S. Cancer Mortality by County: 1950-1969. This source used the 6th revision of the ICD Code.

Table 2-3E. THYROID CANCER MORTALITY RANKINGS FOR COUNTIES IN THE AREA OF THE BUNKER HILL LEAD SMELTER**

County, State	Average Annual Age-Adjusted Cancer Mortality Rates Per 100,000 Population (Male Rate + Female Rate)/2	Ranking
Shoshone, Idaho*	1.65	7
Latah, Idaho	1.0	6
Kootenai, Idaho	1.70	8
Benewah, Idaho	0.9	5
Clearwater, Idaho	0.5	3
Bonner, Idaho	0.55	4
Mineral, Montana	0.0	1.5
Sanders, Montana	0.0	1.5

*County contains Bunker Hill Pb smelter.

**All cancer rates were taken from the HEW publication: U.S. Cancer Mortality by County: 1950-1969. This source used the 6th revision of the ICD Code.

Table 2-3F. MORTALITY RANKINGS FOR ALL CANCERS COMBINED FOR COUNTIES IN THE AREA OF THE BUNKER HILL LEAD SMELTER**

County, State	Average Annual Age-Adjusted Cancer Mortality Rates Per 100,000 Population (Male Rate + Female Rate)/2	Ranking
Shoshone, Idaho*	156.4	8
Latah, Idaho	126.4	5
Kootenai, Idaho	129.5	6
Benewah, Idaho	139.2	7
Clearwater, Idaho	104.3	1
Bonner, Idaho	124.9	4
Mineral, Montana	120.9	3
Sanders, Montana	114.1	2

*County contains Bunker Hill Pb smelter.

**All cancer rates were taken from the HEW publication: U.S. Cancer Mortality by County: 1950-1969. This source used the 6th revision of the ICD Code.

Table 2-4. LEAD SMELTER CANCER MORTALITY DATA SUMMARY[1]

Facility	Percentile Cancer Mortality Rankings[2]					
	Liver and Biliary Passages (ICD-155)	Trachea, Lung and Bronchus (ICD-162,163)	Kidney (ICD-180)	Bladder (ICD-181)	Thyroid (ICD-144)	All Combined
Bunker Hill-Kellog, Idaho	94	94	94	69	82	94
Asarco-E. Helena, Montana	79	50	79	93	-	93
Asarco-El Paso, Texas	88	88	88	63	88	88
St. Joe-Herculaneum, Missouri	42	75	42	75	25	59

[1]All cancer rates were taken from the HEW publication: U.S. Cancer Mortality by County: 1950-1969. This source used the 6th revision of the ICD Code.

[2]All entries in the above table are percentile cancer mortality rankings of counties containing smelters as compared to all contiguous counties. The ranking was accomplished using average annual age-adjusted mortality rates per 100,000 population for the years 1950 through 1969. A representative county cancer rate was obtained by averaging white male and female rates.

Table 2-5. ZINC SMELTER CANCER MORTALITY DATA SUMMARY

Facility	Percentile Cancer Mortality Rankings[2]				
	Liver and Biliary Passages (ICD-155)	Trachea Lung and Bronchus (ICD-162-163)	Kidney (ICD-180)	Bladder (ICD-181)	All Combined
Bunker Hill-Kellog, Idaho *	94	94	94	69	94
Asarco-Amarillo, Texas	45	95	95	40	95
Asarco-Corpus Christi, Texas	32	94	19	82	94
Amax-Blackwell, Okla.	57	82	69	69	94
National Zinc-Bartlesville, Okla.	22	79	79	64	79
N.J.Zinc-Palmerton, Pa.	88	13	92	21	38
St. Joe-Monoca, Pa.	32	69	82	94	57

*This facility is also a Pb smelter.

[1]All cancer rates were taken from the HEW publication: U.S. Cancer Mortality by County: 1950-1969. This source used the 6th revision of the ICD Code.

[2]All entries in the above table are percentile cancer mortality rankings of counties containing smelters as compared to all contiguous counties. The ranking was accomplished using average annual age-adjusted mortality rates per 100,000 population for the years 1950 through 1969. A representative county cancer rate was obtained by averaging white male and female rates.

Table 2-6. COPPER SMELTER CANCER MORTALITY DATA SUMMARY[1]

Facility	Percentile Cancer Mortality Rankings[2]				
	Liver and Biliary Passages (ICD-155)	Trachea, Lung and Bronchus (ICD-162,163)	Kidney (ICD-180)	Bladder (ICD-181)	All Combined
*Asarco-Tacoma, Washington	63	79	46	59	88
Anaconda-Anaconda, Mon.	64	93	36	50	50
Kennecott-McGill, Nevada	69	32	82	82	69
Kennecott-Garfield, Utah	64	50	93	57	79
Cities Service-Copperhill, Tennessee	96	79	63	79	96
Asarco-El Paso, Texas***	88	88	88	63	88
Kennecott-Hurley, New Mexico	70	90	90	30	90
Inspiration-Miami, Ariz.	79	93	93	93	93
Asarco-Hayden, Ariz.	75	95	95	80	95
Phelps Dodge**-Morenci, Ariz.	36	50	50	64	22
Phelps Dodge-Douglas, Ariz.	93	79	22	93	79

*Average ranking of Pierce and King Counties. **Two of six contiguous counties also contain Cu smelters. ***This facility is also a Pb smelter.

[1]All cancer rates were taken from the HEW publication: U.S. Cancer Mortality by County: 1950-1969. This source used the 6th revision of the ICD Code.

[2]All entries in the above table are percentile cancer mortality rankings of counties containing smelters as compared to all contiguous counties. The ranking was accomplished using average annual age-adjusted mortality rates per 100,000 population for the years 1950-1969. A representative county cancer rate was obtained by averaging white male and female rates.

Table 2-7. LEAD SMELTER NON-CANCER MORTALITY DATA SUMMARY[1]

Facility	Percentile Mortality Rankings for Percentages of Total Deaths Due to the Indicated Disease[2]										
	ICD Code[3]										
		390-448	402 & 404	410-413	420-429	400, 401, 403	430-438	440	441-448 480-486	470-474	490-493
Bunker Hill - Kellog, Idaho		19	44	57	7	-	19	94	19	7	32
Asarco - E. Helena, Montana		22	36	64	36	64	64	7	79	36	79
Asarco - El Paso, Texas		38	88	13	63	63	63	63	63	63	63
St. Joe - Herculaneum, Missouri		25	50	43	25	25	59	25	75	75	42

1. All mortality data was taken from: Vital Statistics of the U.S. 1971. Volume II-Mortality, Part B. This source used the 6th revision of the ICD Code.
2. All entries in the above table are percentile non-cancer mortality rankings of counties containing smelters as compared to all contiguous counties. The ranking was accomplished by using the percentages of total deaths due to the above indicated diseases for each of the counties examined.
3. See Table 2.1 for the ICD Code key.

Table 2-8. ZINC SMELTER NON-CANCER MORTALITY DATA SUMMARY[1]

Facility	Percentile Mortality Rankings for Percentages of Total Deaths Due to the Indicated Disease[2]									
	ICD Code[3]									
	390-448	402 & 404	410-413	420-429	400, 401, 403	430-438	440	441-448	470-474 480-486	490-493
Bunker Hill - Kellog, Idaho*	19	44	57	7	-	19	94	19	7	32
Asarco - Amarillo, Texas	77	68	95	50	77	59	40	59	68	31
Asarco - Corpus Christi, Texas	44	63	69	19	82	19	57	32	32	44
Amax - Blackwell, Oklahoma	82	7	82	32	82	57	44	69	32	57
National Zinc - Bartlesville, Oklahoma	64	93	64	7	79	50	64	7	36	50
N.J. Zinc - Palmerton, Pennsylvania	38	71	13	46	34	96	46	63	96	4
St. Joe - Monaca, Pennsylvania	19	82	19	94	32	94	44	44	94	57

* This facility is also a Pb smelter.

1. All mortality data was taken from: Vital Statistics of the U.S. 1971. Volume II-Mortality, Part B. This source used the 6th revision of the ICD Code.
2. All entries in the above table are percentile non-cancer mortality rankings of counties containing smelters as compared to all contiguous counties. The ranking was accomplished by using the percentages of total deaths due to the above indicated diseases for each of the counties examined.
3. See Table 2.1 for the ICD Code key.

Table 2-9. COPPER SMELTER NON-CANCER MORTALITY DATA SUMMARY[1]

	Percentile Mortality Rankings for Percentages of Total Deaths Due to the Indicated Disease[2]										
	ICD Code[3]										
Facility		390-448	402 & 404	410-413	420-429	400, 401, 403	430-438	440	441-448	470-474 480-486	490-493
Asarco - Tacoma, Washington		63	29	71	38	29	79	21	13	29	79
Anaconda - Anaconda, Montana		93	79	93	79	-	50	36	22	7	93
Kennecott - McGill, Nevada		19	-	94	13	-	44	57	82	82	13
Kennecott - Garfield, Utah		36	79	22	50	79	50	64	50	50	36
Cities Service - Copperhill, Tennessee		54	54	63	63	21	38	79	54	54	29
Asarco - El Paso*, Texas		38	88	13	63	63	63	63	63	63	63

* This facility is also a Pb smelter.

1. All mortality data was taken from: Vital Statistics of the U.S. 1971. Volume II - Mortality, Part B. This source used the 6th revision of the ICD Code.
2. All entries in the above are percentile non-cancer mortality rankings of counties containing smelters as compared to all contiguous counties. The rankings was accomplished by using the percentages of total deaths due to the above indicated diseases for each of the counties examined.
3. See Table 2.1 for the ICD Code key.

Table 2-9 (continued). COPPER SMELTER NON-CANCER MORTALITY DATA SUMMARY[1]

Facility	Percentile Mortality Rankings for Percentages of Total Deaths Due to the Indicated Disease[2] ICD Code[3]										
		390-448	402 & 404	410-413	420-429	400, 401, 403	430-438	440	441-448	470-474 480-486	490-493
Kennecott - Hurley, New Mexico		50	90	70	10	30	50	70	20	50	70
Inspiration - Miami, Arizona		50	64	50	22	93	7	7	50	64	36
Asarco - Hayden, Arizona		55	75	55	25	95	15	5	65	75	35
Phelps Dodge - Morenci, Arizona**		22	-	36	79	-	36	64	64	7	22
Phelps Dodge - Douglas, Arizona		50	29	22	36	79	64	50	79	36	64

** Two of six contiguous counties also contain Cu smelters.

1. All mortality data was taken from: Vital Statistics of the U.S. 1971. Volume II - Mortality, Part B. This source used the 6th revision of the ICD Code.
2. All entries in the above are percentile non-cancer mortality rankings of counties containing smelters as compared to all contiguous counties. The rankings was accomplished by using the percentages of total deaths due to the above indicated diseases for each of the counties examined.
3. See Table 2.1 for the ICD Code key.

OBSERVED MORTALITY ASSOCIATIONS FOR COUNTIES CONTAINING Cu, Pb, OR Zn SMELTERS

Tables 2-4 through 2-9 presented mortality profiles for each county which contained a Cu, Pb or Zn smelter chosen for this analysis. Certain disease specific mortality associations appeared when the summary data in these tables was inspected closely; these mortality associations are listed in Tables 2-10 through 2-12. It should be emphasized again that the basis for determining excess mortality was a comparison between mortality parameters in smelter-containing versus all contiguous counties. It is interesting to note that only two excess mortality associations, cancer of the trachea, lung and bronchus and all cancers combined, were observed for the majority of Cu, Pb and Zn smelters examined. Another particularly striking industry specific association was the association of excess mortality due to hypertension and hypertensive heart disease with only the majority of zinc smelters.

The five diseases for which excess mortality was associated with the majority of lead smelters were (Table 2-10):

1. All cancers combined.
2. Cancer of the liver and biliary passages.
3. Cancer of the trachea, lung and bronchus.
4. Cancer of the kidney.
5. Cancer of the bladder.

If mortality from these diseases was not associated with counties containing primary lead smelters, then the average disease specific percentile mortality ranking of all these counties would be expected to be 50 for each of the five diseases. From Tables 2-4 and 2-7, the average percentile mortality ranking of all the counties containing lead smelters examined, for each of the five diseases can be calculated to be:

1. 76 = (94 + 79 + 88 + 42)/4 for cancer of the liver and biliary passages,
2. 77 for cancer of the trachea, lung and bronchus,
3. 76 for cancer of the kidney,
4. 75 for cancer of the bladder,

Table 2-10. OBSERVED MORTALITY ASSOCIATIONS FOR COUNTIES CONTAINING PRIMARY LEAD SMELTERS

1. An increase in the percentage of total deaths or high absolute mortality rates for the following causes of death are associated with the majority of Pb smelters examined ($\geq$ 75%):

   a. All cancers combined.

   b. Cancer of the liver and biliary passages.

   c. Cancer of the trachea, lung and bronchus.

   d. Cancer of the kidney.

   e. Cancer of the bladder.

2. Both the Asarco-El Paso, Texas and the Asarso-East Helena, Montana Pb smelters have associated, elevated percentages of total deaths due to bronchitis, emphysema and asthma.

3. The St. Joe-Herculaneum, Missouri and the Asarco-El Paso Pb smelters have associated, elevated percentages of total deaths due to influenza and pneumonia.

4. The Bunker Hill-Kellog, Idaho and the Asarco-El Paso, Texas Pb smelters have associated, elevated percentages of total deaths due to arteriosclerosis.

5. The Asarco-E. Helena, Montana and the St. Joe-Herculaneum, Missouri Pb smelters have associated, elevated percentages of total deaths due to small vessel disease other than arteriosclesosis.

6. St. Joe-Herculaneum, Missouri is the only Pb smelter which does not have associated high mortality rates for cancer of the kidney, thyroid and liver and biliary passages.

Table 2-11. OBSERVED MORTALITY ASSOCIATIONS FOR COUNTIES CONTAINING PRIMARY ZINC SMELTERS

1. An increase in the percentage of total deaths or high absolute mortality rates for the following causes of death are associated with the majority of Zn smelters examined ($\geq$ 67%):

   a. Hypertension.

   b. Hypertensive heart disease.

   c. All cancers combined.

   d. Cancer of the trachea, lung and bronchus.

   e. Cancer of the kidney.

   f. Cancer of the bladder.

2. Both the Asarco-Amarillo, Texas and the Amax-Blackwell, Oklahoma Zn smelters have associated, elevated percentages of total deaths due to major cardiovascular disease and ischemic heart disease.

3. Both the New Jersey Zinc-Palmerton, Pennsylvania and the St. Joe-Monaca, Pennsylvania Zn smelters have associated, elevated percentages of total deaths due to cerebrovascular disease and influenza and pneumonia.

4. New Jersey Zinc-Palmerton, Pennsylvania and St. Joe-Monaca, Pennsylvania are the only Zn smelters examined which do not have associated, elevated percentages of total deaths due to hypertension.

5. Both the Asarco-Amarillo, Texas and the Asarco-Corpus Christi, Texas Zn smelters have associated, extremely high mortality rates for cancer of the trachea, lung and bronchus.

NOTE: The Bunker Hill-Kellog, Idaho facility is both a zinc and lead smelter. For this reason, the Bunker Hill facility was not used in constructing zinc smelter mortality associations.

Table 2-12. OBSERVED MORTALITY ASSOCIATIONS FOR COUNTIES CONTAINING PRIMARY COPPER SMELTERS

1. High, absolute mortality rates for the following causes of death are associated with the majority of Cu smelters examined ($\geq$ 70%):

   a. All cancers combined.

   b. Cancer of the liver and biliary passages.

   c. Cancer of the trachea, lung and bronchus.

2. Increased percentages of the total deaths due to hypertensive heart disease are associated with the following Cu smelters (40% of those examined):

   a. Anaconda - Anaconda, Montana.

   b. Kennecott - Garfield, Utah.

   c. Kennecott - Hurley, New Mexico.

   d. Asarco - Hayden, Arizona.

3. Increased percentages of the total deaths due to hypertension are associated with the following Cu smelters (40% of those examined):

   a. Kennecott - Garfield, Utah.

   b. Inspiration - Miami, Arizona.

   c. Asarco - Hayden, Arizona.

   d. Phelps Dodge - Douglas, Arizona.

4. Increased percentages of the total deaths due to ischemic heart disease are associated with the following Cu smelters (30% of those examined):

   a. Anaconda - Anaconda, Montana.

   b. Kennecott - McGill, Nevada.

   c. Kennecott - Hurley, New Mexico.

Table 2-12.(continued) OBSERVED MORTALITY ASSOCIATIONS FOR COUNTIES CONTAINING PRIMARY COPPER SMELTERS

5. Increased percentages of the total deaths due to bronchitis, emphysema and asthma are associated with the following Cu smelters (30% of those examined):

   a. Asarco - Tacoma, Washington.

   b. Anaconda - Anaconda, Montana.

   c. Kennecott - Hurley, New Mexico.

6. Both the Anaconda - Anaconda, Montana and the Kennecott - Hurley, New Mexico Cu smelters have associated, elevated percentages of total deaths due to hypertensive heart disease, ischemic heart disease, and bronchitis, emphysema and asthma.

7. The Kennecott - Garfield, Utah and the Asarco - Hayden, Arizona Cu smelters have associated, elevated percentages of total deaths due to hypertension and hypertensive heart disease.

8. High, absolute mortality rates for cancer of the kidney are associated with the following Cu smelters (50% of those examined):

   a. Kennecott - McGill, Nevada.

   b. Kennecott - Garfield, Utah.

   c. Kennecott - Hurley, New Mexico.

   d. Inspiration - Miami, Arizona.

   e. Asarco - Hayden, Arizona.

9. High, absolute mortality rates for cancer of the bladder are associated with the following Cu smelters (50% of those examined):

   a. Kennecott - McGill, Nevada.

   b. Cities Service - Copperhill, Tennessee.

   c. Inspiration - Miami, Arizona.

Table 2-12. (continued) OBSERVED MORTALITY ASSOCIATIONS FOR COUNTIES CONTAINING PRIMARY COPPER SMELTERS

d. Asarco - Hayden, Arizona.

e. Phelps Dodge - Douglas, Arizona.

NOTE: The Asarco - El Paso, Texas facility is both a copper and lead smelter. For this reason, the Asarco facility was not used in constructing copper smelter mortality associations.

5. 84 for all cancers combined.

It is clear that the average percentile mortality ranking for each of these five diseases is above 50. Similar calculations for the excess disease-specific mortality associated with the majority of either Cu or Zn smelters lead to an identical conclusion.

The only comparison which can be made between mortality rates in smelter-containing counties and national mortality rates is for cancer mortality data. For this purpose, a companion document to the one used for generating cancer mortality profiles for smelter-containing counties, Atlas of Cancer Mortality for U.S. Counties: 1950-1969,[4] can be employed. This atlas has tabulated the counties which have significantly elevated cancer mortality rates as compared to national rates. A county with a significantly elevated cancer rate was considered to be one whose 95 percent confidence interval for a specific cancer mortality rate did not overlap with the 95 percent confidence interval for the national mortality rate for that same cancer. For three of the five cancers which were examined for all Cu, Pb and Zn smelters, large percentages of smelter-containing counties with significantly high mortality rates as compared to the percentages of counties in the continental U.S. with significantly high mortality rates were found. For cancer of the liver and biliary passages, 3 percent of all continental U.S. counties had significantly elevated male rates and 7 percent had significantly elevated female rates. Of the twenty Cu, Pb or Zn smelter-containing counties examined in this study, 10 percent had significantly elevated male mortality rates for cancer of the liver and biliary passages and 20 percent had significantly elevated female rates. Similarly, the percentage of continental U.S. counties which had significantly elevated male/female rates versus the percentage of smelter-containing counties with significantly elevated male/female rates was 7 percent/5 percent versus 20 percent/20 percent for cancer of the trachea, lung and bronchus and 5 percent/5 percent versus 10 percent/10 percent for all cancers combined.

Table 2-13 lists the smelters and smelter-containing counties which had significantly elevated cancer mortality rates as compared to national mortality rates. Only the three cancers mentioned previously are included in the table because kidney and bladder cancer mortality rates in smelter-containing counties were not significantly elevated when compared to national rates. The copper smelter located in Tacoma, Washington is situated on the border of two counties. For this reason, both of the counties which the smelter borders were considered as smelter-containing counties.

Table 2-13. COPPER, LEAD OR ZINC SMELTER-CONTAINING COUNTIES WITH SIGNIFICANTLY ELEVATED CANCER MORTALITY RATES

Facility	County of Location	Type of Smelter	Significantly Elevated Mortality Rates As Compared to National Mortality Rates*					
			Cancer of the Liver & Biliary Passages		Cancer of the Trachea Lung and Bronchus		All Cancers Combined	
			Male	Female	Male	Female	Male	Female
Asarco-El Paso, Texas	El Paso	Lead/ copper	†	†		†		
St. Joe-Herculaneum, Missouri	Jefferson	Lead				†		
Asarco - Amarillo, Texas	Potter	Zinc			†			
Asarco-Corpus Christi, Texas	Nueces	Zinc	†		†	†		
N.J. Zinc-Palmerton, Pennsylvania	Carbon	Zinc		†				†
St. Joe-Monaca, Pennsylvania	Beaver	Zinc		†	†		†	†
Asarco-Tacoma, Washington	King**	Copper				†	†	
Anaconda-Anaconda, Montana	Deerlodge	Copper			†			
Phelps Dodge-Douglas, Arizona	Cochise	Copper		†				

* All data for this table was taken from Reference 4. Significance is defined to be the non-overlap of 95% confidence intervals for county and national mortality rates.

** King County is one of the two counties on which this smelter borders.

EXPLANATIONS FOR THE OBSERVED MORTALITY ASSOCIATIONS OF COUNTIES CONTAINING Cu, Pb OR Zn SMELTERS

Although knowledge of which diseases are claiming the lives of inhabitants of smelter-containing counties is important, knowledge of why certain disease specific excess mortality is associated with primary Cu, Pb and Zn smelting is of far greater importance. In an attempt to explain the mortality associations presented in the previous section, two sets of fundamental smelting characteristics were examined. First, the unit processes employed in smelting which are detailed in the main body of this report were listed for each facility investigated, and observed smelter mortality associations were compared with this list to see if unit processes influenced disease specific mortality. To avoid redundancy, no descriptions of the unit processes associated with excess mortality will be presented in this section; readers are therefore urged to refer to the appropriate sections of the main body of this report for unit process descriptions. The second set of smelter characteristics examined were the compositions of the ore concentrates smelted. Unfortunately, lead smelter ore concentrate compositions were not available and were necessarily omitted. All ore concentrate composition data were taken from unpublished EPA sources and are only considered to be of order of magnitude accuracy. Consequently, Tables 2-15 and 2-16 only list ranges of element concentrations in zinc and copper ore concentrates for the nine elements examined. Table 2-14 gives the definitions of levels of element concentrations in ore concentrates used for this analysis.

No associations between disease specific excess mortality which was observed for the majority of either Cu, Pb or Zn smelters and unit processes could be determined from this analysis. In other words, the disease specific excess mortality linked with the majority of either primary lead, copper or zinc smelters is probably not a result of the unit processes employed. The most probable explanation for these industry specific mortality associations (see Tables 2-10, entry [1]; 2-11, entry [1]; and 2-12, entry [1]) is the common chararacteristics of a lead, copper or zinc ore concentrate. For example only zinc ore concentrates characteristically contain high concentrations of both zinc and cadmium (Table 2-15).

Due to a lack of ore concentrate composition data for lead smelters, the ability to explain the observed mortality associations of these facilities was severely limited. However, the absence of one lead smelting unit process was found to be associated with the absence of excess mortality from three diseases. The St. Joe smelter located in Herculaneum, Missouri was the only lead smelter examined in this study which did not use slag fuming furnaces, a significant emissions source

Table 2-14. DEFINITION OF RANGES OF ELEMENT CONCENTRATIONS IN Cu AND Zn SMELTER ORE CONCENTRATES

	Range of Element Concentration in Ore Concentrates (All entries are in ppm unless otherwise noted)		
Element	Low (L)	Medium (M)	High (H)
As	< 200	200 - 999	≥ 1000
Hg	< 1	1 - 9.9	≥ 10
Cd	< 100	100 - 999	≥ 1000
Pb	< 1000	1000 - 9,999	≥ 1%
Ni	< 20	20 - 99	≥ 100
Mn	< 100	100 - 999	≥ 1000
Zn	< 1%	1% - 29.9%	≥ 30%
Sb	< 100	100 - 999	≥ 1000
Se	< 5	5 - 99	≥ 100

Table 2-15. QUANTITIES OF NINE ELEMENTS IN Zn SMELTERS ORE CONCENTRATES

Facility	Quantities of Elements in Ore Concentrates (see Table 2.14)								
	As	Hg	Cd	Pb	Ni	Mn	Zn	Sb	Se
Bunker Hill - Kellog, Idaho	-	-	-	-	-	-	-	-	-
Asarco - Amarillo, Texas	H	H	H	M	L	M	H	L	-
Asarco - Corpus Christi, Texas	H	M	H	M	L	H	H	M	-
Amax - Blackwell, Oklahoma	L	M	H	M	M	M	H	L	L
National Zinc - Bartlesville, Oklahoma	M	L	H	M	L	M	H	L	L
N. J. Zinc - Palmerton, Pa	L	L	H	L	L	M	H	L	-
St. Joe - Monaca, Pa	L	H	H	L	L	M	H	L	M

NOTE: L = Low, M = Medium, H = High

Table 2-16. QUANTITIES OF NINE ELEMENTS IN Cu SMELTER ORE CONCENTRATES

Facility	Quantities of Elements in Ore Concentrates (See Table 2.14)								
	As	Hg	Cd	Pb	Ni	Mn	Zn	Sb	Se
Asarco - Tacoma, Washington	H	H	M	M	H	H	M	H	-
Anaconda - Anaconda, Montana	H	L	M	M	M	M	M	M	M
Kennecott - McGill, Nevada	L	M	L	L	H	M	L	M	M
Kennecott, Garfield, Utah	M	L	L	M	H	M	L	M	H
Cities Service - Copperhill, Tennessee	-	-	-	-	-	-	-	-	-
*Asarco - El Paso, Texas	L	L	L	H	H	M	L	L	-
Kennecott, Hurley, New Mexico	L	L	L	L	H	M	L	M	M
Inspiration - Miami, Arizona	-	-	-	-	-	-	-	-	-
Asarco - Hayden, Arizona	L	M	M	M	H	M	M	L	-
Phelps Dodge - Morenci, Arizonz	L	M	L	L	M	M	L	L	M
Phelps Dodge - Douglas, Arizona	L	M	M	M	M	M	M	M	M

NOTE: L = Low, M = Medium, H = High

*Also a Pb smelter

for As, Pb, Zn and Cd. This same smelter was also the only lead smelter examined which did not have associated elevated mortality rates for cancer of the kidney, thyroid, and liver and biliary passages.

Upon comparison of zinc ore concentrate compositions and zinc smelter mortality associations, several significant facts appeared. Excess mortality from hypertension and hypertensive heart disease was noted for 4 out of the 6 primary zinc smelters investigated (Table 2-11). The only two zinc smelters examined which did not have associated excess hypertension mortality, New Jersey Zinc - Palmerton, Pennsylvania and St. Joe - Monaca, Pennsylvania, were also the only two that smelted ore concentrates with low levels of lead. These same two primary zinc smelters were the only two examined which had associated excess mortality from cerebrovascular disease, and influenza and pneumonia. It appears that low levels of lead in zinc ore concentrates drastically alters the mortality profiles for counties containing these facilities. Only 2 of the 6 smelters examined which only smelt zinc ores, Asarco - Corpus Christi, Texas and Asarco - Amarillo, Texas, had percentile cancer of the trachea, lung and bronchus mortality rankings greater than 90 (Table 2-5). In fact, the Asarco - Corpus Christi facility had significantly elevated rates for both male and female cancer of the trachea, lung and bronchus, while the Asarco - Amarillo smelter had significantly elevated male cancer mortality rates for the same disease (Table 2-13). These same two zinc smelters are also the only two which process zinc ore concentrates with high levels of arsenic, an element previously linked epidemiologically with human lung cancer (see Section 1.0). The counties containing smelters which process zinc ore concentrates with high levels of arsenic thus seem to be at great risk of having significantly high mortality rates for cancer of the trachea, lung and bronchus.

Explanations for the mortaility associations of copper smelters based upon unit processes and ore concentrate compositions are not nearly as strong as those presented for lead and zinc smelters. No associations between unit processes and copper smelter associated mortality could be determined from this analysis. Although copper ore concentrate composition data yielded some clustering of ore concentrate characteristics and disease specific excess mortailty, no clear cut associations such as those found for zinc smelters could be determined. The reader is thus cautioned against overinterpretation of the ore concentrate composition versus excess mortality associations presented for copper smelters.

Most of the copper smelters which had associated excess mortality from hypertension or hypertensive heart disease smelted ore concentrates with medium levels of lead and high levels of nickel. Both of the

copper smelters which had associated excess mortality from hypertension and hypertensive heart disease, Kennecott - Garfield, Utah and Asarco - Hayden, Arizona, also had medium levels of lead and high levels of nickel in their ore concentrates. All copper smelters with associated excess mortality from ischemic heart disease had medium levels of both antimony and selenium in the ore concentrates which they smelted. Excess mortality from cancer of the kidney around copper smelters was always accompanied by ore concentrates high in nickel. Once again, the reader is cautioned against overinterpretation of these copper smelter mortality versus ore concentrate composition associations.

Specific References for Section 2.0

1. Mason, T.J., et al. U.S. Cancer Mortality by County: 1050-1969. DHEW Publication # (NIH) 74-615.

2. Vital Statistics of the U.S. 1971. Volume II - Mortality, Part B. Table 7-9. P.H.S. HEW (HRA) 75-1102. Rockville, Maryland. 1974.

3. Daniel, W.W. Biostatistics: A Foundation for Analysis in the Health Sciences. John Wiley and Sons, Inc., New York, 1974.

4. Mason, T.J., et al. Atlas of Cancer Mortality for U.S. Counties: 1950-1969. DHEW Publication # (NIH) 75-780.

3.0 CONCLUSIONS AND AN ASSESSMENT OF THE PUBLIC HEALTH HAZARD OF PRIMARY Cu, Pb AND Zn SMELTING

It is clear from the epidemiological analysis presented in the previous section that excess mortality is associated with counties containing primary Cu, Pb and Zn smelters. Elevated rates of cancer of the trachea, lung and bronchus and all cancers combined have been found in association with the majority of Cu, Pb and Zn smelters. Excess mortality from cancer of the kidney and bladder was linked with the majority of Pb and Zn smelters, while elevated rates of cancer of the liver and biliary passages was associated with the majority of the Pb and Cu smelters. Only the majority of Zn smelters had associated excess mortality from hypertension and hypertensive heart disease. Perhaps one or two of these associations could be attributed to some strange quirk of probability, but the implication of all these mortality associations combined is undeniable. Counties containing primary Cu, Pb or Zn smelters are exposed to a significant public health hazard.

The two elements contained in smelter ore concentrates which were most strongly linked with excess mortality by the analysis presented in Section 2.0 are arsenic and lead. Smelting zinc ore concentrates with high levels of arsenic was clearly associated with significantly elevated rates of cancer of the trachea, lung and bronchus. Numerous previously published epidemiological studies have suggested the relationship between excessive cadmium exposure and hypertension. The results of this study strongly show that a relatively narrow range of lead exposure must also be present before excess hypertension mortality is observed. Excess hypertension mortality was only noted in association with facilities which processed ore concentrates with medium levels of lead. The smelting of ore concentrates with either low or high levels of lead was not associated with excess hypertension mortality regardless of the levels of cadmium present.

The solution for arsenic induced lung cancer around facilities which smelt high arsenic zinc ore concentrates is fairly straight forward. Sufficient reduction of the levels of arsenic emitted by these facilities should solve the problem. Excess mortality from hypertension as a result of a synergistic relationship between cadmium and lead poses a somewhat more complex regulation problem. Zinc and copper smelters which have intermediate concentrations of lead in their ore concentrates will probably need to reduce lead emissions to prevent excess hyperten-

sion and hypertensive heart disease mortality. However, lead smelters which have much higher lead emissions will need to achieve a far greater relative emissions reduction to bring lead emissions to a safe absolute level. If lead smelters were only required to achieve a percent lead emissions reduction equivalent to that required to eliminate Pb associated excess mortality around Cu or Zn smelters, excess mortality from hypertension or hypertensive heart disease which was not found to be associated with lead smelters might result.

Three other elements present in copper ore concentrates were more weakly linked with disease specific excess mortality by this study. The possible synergistic action of antimony and selenium in relation to excess mortality from ischemic heart disease should be investigated, and appropriate emissions standards set as necessary. Along the same lines, the association of high nickel levels in copper ore concentrates and elevated levels of cancer of the kidney should be examined further. Nickel has previously been linked epidemiologically with occupational induced cancers (see Section 1.0). Thus, its possible role in induction of kidney cancer, and the possible need for regulation of nickel emissions is not a great surprise.

One specific smelting unit process was linked with excess mortality by this study. The use of slag fuming furnaces by lead smelters was strongly associated with elevated rates of cancer of the kidney, thyroid, and liver and biliary passages. It is probable that strict emissions control of slag fuming furnaces or unit process modifications will be required to eliminate this public health risk.

In conclusion, it must be stated that the associations made concerning smelter induced excess mortality and its most probable causes reflect the current state of knowledge as embodied in the literature and as established by the epidemiological analysis undertaken by this study. Further investigations involving clinical evaluations of the exposed populations should validate and expand many of the associations just presented.

APPENDIX B

ECOLOGICAL EFFECTS

In the past several years efforts have been made to determine the ecological impacts resulting from mining, milling, transport, and smelting of nonferrous metals. There are five major sources of pollutants from the copper, zinc, and lead industries; (1) mine water, (2) mill waters, (3) solid wastes from both mining and milling (tailings and spoil), (4) concentrates resulting from the milling process, and (5) smelter emissions.

Water from mining and milling operations often receives treatment in at least a single tailing pond before release to receiving streams; the discharge does, however, cause some changes in receiving streams. Turbidity and suspended solids are usually increased greatly. Heavy metals are often associated with these suspended solids. Chemicals related to flotation processes often cause excessive productivity such as algae blooms or excessive growth in aquatic vegetation which can cause blocking of photosynthetic energy input and thus eliminate normal stream populations. The effects or toxicity of heavy metals on algae, benthic organisms, and various fish species have been studied for many years. Most studies have been in the form of bioassays. Though they often yield varying results, the bioassays have shown that heavy metals such as copper, lead, and zinc may be toxic to aquatic life in the concentrations reached in receiving streams. Bioassays, however, are only indicators of what may actually take place, and to date very little on-site work has been done. Until this is accomplished, the effects of mining and milling on aquatic life will not be known conclusively.

For the past 60 years the Sudbury, Ontario, area has been an important center for mining and smelting of metals, including copper, nickel, and cobalt. This is one of few areas where field studies of the terrestrial and aquatic impacts of smelters have been undertaken. The area has been heavily contaminated by a number of heavy metals, notably nickel, copper, and iron.

Contamination of water bodies has occurred in four principal ways:

1. Aerial contamination occuring directly with dust fallout into water bodies. Metal-containing dust is blown off the land.

2. As a result of the loss of terrestrial vegetation cover, the soil is subject to extreme leaching and erosion. This, together with the increased acidity of the soil, which increases the solubility of heavy metals in the soil, causes runoff into the water bodies. This runoff water carries large quantities of heavy metals.

3. Water percolates through slag heaps and mine tailings dumped from the mining operation and thus transfers heavy metals to water bodies.

4. Acidic mine streams enter water bodies or are pumped to the surface adding a further source of metals.

The flora and fauna of several lakes in the mining and smelter area have been severely depleted.[1] Since copper, and particularly copper sulfate, is the most commonly used algicide in reservoirs and water supplies, this depletion of algal flora is hardly surprising.

A study of copper pollution in the Churnet and Dove Rivers in the United Kingdom showed that discharge from copper works severely reduced algal numbers. Effects on species numbers and composition could be detected for a distance of 30 miles downstream, at which point levels of 0.1 ppm Cu were recorded.[2] Animal numbers were similarly affected, and animal life was eliminated for a distance of 10 miles.

The Sudbury studies also indicate that a combination of high concentrations of copper and nickel is likely to be especially harmful to living organisms, since it has been shown that copper and nickel act synergistically in their toxicity to algae and that this synergism is increased at low pH levels. Nickel by itself, at a concentration of 0.5 ppm, has killed laboratory strains of algae.

Little information is available on the effects of lead smelters on local ecology. To date the only field study has been in the area of the New Lead Belt in Missouri. Temporary excessive algae and aquatic vegetational growth has been observed in receiving streams. Diversity in species has been reduced in some receiving streams. Though this does not necessarily mean that the quality of the stream has been degraded, it does mean that species used in the index have changed.

Other studies have shown that in soft water lead may be very toxic to fish at a concentration of 0.1 milligram per liter. In hard water this concentration is not toxic.[3]

Zinc exhibits its greatest toxicity toward fish and aquatic organisms. In soft water, concentrations of zinc ranging from 0.1 to 1.0 milligram per liter have been reported to be lethal, but calcium is antagonistic toward such toxicity. Sensitivity to zinc varies with species, age, and condition of the fish, as well as the physical and chemical characteristics of the water.[4]

Though arsenic is generated in most smelter operations and its effects on humans are well known, little field data are available as to its effects on aquatic organisms. A study pertaining to sewage treatment processes showed that arsenic concentrations of 3 to 20 milligrams per liter have not harmed aquatic insects such as immature dragonflies, damselflies, and mayflies.[5]

The terrestrial environment, like the aquatic environment, is affected by smelter operation. A study near a copper smelter in Wollongong, New South Wales, Australia, showed statistically significant relationships between distance from the smelter and concentrations of heavy metals in the vegetation. Significant relationships were found between distance from the smelter (for a radius of 3 kilometers and beyond) and the levels of easily extractable copper and zinc in the soil, and also between distance from the smelter and the content of copper and zinc in herbage.[6,7] Over a 0.5 kilometer distance, significant correlations were found for copper and nickel. Highly significant correlations were found between distance from the chimney and the levels of easily extractable copper, zinc, lead, and cadmium in the soil.

Some bioassays have shown that selected plants absorb these metals into their various structures; toxic effects have been shown. However, few field-documented cases of excessive damage due to heavy metals have been reported. Several cases have been reported in which terrestrial plants have developed metal tolerances and have adapted to high metal concentrations. The evolution of metal resistant species or ecotypes has also been demonstrated in relation to man's industrial activities.[8] Much work remains to be done, however, on the effects of relatively high, frequent intakes of particular heavy metals, and also on the aggregate and interaction effects of two or more heavy metals when individually they are at sublethal levels.

Research indicates that sulfur dioxide fumigation resulting from smelter operations will inflict serious damage on local vegetation. Usually the fumigation must reach the interior of the leaf to inflict injury. The sulfur dioxide is partially converted to sulfurous and sulfuric acids. As a result of these chemicals, the cells lose their capacity to hold and translocate water; thus they dry out and die. Critically impaired water conduction is particularly evident in broadleaf plants; tissues between the veins are generally the first to

shrink and die. In addition, the chlorophyll of leaf cells is destroyed, possibly by the strong reducing properties of sulfur dioxide and sulfurous acid. Evidence of damage is early loss of green color in the affected areas, even while the leaves are still turgid. Defoliation occurs last.

The severity of injuries to plants exposed to a given dosage of sulfur dioxide varies with different species. Plants are most likely to be injured by sulfur dioxide when there is a favorable growing temperature, high relative humidity, bright weather, and protracted wind from the direction of the sulfur dioxide source. Although these factors in combination may have a slight to moderately additive effect, their primary influence is more analogous to links in a chain; a critical deficiency in any one of them may preclude injury despite the adequacy of the other factors for it. Apparently it is the rate, not the amount, of sulfur entry into the leaf that determines whether injury occurs.

In a study of smelter injury to forest trees in Washington and Montana, reproduction was scarce. This appeared to be a result of reduced cone crops and of the susceptibility and increased mortality of seedlings.[9]

Eighteen years after the initiation of sulfur dioxide abatement measures in the Columbia River Valley area, there was no evidence of current injury to timber or plants. Moreover, the appearance of individual trees did not suggest residual effects of earlier damage. Timber stands in zones of greatest damage were still thinner than elsewhere but there was no denuded area so large as to preclude fairly prompt restocking.

Conclusions and Recommendations

Some adverse environmental impacts may result from the mining, milling, and smelting of primary metals. Most evidence thus far is in the form of bioassays, with very little field documentation of finds. It is therefore obvious that full scale long range field documentation studies are required. These studies must look not only at the short-term ecological effects, but more importantly at the long-term effects. The following are basic questions to be studied: (1) Can biotic life in receiving streams and terrestrial habitats adapt to slightly higher concentrations of heavy metals? (2) How are the quality and quantity of biotic life altered as a result of mining, milling, and smelting operations? and (3) Can aquatic and terrestrial life recover their original stable states after mining operations cease?

REFERENCES

1. Stokes, P.M., T.C. Hutchinson, and K. Krauter. Heavy-metal Tolerance in Algae Isolated from Contaminated Lakes Near Sudbury, Ontario. Canadian Journal of Botany. Volume 51:2155-2168. 1973.

2. Butcher, R.W. Studies on the Ecology of Rivers. VI. The Aglol Growth of Highly Calcareous Streams. Journal of Ecology 33:268-283. 1946.

3. Dourdoroff, P. and M. Katz. Critical Review of Literature on the Toxicity of Industrial Wastes and Their Components to Fish. II. The Metals, as Salts. Sewage and Industrial Wastes. 25. 1953.

4. Schneider, Robert F. The Impact of Various Heavy Metals on the Aquatic Environment. U.S. Environmental Protection Water Quality Office. NTIS, Springfield Virginia.

5. Rudolfs, W., G.E. Barnes, G.P. Edwards, H. Heukelekian, E. Hurwitz, C.E. Renn, S. Steinberg, and W.F. Vaughan. Review of Literature on Toxic Materials Affecting Sewage Treatment Processes, Streams and BOD Determinations. Sewage and Industrial Wastes. 22. 1950.

6. Beavington, F. Contamination of Soil with Zinc, Copper, Lead, and Cadmium in the Wollongong City Area. Australian Journal of Soil Resources 11:27-31. 1973.

7. Beavington, F. Some Aspects of Contamination of Herbage with Copper, Zinc, and Iron. Environmental Pollution. 8:65-71. 1975.

8. Antonovics, J., A.D. Bradshaw, and R.G. Tuner. Heavy Metal Tolerance in Plants. Advanced Ecological Research 7:1-85. 1971.

9. Scheffer T.C., and G.G. Hedgecock. Injury to Northwestern Forest Trees by Sulfur Dioxide from Smelters. U.S. Department of Agriculture, Forest Service. Technical Bulletin No. 1117. June 1955.