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An Exposure and Risk Assessment for Lead

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16. Abstract (Limit: 200 words)

This report assesses the risk of exposure to lead. This study is part of a program to identify the sources of and evaluate exposure to 129 priority pollutants. The analysis is based on available information from government, industry, and technical publications assembled in July of 1981.

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

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AN EXPOSURE AND RISK ASSESSMENT FOR LEAD

by

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FOREWORD

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

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

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

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

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Richard Healy, Richard Silver, and Ruth Wilbur reviewed the document at U.S. EPA.

1.0 TECHNICAL SUMMARY

The Monitoring and Data Support Division, Office of Water Regulations and Standards of the U.S. Environmental Protection Agency is conducting risk assessments for pollutants that may enter and traverse the environment, thereby leading to exposure to humans and other biota. The program is in response to Paragraph 12 of the NRDC Consent Decree. This report is a risk assessment for lead using available data and quantitative models where possible to evaluate overall risk.

1.1 RISK CONSIDERATIONS

Children are particularly susceptible to the toxic effects of lead and lead exposure is still considered a major problem for children in the United States today. Lethalities are not as common as they once were; however, long-term effects such as learning and behavioral problems may occur, and remain after symptoms of clinical lead toxicity have disappeared. Ingestion of paint chips is considered the major source of lead exposure in children, especially in those from 1-3 years of age. Ingestion of contaminated dirt and dust is also a significant source of exposure in the vicinity of heavy traffic areas and industrial areas, especially smelters.

In adults, the effects of lead toxicity are on a much more limited scale. The primary exposure route is through food; a large part of this exposure results from processing and contamination of canned goods with lead solder. In isolated situations of high concentration of lead in air or drinking water, however, these pathways can dominate food exposure. Lead poisoning has also been observed in adults as a result of moonshine consumption, and the consumption of food from improperly glazed earthenware.

Risk to aquatic organisms as a result of exposure to lead does not appear to be widespread, because lead concentrations in the aquatic environment are generally low compared to levels at which effects are generally observed. Waterfowl, however, are still at risk because of exposure to lead shot. The use of lead shot, however, is being severely limited, and this problem is expected to diminish rapidly.

1.1.1 Humans

The effects of lead on humans have been extensively studied. However, exposure levels that were once considered acceptable have been lowered because of the increasing evidence of subtle effects at blood lead values that result in no overt symptoms of lead toxicosis.

Although human data on the carcinogenicity of lead are limited, no evidence suggests that lead is carcinogenic in humans. Feeding experiments with rodents have indicated some carcinogenic activity; however, this activity occurs at levels that are far in excess of the maximum tolerated dose of lead in humans (550 mg elemental lead per day).

Evidence for chromosomal abnormalities in humans resulting from lead exposure is inconclusive and contradictory. Studies in animals are also conflicting. However, lead can exert a profound, adverse effect on the fetus and can interfere with the reproductive ability of both men and women exposed to high levels (blood lead levels (PbB) of 30-40 μg/100 ml). Such levels are most common in occupational situations. Because the fetus develops so rapidly, it is particularly vulnerable to intrauterine exposure to lead. Such effects as increased incidences of stillbirths, preterm deliveries, and early fetal membrane rupture have been associated with elevated exposure to lead. Incidences of decreased fertility in occupationally-exposed men have also been reported. However, no data suggest that lead is teratogenic in man.

Other toxic effects of lead are directed primarily at three target organs; the erythroid cells of the bone marrow, the kidney, and the central and peripheral nervous system. The inhibitory effects of lead on erythropoiesis are reversible; however, severe acute or chronic lead poisoning may be followed by irreversible injury to the kidney and nervous system.

The disruption of hemoglobin synthesis is generally considered the first observable adverse effect of lead exposure and is the effect most commonly found. The inhibition of heme synthesis can eventually result in clinical anemia at blood levels greater than 80 μg/100 ml in adults, although mild anemia may occur at blood lead levels of 50 μg/100 ml. Children may be affected at levels of about 40 μg lead/ml of blood. A depression of ALAD (an enzyme necessary for the production of heme) activity has been observed at PbB of 10-20 μg/100 ml or lower.

Proximal tubular dysfunction can occur in both children (PbB=40-120 μg/100 ml) and adults (PbB >70 μg/100 ml) and is generally noted after short-term exposure. Although little is known about dose-response relationships, PbB greater than 70 μg/100 ml for prolonged periods may give cause to irreversible functional and morphological renal changes.

The effects of lead on the central nervous system are probably the most serious. Manifested as encephalopathy, effects on the central nervous system are seen more frequently in children than adults. Commonly observed symptoms include dullness, hyperkinetic or aggressive behavior, headaches, muscular tremors, hallucinations, and in severe cases, convulsions, mania, paralysis, and coma. The minimal PbB levels associated with encephalopathy are estimated to be about 80 μg/100 ml. Subtle impairment of cerebral function has been observed at levels over 40 μg/100 ml. The peripheral nervous system may also be affected by exposure to lead. Such effects as slowed nerve conduction have been reported at blood levels exceeding 50 μg/100 ml.

The pathways of human exposure to lead are numerous and sometimes complex. Food is generally considered the largest source of lead exposure in adults, although both drinking water and air can be more significant in some situations. Consumption of paint chips is considered

an important source of lead in children, and probably the most widespread; however, ingestion of dust and dirt may also be important in urban and smelter areas. Sources of lead in the diet may include contamination resulting from processing and lead-soldered cans, past use of lead arsenate pesticides, deposition on soil or plants from such sources as automobile and smelter emissions, contamination of moonshine whiskey, leaching from improperly glazed earthenware, and uptake from cooking water. The contamination of food through the use of lead solder in cans represents the most widespread source of lead in the diet. The "average" adult consumes 100-200 μg lead/day in food, while children consume about 100 μg/day. Up to 50% of this amount has been attributed to lead solder in cans. Persons living near sources of lead may consume more because of the contamination of local crops.

Drinking water does not generally contribute greatly to human exposure. However, concentrations greater than 50 μg/l can result from lead service lines, plumbing, solder, and storage containers.

Inhalation can be an important exposure pathway for adults in urban or industrial areas (i.e. smelters). In these areas, the primary sources of lead are automobiles and lead industries. The deposition of lead on dirt and dust provides an important exposure route for children via ingestion; however, this exposure route is not well quantified.

The ingestion of paint and contaminated soil is especially serious in children having pica (tendency to ingest nonfood items). It has been estimated that one-third to one-half of the 1-3-year-old children have pica. However, little is known about their behavior or the amount ingested.

An examination of the literature on human blood levels of lead revealed a typical range of 9-24 μg/100 ml in the blood of adults. Most of this (6-18 μg of lead per 100 ml of blood) is attributed to intake from food. Assuming a log-normal distribution of blood levels, less than 5% of the urban population would have blood levels greater than 30 μg/100 ml, and less than 0.5% of the rural population. Mean levels greater than 20 μg/100 ml have been reported in adults in the immediate vicinity of highways, and levels greater than 40 μg/100 ml in adults living near a smelter.

Blood levels of lead in children are consistently higher than adults in the same environment. Lead screening programs detect about 40,000 children annually with blood lead levels greater than 30 μg/100 ml. Again, elevated blood levels (greater than 30-40 μg/100 ml) have been found in a large proportion of children living near roadways and urban areas, in rural areas with lead paint problems, near primary and secondary smelters, and near battery plants.

1.1.2 Biota

Sublethal effects of lead in the laboratory (including developmental irregularities) have been observed at concentrations of less than

10 μg/l for rainbow trout in soft water. Several other freshwater fish species are similarly affected in the range of 10-100 μg/l. A variety of freshwater fish and invertebrate experience chronic toxicity in concentrations of 100-1000 μg/l in soft and moderately hard water, based primarily on effects on early life stages. Acute effects are observed in a few species at the upper end of this range. Most freshwater aquatic species experience acute effects in the range of 1-100 mg/l total lead.

Aquatic organisms may be exposed to lead through water, diet, and sediment. The relative importance of these routes is highly variable, and depends on the species and the environment. Exposure is enhanced in the presence of high concentrations of lead, especially in soft, acidic waters. Mean total lead concentrations were generally below 50 μg/l in 1979. However, concentrations of approximately 25,000 μg/l, which sometimes results in fish kills, have been reported near such sources as an old smelter and a tailings pond. In addition, a concentration of less than 1000 μg/l resulted in a fish kill in 1970.

High concentrations of total lead (mean >50 μg/l, with at least 20% of the observation levels >100 μg/l) have been found in the Catawba-Wateref Basin, the James River, the Fox River-Wolf Creek drainage basin, and the Kootenai River. Identifying areas with high aquatic exposures is difficult because of analytical problems and the inherent problems with aggregations of monitoring data. The identification of localized lead exposure areas requires a more detailed investigation than was conducted here. In addition, the local conditions including pH, hardness, etc. greatly influence toxicity. These factors have only been considered on a broad scale in this analysis.

The data on lead toxicity in waterfowl, ducks, and gallinaceous birds clearly indicate that ingestion of spent lead shot can be lethal; numerous incidences of waterfowl mortality as a result of ingesting spent lead shot have been reported. Waterfowl kills are more prevalent in areas under heavy hunting pressure (such as the Mississippi Flyway) and in lakes and marshes with hard bottoms, where lead shot can accumulate.

1.2 SOURCES OF LEAD TO THE ENVIRONMENT

In 1976, the supply of lead was approximately 1,515,000 kkg. About 34% of this supply resulted from domestic ore production, while about 35% resulted from secondary production. The remaining supply consisted of imported ores and metals, and industry stocks.

Approximately 49% of the industrial supply was consumed in the manufacture of lead-acid storage batteries and the production of battery oxides. The production of antiknock gasoline additives consumed about 14% of the supply. The remaining 37% of the industrial supply was used for such purposes as the production of red lead and litharge, ammunition, solder, weights and ballast, pigments, cable covering, brass and bronze, sheet lead, bearing metals, caulking metals, pipes, and type metal.

Of the identified releases to the environment (~355,000-360,000 kkg), 53 % are airborne and 46% are in the form of solid waste. Known releases to surface waters and POTWs account for less than 1% of the total.

Of the estimated 190,000 kkg lead released to the atmosphere, the releases from the use of leaded gasoline comprise about 93%. Releases from fossil fuel combustion, lead smelters, iron and steel production, battery manufacture, and copper and zinc smelting, each contribute about 1-2% of the total known airborne releases. However, some of these sources may be important in local areas.

It was difficult to quantify releases of lead to water because of large gaps in the data. However, aquatic discharges from the major uses of lead are assumed to be small. Of the known releases to the aquatic environment (800-950 kkg), 53%-63% are attributed to the iron and steel industry, 21% to lead production, 6% each to battery production and the nonferrous metal industry, and 4% each to the inorganic chemical and pulp and paper industries. Urban runoff probably represents the major source of lead to the aquatic environment, and perhaps as much as 21,000 kkg to surface waters and POTWs. However, these releases are largely a result of the deposition of airborne releases.

Solid wastes represent a large source of lead (~165,000-168,000 kkg) to the environment. Solid wastes are primarily produced as a result of domestic ore production (34% of the known total), and ammunition use (30% of the total). Other important sources include solder, weights and ballast, bearing metals, and iron and steel production.

Numerous uncertainties exist in the materials balance for lead. The releases from the production, use, and disposal of lead-acid storage batteries are largely unknown, although lead-acid storage batteries represent the single largest use of lead. In addition, releases from other uses are unknown, and releases from relatively minor uses may be of concern, although they are believed to be small compared with airborne releases and solid waste.

1.3 FATE AND DISTRIBUTION OF LEAD IN THE ENVIRONMENT

Since the atmosphere receives the greatest portion of releases, it is important to consider the fate of lead in this media. Transport of lead in the atmosphere depends on particle size, chemical form, and the distribution and height of the release. Particles larger than 20 μm are rapidly deposited. Although automobile exhaust, the largest single source, contains lead in extremely small aerosol sizes, with a mass median diameter of 0.5 μm or less, large particles may be released during cold start or acceleration. These larger particles are deposited onto the roadway, or within a few meters. Smaller particles also are deposited, to some extent, near the roadway by impaction; however, some fraction is carried a distance (~100 m) from heavily travelled roads

and may be deposited by washout. Still, precipitation may carry some of the particles a considerable distance; thus, more remote areas may be contaminated.

Lead releases from smelter, fossil fuel combustion, and iron and steel production are primarily from elevated point sources and are generally less than 1 μm in size. Consequently, these particulates are widely dispersed and are primarily deposited by precipitation. Smelter emissions result in significant deposition of lead at distances up to two miles from the smelter. Fugitive dusts account for higher deposition near the source. Airborne releases from mining and milling operations will largely be in the form of fugitive dusts, resulting in localized deposition.

The deposited lead forms from all atmospheric sources are largely insoluble. Lead accumulates in plants, leaf litter, and soil. Runoff and erosion introduce lead into surface waters as a suspended solid, which results in sediment concentration.

The monitoring data for lead generally reflect these fate pathways. The range of lead levels in air for remote areas of the continental United States is 0.0001-0.01 $\mu g/m^3$. In contrast, urban areas show levels of 0.5-10 $\mu g/m^3$, which are considerably higher, primarily because of automobile use. The monitoring data show rapid deposition; and it has been estimated that lead levels in air generally decrease by about 50% between 10 and 20 meters from the highway.

Elevated atmospheric lead levels are also found in the vicinity of point sources, such as smelters, in the range of 0.4-4 $\mu g/m^3$. Considerably higher levels have occasionally been reported, however.

Direct sources of lead to water are largely unidentified, although probably relatively small. Lead reaching surface waters is likely to be strongly sorbed onto suspended solids and sediments. Because lead in the sediment is strongly sorbed, it is unlikely to be desorbed as a result of a physical disturbance. Changes in the water chemistry, for example, pH, could result in an increased solubilization of lead. Lead found in the water column is expected to be strongly complexed by organic molecules. Lead can bioconcentrate in aquatic organisms up to 2-4 orders of magnitude above water concentrations. It appears to be fairly persistent in aquatic biota, with a lifetime of at least several months. Little evidence suggests biomagnification of lead in aquatic food chains.

Typical levels of lead in U.S. waters are less than 25 μg/l. Levels of lead in seawater are considerably lower, on the order of 0.005 μg/l. Lead concentrations in surface waters are higher in urban areas than in rural areas.

Sediments contain considerably higher levels of lead than surface waters. Coastal lead sediment contains approximately 100 mg/kg, while the average lead in river sediments was estimated to be about 20 mg/kg. Higher concentrations were found in STORET data, with mean concentrations ranging from 27-267 mg/kg, during 1973-79.

Lead transported to soil is quite strongly sorbed, and, under most conditions, is not subject to leaching. The movement of lead with the erosion of soil particles is likely, however. Entrainment of soil particles is also a possible route of lead transport. In addition, uptake of lead into plants can occur, although only a small portion of the total lead in any soil is available for plant uptake. Bioconcentration factors are generally less than 1, although they may be higher in the roots.

Monitoring data for lead in soils largely reflect airborne deposition patterns. An average concentration for lead in U.S. soils appears to be about 20 mg/kg in uncontaminated soils. Elevated concentrations are found in the vicinity of highways (up to 7600 mg/kg), and, in general, in urban areas where a range of 100-800 mg/kg lead in soil is found. Elevated concentrations are also found in the vicinity of smelters (up to about 8000 mg/kg), in the vicinity of houses that at one time were painted with lead-containing paints, and in old orchard soils. Urban dust is also found to contain high levels of lead. Concentrations of about 1000-1600 mg/kg are found in urban residential areas and 1400-2400 mg/kg in commercial areas.

2.0 INTRODUCTION

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

This document is a brief assessment of the effects, exposure, and potential risks that result from the production, use, release, and distribution of lead. Numerous authors have noted a number of problems associated with an analysis of lead. First, the literature on lead is voluminous and could not be comprehensively reviewed within the time constraints of this report. Therefore, previous reviews have been extensively used and have been supplemented with information from the recent literature. Although this may not be the most satisfactory approach, in general, it was deemed the most appropriate and effective for the purposes of this report.

Second, the problems with analyses, and sample and laboratory contamination have been widely acclaimed. Statements, in this report, have been qualified on the basis of recent analytical data. However, older data, and even recent data, can reflect this problem and could not be actively addressed in this work.

Third, and perhaps most significant, regulatory mechanisms are currently being effected for lead from many sources. Thus, exposure estimates and conclusions can be expected to change over the next ten years. This report attempts to point out which exposure routes will be affected, and which exposure routes will continue to be a problem.

This report is organized as follows:

- Chapter 3.0 contains information on the production, discharge, and disposal of lead.
- Chapter 4.0 describes available monitoring data and a consideration of the fate of lead in five specific pathways.
- Chapter 5.0 discusses the effects of lead on humans and describes exposure scenarios.
- Chapter 6.0 considers the effects and exposure of biota to lead.
- Chapter 7.0 discusses risk considerations for various subpopulations of humans and biota.

3.0 MATERIALS BALANCE

3.1 INTRODUCTION

Lead emissions to the environment may result from primary and secondary production of lead and from the use of refined lead in manufactured products. In addition, the concentrations of lead that occur naturally in the environment may cause inadvertent emissions as a result of the processing of these materials. This section of the report has been assimilated from four basic sources: government publications, trade journals, standard references and text, and data published by the American Bureau of Metal Statistics. This materials balance examines releases of lead to the environment of the United States for 1976.

Although more recent data are available for some of the use categories and for most of the production categories, the most accessible and reliable literature is for 1974-76. The most definitive source of information on lead emissions is Nriagu (1978), whose information and assumptions are based on data for 1974 and 1975. Also, in considering the ranges of uncertainty in the information available on lead uses and emissions -- typically an order of magnitude or more -- attempts to scale data for 1974 and 1976 to a later year, such as 1978, would require adjustment factors that are small in comparison to the data uncertainties; therefore, such adjustments seem unwarranted. In effect, the data presented are approximately as reliable for any year between 1974 and 1978 as for the year 1976.

Lead is recovered from lead sulfide ores. In 1976, domestic mine production was 518,000 kkg. The total primary U.S. smelter/refinery production in that year was 597,000 kkg (domestic plus imported ores), while secondary production (scrap processing) contributed 526,000 kkg to the U.S. supply. Imports provided 79,000 kkg of ore and 129,000 kkg of lead metal in 1976. Industry stocks (January 1) contributed 263,000 kkg, which brought the total U.S. 1976 supply to 1,515,000 kkg. At the end of 1976, 5400 kkg of the supply was exported and 140,000 kkg remained in industry stocks, leaving 1,370,000 kkg for industrial consumption. Table 3-1 presents a summary of the lead supply, demand, and emissions. Notes explaining the data in Table 3-1 are in Appendix A. Where firm data were unavailable, estimates were based on engineering assumptions about manufacturing and use processes.

Approximately 49% of the industrial supply was consumed in the manufacture of lead-acid storage batteries and the production of battery oxides. About 14% of the supply was consumed for the production of antiknock gasoline additives. The remaining 37% of the industrial supply was used in twenty-two other categories. Figure 3-1 is a graphic representation of the flow of lead from commerce and inadvertent sources to the media.

TABLE 3-1. SUMMARY OF U.S. LEAD SUPPLY, USE, AND EMISSIONS, 1976[a]

	(kkg)					
	Supply	Consumption	Airborne Emissions	Aquatic Discharges	Discharges to POTWs	Solid Wastes
Production and Emissions						
Domestic ore mining and milling, smelting, and refining	518,000 (1)		580 (3)	NA	0	
Imported ores (smelting and refining)	79,000 (1)		820 (3)	180 (34)	0	56,000 (37)
Imported metals	129,000 (1)		130 (4)	-	0	380 (4)
Industry stocks, January 1	263,000 (1)		-	-	-	-
Secondary production	526,000 (1)		-	-	-	-
			180 (5)	NA	NA	NA
Uses and Emissions						
Lead-acid storage batteries (e.g., grids and posts)		348,000 (2)	1,700 (6)	3-110 (35)	10-430 (35)	~0 (35)
Storage battery oxides		398,000 (2)	NA (6)	NA (35)	NA (35)	NA (35)
Gasoline antiknock additives		217,000 (2)	176,000 (7)	NA (36)	NA (36)	NA (36)
Red lead and litharge		77,000 (2)	NA	NA	NA	NA
Ammunition		66,000 (2)	670 (8)	NA (8)	~0 (8)	50,000 (8)
Solder		57,000 (2)	1,150 (9)	NA (9)	NA (9)	5,730 (9)
Weights and ballast		20,000 (2)	100 (10)	NA (10)	NA (10)	10,000 (10)
Pigments		15,000 (2)	210 (11)	15 (11)	50 (11)	360 (11)
Cable covering		14,000 (2)	10 (12)	NA (12)	~0 (12)	3,610 (12)
Brass and bronze		14,000 (2)	710 (13)	NA (13)	~0 (13)	2,840 (13)
Sheet lead		22,000 (2)	20 (14)	~0 (14)	~0 (14)	2,220 (14)
Bearing metals		12,000 (2)	60 (15)	NA (15)	~0 (15)	9,480 (15)
Calking metals		11,000 (2)	10 (16)	NA (16)	~0 (16)	2,720 (16)
Pipes (traps and bends)		13,000 (2)	10 (17)	NA (17)	NA (17)	3,130 (17)
Collapsible tubes		2,110 (2)	2 (18)	0 (18)	0 (18)	2,110 (18)
Type metal		14,000 (2)	70 (19)	~0 (19)	NA (19)	140 (19)
Terne metal		1,400 (2)	10 (20)	~0 (20)	~0 (20)	70 (20)
Annealing		2,600 (2)	260 (21)	~0 (21)	~0 (21)	520 (21)
Miscellaneous chemicals		130 (2)	NA	NA	NA	NA
White lead		2,700 (2)	10 (22)	NA (22)	NA (22)	310 (22)
Lead plating		350 (2)	~0 (23)	NA (23)	NA (23)	NA (23)
Casting metal		6,080 (2)	60 (24)	NA (24)	NA (24)	1,520 (24)
Galvanizing		1,140 (2)	110 (25)	~0 (25)	~0 (25)	350 (25)
Foil		4,650 (2)	10 (26)	0 (26)	0 (26)	2,320 (26)
Other		48,000 (2)	NA	NA	NA	NA
Exports, metal		5,440 (2)	-	-	-	-
Industry stocks, December 31		140,000 (2)	-	-	-	-
Inadvertent Emissions						
Combustion of oil			2,630 (27)	0	0	0
Combustion of coal			480 (28)	NA	0	1,430 (28)
Iron and steel production			1,240 (29)	500 (29)	500 (29)	3,730 (29)
Copper and zinc smelting			1,800 (29)	NA	NA	5,400 (29)
Timber products			0	~0	~0 (29)	NA
Leather tanning			0	~0 (29)	~0 (29)	NA
Petroleum refining			NA (30)	~0 (29)	~0 (29)	~0
Paint and ink manufacturing			0	0	~0 (29)	NA
Coal mining			NA (31)	NA (29)	NA	NA (31)
Inorganic chemical manufacturing			NA	10-50 (29)	4 (29)	NA
Textiles			0	10 (29)	10 (29)	NA
Pulp and paper			~0	40 (29)	10 (29)	NA
Rubber products			~0	NA (29)	0 (29)	NA
Laundries			0	0 (29)	~0 (29)	0
Miscellaneous chemicals (pesticides) manufacturing			NA	NA (29)	NA (29)	NA
Nonferrous metals			NA	50 (29)	10 (29)	NA
Fertilizer (phosphatic)			~0	NA (32)	~0 (32)	95 (32)
POTW discharges			60-960 (33)	10 (33)	-	250-3,680 (33)
TOTAL	1,515,000	1,513,000	189,000-190,000	800-950	590-1,010	165,000-168,000

Note: NA = Not available
Numbers may not add due to rounding.

[a]Circled numbers refer to notes in Appendix A.

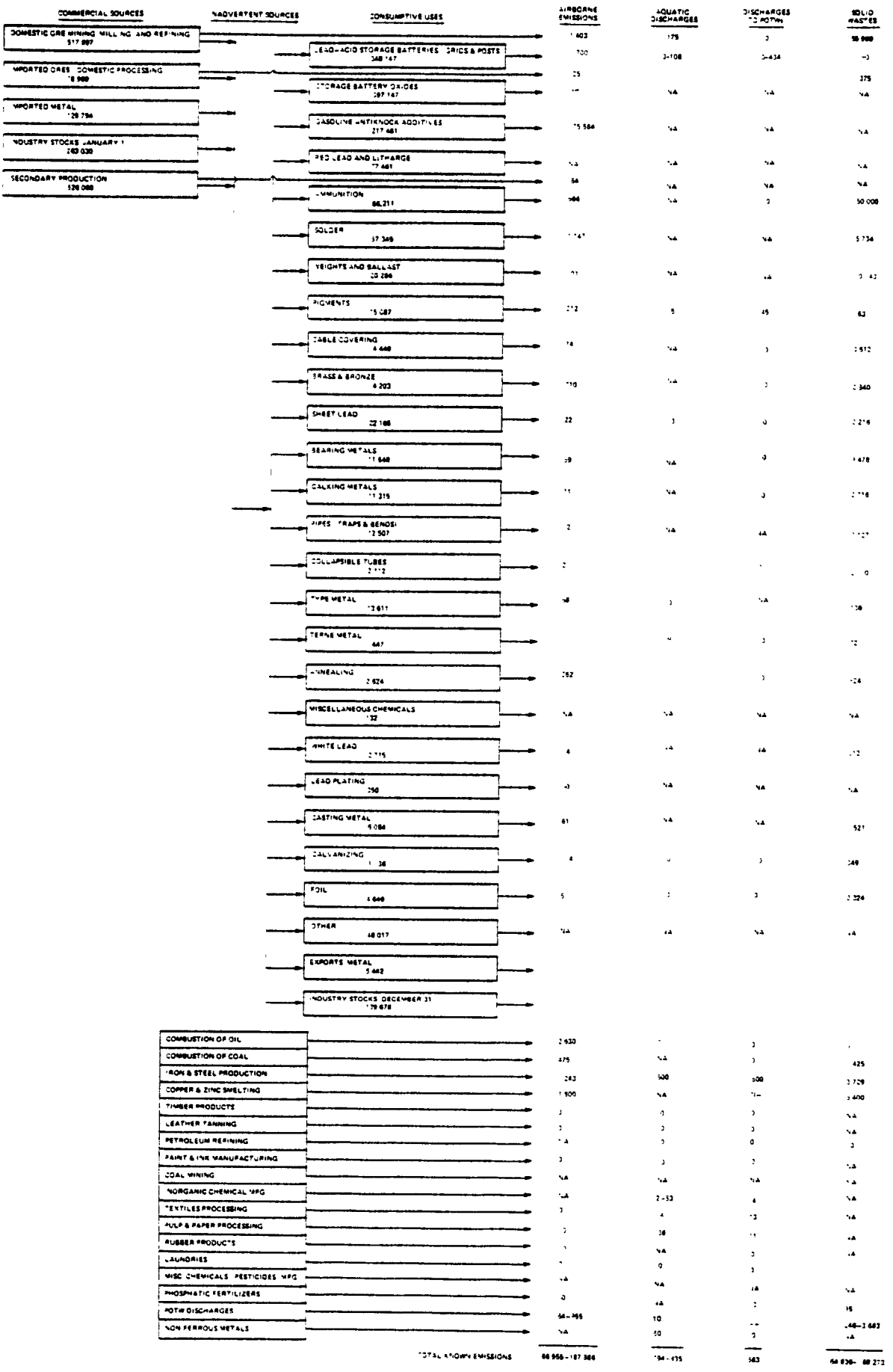

FIGURE 3–1 ENVIRONMENTAL FLOW OF LEAD, 1976 (kkg)

Of all estimated emissions of lead to the environment, 53% are airborne and 47% go to land. Identified emissions to water and POTWs account for <1% of all emissions. Emissions from the use of leaded gasoline comprise over 93% of all airborne emissions of approximately 190,000 kkg. Although few sources of aquatic discharges were quantified, aquatic discharges from iron and steel production are the primary source of waterborne wastes, which amount to approximately 53-63% of aquatic discharges. Solid wastes, which are primarily generated during mining and beneficiation processes and as a result of ammunition use, contain between 165,000 kkg and 168,000 kkg of lead.

3.2 PRODUCTION OF LEAD

Lead is recovered from naturally-occurring sulfide ores, which typically contain between 1 and 6% lead. Zinc and, to a lesser extent, other metal sulfides are often associated with lead sulfide. The domestic lead production is comprised of primary and secondary (scrap) sources. Six domestic smelters and five refineries processed 518,000 kkg of domestically mined ores and 79,000 kkg of imported ores in 1976 (Nriagu 1978). Capacities and locations of domestic lead smelters are presented in Table 3-2. Approximately 85 companies operating 115 plants produce lead and lead alloys for industrial use from recycled materials, principally old batteries. Two companies, NL Industries and RSR Corporation, operate about 18 secondary smelters; these smelters account for over one-half of the total secondary lead production (U.S. Bureau of Mines 1977a). Thirteen companies operating approximately 24 plants account for most of the remaining secondary lead production (U.S. Bureau of Mines 1977a).

3.2.1 Primary Production

In 1963, vast deposits of lead ore were discovered in Missouri; and, in 1976, Missouri contributed 82% of the total domestic production. Idaho contributed 9%, Colorado 4%, and Utah 3%. The 25 leading mines listed in Table 3-3 accounted for over 99% of the total U.S. production (U.S. Bureau of Mines 1977b).

Prior to smelting, standard crushing, grinding, and flotation methods are used to beneficiate lead ore at the mine site. To obtain the end product, three operations take place at the smelter (U.S. Bureau of Mines 1977a).

- The ore concentrate is sintered with additional charge material of recycled sinter, sand, and other inerts; this process oxidizes the lead and sulfur when they change to oxides and agglomerates the charge to form a dense permeable mass suitable for furnace feed.

- The lead oxide in the sinter is reduced to produce molten lead.

- The resultant lead bullion is refined to eliminate impurities.

TABLE 3-2. CAPACITIES AND LOCATIONS OF U.S. LEAD SMELTERS AND REFINERIES, 1977

Company	Location	Capacity (kkg)
Bunker Hill	Bradley, ID[a]	117,910
St. Joe Minerals	Herculaneum, MO[a]	204,075
AMAX	Boss, MO[a]	126,980
ASARCO	Glover, MO[a]	99,770
	El Paso, TX[b]	81,630
	East Helena, MT[b]	81,630
	Omaha, NE[b]	163,260

[a]Denotes primary smelter and refinery.

[b]ASARCO plants at El Paso, Texas and East Helena, Montana are smelters only. They supply feedstock to ASARCO's refinery at Omaha, Nebraska.

Source: U.S. Bureau of Mines (1977a).

TABLE 3-3. U.S. LEADING LEAD-PRODUCING MINES IN ORDER OF OUTPUT, 1975

Mine	County and State	Operator
Buick	Iron, MO	AMAX Lead Co.
Fletcher	Reynolds, MO	St. Joe Minerals Corp.
Magmont	Iron, MO	Cominco American, Inc.
Ozark	Reynolds, MO	Ozark Lead Co.
Brushy Creek	Reynolds, MO	St. Joe Minerals Corp.
Viburnum #29	Washington, MO	St. Joe Minerals Corp.
Viburnum #28	Iron, MO	St. Joe Minerals Corp.
Bunker Hill	Shoshone, ID	The Bunker Hill Co.
Lucky Friday	Shoshone, ID	Hecla Mining Co.
Indian Creek	Washington, MO	St. Joe Minerals Co.
Star Unit	Shoshone, ID	Hecla Mining Co.
Viburnum #27	Crawford, MO	St. Joe Minerals Co.
Leadville Unit	Lake, CO	ASARCO, Inc.
Idarado	Ouray & San Miguel, CO	Idarado Mining Co.
Bingin	Utah, UT	Kennecott Copper Co.
Ontario	Summit, UT	Park City Ventures
Sunnyside	San Juan, CO	Standard Metals Corp.
Balmat	St. Lawrence, NY	St. Joe Minerals Corp.
Pan American	Lincoln, NV	St. Patrick Mining Co.
Austinville and Ivanhoe	Wythe, VA	New Jersey Zinc Co.
Camp Bird	Ouray, CO	Federal Resources Co.
Emperius	Mineral, CO	Minerals Engineering Co.
Ground Hog	Grant, NM	ASARCO, Inc.
Pend Oreille	Pend Oreille, WA	The Bunker Hill Co.
Eagle	Eagle, CO	New Jersey Zinc Co.

Source: U.S. Bureau of Mines (1977b).

Sintering is carried out in updraft sintering machines of a type commonly used in the metals production industry. The lead oxide is then reduced in a blast furnace that separates the charge into four layers: slag, speiss (essentially arsenic and antimony), matte (mostly copper and other metal sulfides), and product lead metal. The lead contains varying amounts of other metals that are removed by refining.

Usually, copper drossing is the first step in refining. The bullion is maintained above the melting point of lead but below the melting point of copper. The copper begins to freeze and floats to the surface. The scum floating on the surface is referred to as dross and is skimmed and processed for metal recovery.

Other impurities, such as arsenic, antimony, tin, gold, and silver, are removed by similar operations of melt-freeze cycles. The refining process is complex and variable depending on the impurities present in the original concentrate.

Emissions from Primary Production

Environmental emissions of lead from primary production are in the form of particulate airborne emissions from mining, milling, and materials handling operations; airborne emissions are also associated with blast furnaces and drossing operations. However, in smelting and refining operations, precautions are generally taken to control emissions. Particulates in off-gas streams are cooled and treated by baghouses. Some plants also use the off-gases to produce sulfuric acids. These mechanisms effectively reduce a large portion of the airborne emissions of lead. The total known airborne emissions from mining, milling, smelting, and refining processes are estimated at 1500 kkg for 1976, for both domestic ores and foreign ores that are smelted and refined domestically.

Aquatic discharges may result from the flotation beneficiation process. Aquatic discharges are probably small when compared with other emissions from primary production because lead metal and most lead compounds have extremely low solubility in water. Based on the releases of one plant, it has been estimated that losses of approximately 180 kkg have resulted from smelting and refining operations.

Solid wastes from primary production occur as unrecovered metal in mine tailings; however, from emission control systems, they occur as sludges. The total estimated amount of solid wastes generated by primary production from both foreign and domestic ores is 56,000 kkg.

Detailed explanations of the emissions associated with primary production are given in Appendix A.

3.2.2 Secondary Production

The recovery of lead from scrap metal is a significant contributor to the annual lead supply. In 1976, 526,000 kkg of lead (approximately

35% of the total supply) was recovered from scrap. Nearly 70% of secondary production of lead is derived from storage batteries (U.S. Bureau of Mines 1977a).

The remaining 30% is composed of solder, type metals, drosses, and residues. The processing procedure depends on the composition of the scrap. In general, secondary lead production has three major phases.

Pretreatment involves crushing and breaking the scrap metal into suitable sizes and smelting and refining of the crushed scrap.

Crushing of the scrap is normally accomplished by jaw crushers. Smelting and refining operations are similar to the primary lead production, except some plants employ reverberatory furnaces rather than blast furnaces. In addition, secondary lead processors do not employ a sintering step because (1) lead is present in oxide form in the scrap, and (2) the scrap material contains essentially no sulfide.

Emissions from Secondary Production

Environmental emissions of lead from secondary production operations are largely unquantifiable. Smelting and refining of secondary lead is estimated to generate 180 kkg of airborne lead. Data on aquatic discharges and solid waste generation are unavailable.

3.3 USES OF LEAD

Refined lead has a minimum purity of 99.85% and is marketed in four grades: (1) corroding lead, a designation used to describe lead of high purity; (2) chemical lead, lead of high purity but not desilverized; (3) acid-copper lead, made by adding copper to fully-refined lead; and (4) common desilverized lead, a designation used for fully-refined desilverized lead.

Over 49% of the total U.S. demand, 746,000 kkg, is used for the production of lead storage batteries. Approximately 47% of this total (348,000 kkg) is used for the production of the battery posts and grids. The remaining 398,000 kkg is used in the manufacture of lead oxides that is used in batteries (Lead Industries Association, Inc. 1978). The life span of storage batteries is typically 2-4 years; almost all the spent batteries are reprocessed by secondary smelters to recover the lead values. Recycled batteries are the major source of feedstock for the secondary smelters.

The next largest use of lead is in the manufacture of gasoline antiknock additives, tetraethyl-lead and, to a lesser extent, tetramethyl-lead. The production of these additives consumed 217,000 kkg of lead (approximately 14% of the total demand) in 1976. These additives are used to increase the octane rating of gasolines. Except for a slight increase in 1976, the consumption of lead for additive production has decreased since 1972. Consumption of lead for automotive applications will probably

continue to decrease because later model automobiles have catalytic converters that preclude the use of leaded gasoline.

The remaining 37% of the lead consumed has many uses. The American Bureau of Metal Statistics and the Lead Industries Association, Inc. (American Bureau of Metal Statistics 1978) compiles annually a list of twenty-two applications for the remaining 37% of the lead supply. None of these applications consumes more than 5.5% of this supply. Use patterns for 1965-76 are shown in Table 3-4.

One of the larger uses of lead is the production of red and white lead used in paint pigments. In the past, lead-base paints were used extensively; however, because of their toxicity, use has been restricted to protective exterior coatings for bridges and ships or in applications where lead-base paints are sealed with a nonlead paint coating.

Most of the remaining uses of lead are congruent with the properties of the metal. Lead is dense, malleable, has a relatively low melting point, and is almost insoluble in water. In addition, lead is highly resistant to corrosive chemicals and high frequency electromagnetic radiation and it has good lubricative properties. Consequently, it is used for weights and ballast in sailboats and for fishing tackle and also for ammunition and ordnance.

Lead is a soft metal; thus it is amenable to uses such as soldering and calking metals. Collapsible tubes for material packaging, such as artists' paints, are made of lead, particularly if the material is corrosive. For use in structural applications, lead must be alloyed with other metals to increase its strength and hardness. Terne metals (ternary lead alloys) are used in structural applications. Other alloys include type metal, used in the printing industry, casting metal and specialized brass and bronze alloys.

Because lead is relatively insoluble in water, it has been used for plumbing pipes; recently, however, this use has become specialized and more limited to applications where lead's corrosive resistant property is important. Lead or lead alloys are used for piping and process vessels in the chemical process and other industries where the handling of corrosive fluids is necessary. Lead or its alloys are also used for cable sheathing, galvanizing, and electroplating.

Sheet lead is used for radiation shielding in hospitals and nuclear plants. Lead foil is often used for the transport of radioactive material. Because lead has good lubricative properties, it is used for bearings and in specialized alloys. Lead chemicals are used in pesticides and for glazing operations in the glass and ceramic industry. Environmental emissions from these sources (see Appendix A) result from the volatilization, leaching, or disposal of lead or lead bearing materials (U.S. Bureau of Mines 1977a).

TABLE 3-4. U.S. SUPPLY AND DEMAND RELATIONSHIPS OF LEAD, 1965-76

Components of U.S. Supply	Lead (kkg) 1965	1966	1967	1968	1969	1970	1971	1972	1973	1974	1975	1976
Domestic Ores	279,356	294,775	239,448	331,055	476,175	487,059	530,595	528,781	522,432	531,502	482,524	517,85[illegible]
Foreign Ores	106,119	115,189	113,375	110,654	117,910	127,887	73,467	95,235	100,677	87,979	96,142	78,909
Secondary (Scrap)	449,872	439,895	432,639	427,197	468,012	458,942	444,430	451,686	488,873	545,107	511,548	526,060
Sales of Govt Stockpile	48,071	58,048	25,396	26,303	19,954	10,884	9,070	40,815	191,377	241,262	6,348	0
Imports, Metal	202,261	258,496	330,148	306,566	253,053	222,215	175,051	223,122	161,446	107,026	89,793	128,794
Industry Stocks Jan. 1	179,586	175,050	186,842	209,517	153,283	206,796	295,682	224,029	239,448	194,098	261,217	263,030
TOTAL	1,265,265	1,341,453	1,327,848	1,411,292	1,488,387	1,513,783	1,528,295	1,563,668	1,704,253	1,706,974	1,447,572	1,514,690
Distribution of Supply												
Industry Stocks Dec. 31	175,051	186,842	209,517	153,283	206,796	295,682	224,029	239,448	194,098	261,216	263,030	139,678
Exports, Metal	7,256	4,535	6,349	7,256	4,535	7,256	5,442	7,256	60,769	56,234	19,047	5,442
TOTAL INDUSTRY DEMAND	1,082,958	1,150,076	1,111,982	1,250,753	1,277,056	1,210,845	1,298,824	1,316,964	1,449,386	1,389,524	1,165,495	1,369,570
End Use Demand Pattern												
Gasoline Additives (SIC 2911)	204,075	224,029	224,029	237,634	245,797	253,053	239,448	253,053	248,518	226,750	189,563	217,461
Transportation (SIC 3691, 37)	354,637	366,428	357,358	410,871	446,244	497,036	566,875	605,876	634,900	711,995	624,016	710,181
Construction (SIC 344)	124,259	119,724	108,840	106,119	107,933	109,747	90,700	79,816	78,909	70,746	62,583	43,350
Paints (SIC 2851)	98,863	108,840	93,421	99,770	92,514	89,793	73,467	80,723	98,863	105,212	71,653	96,142
Ammunition (SIC 348)	51,699	70,746	71,653	74,374	71,653	66,211	79,816	77,095	73,467	78,909	68,025	66,211
Electrical (SIC 3356, 3357, 3691)	85,258	92,514	88,886	78,909	87,072	78,909	118,817	116,096	122,445	120,631	68,932	116,096
Other	164,167	167,795	167,795	243,076	225,843	116,096	129,701	104,305	192,284	75,281	80,723	118,129
TOTAL	1,082,958	1,150,076	1,111,982	1,250,753	1,277,056	1,210,845	1,298,824	1,316,964	1,449,386	1,389,524	1,165,495	1,369,570

Sources: U.S. Bureau of Mines (1977a); American Bureau of Metal Statistics (1978).

3.3.1 Emissions from the Use of Lead

Although batteries account for a large portion of lead use, they are enclosed packages of lead; thus, none of this lead escapes into the environment during use (Hepple 1971). However, limited data substantiate that airborne and aquatic emissions have been estimated to be 1700 and 3-110 kkg, respectively, during the manufacture of batteries (see Appendix A, notes 6 and 35). Solid waste discharges are thought to be insignificant because of recycling.

The single largest anthropogenic environmental discharge of lead results from the use of organic lead compounds as antiknock additives in gasoline. Approximately 176,000 kkg of airborne lead emissions occur annually from the combustion of gasoline. This source comprises over 93% of the airborne lead emissions from all sources.

Aquatic discharges of lead from use activities are not well defined. Lead discharges to the aquatic media are not believed to be significant because lead and most lead compounds have extremely low solubility in water. The only known aquatic discharges from use activities are derived from pigment manufacture and are estimated to be approximately 15 kkg. In addition, an estimated 45 kkg of lead are discharged to POTWs from pigment production.

Solid waste results from the disposal of lead or lead bearing materials or from nonreusable products, such as ammunition and ordnance. Ammunition and ordnance are the largest contributors of solid waste from lead use activities. They contributed about 50,000 kkg in 1976, which is about 51% of all identified solid waste emissions from use activities. Other important sources include lost and discarded weights and ballasts (~10,000 kkg), discarded bearings (~9500 kkg), and spent soldering metals (~5700 kkg). Other sources of solid waste from the lead use categories and from lead bearing materials are summarized in Table 3-1 and Figure 3-1. Gathering of data and estimates is detailed in Appendix A.

3.3.2 Inadvertent Sources of Lead Emissions

At low level concentrations, lead is ubiquitous in the biosphere. It occurs naturally in virtually every mineral and many organisms. The processing of these materials may result in lead emissions because of its presence in these materials. This is particularly true if large quantities of the material are processed. For example, the combustion of fossil fuels and smelting of other important metals result in anthropogenic environmental discharges of lead. Lead and lead chemicals are present in some products that are used by industry in their material processing. Most of the important sources of lead, where lead is inadvertently discharged, are presented in Table 3-1 and Figure 3-1.

Urban runoff is likely to be an important source of lead to aquatic environments. In stormwater runoff from one city, lead concentrations ranged from 0.1-2.85 mg/l, with a mean concentration of 0.46 mg/l

(U.S. EPA 1974). Similarly, Bennett and coworkers (1981) reported levels of approximately 1 mg/l in stormwater and snowmelt runoff from Boulder, Colorado.

Considerable other data on levels of lead in urban runoff will not be reviewed here because it is extremely difficult to choose a representative concentration. For example, assuming a concentration of 1 mg/l and a runoff volume of 21×10^{12} l/yr (U.S. EPA 1977), a loading of 21,000 kkg may be estimated. About 82% of this, or 17,000 kkg, would go directly to surface waters, while about 4000 kkg would go to POTWs. Even though this is a rough estimate, it does represent the largest source to aquatic environments. This value, however, is not shown in Table 3-1 or Figure 3-1, because it is believed to be largely attributable to airborne releases.

3.4 FUTURE PROJECTIONS FOR LEAD

The U.S. Bureau of Mines (1977a) has projected an annual growth rate of 1.8% for lead production and consumption through the year 2000. The growth areas of lead by end use pattern are presented in Table 3-5.

The largest area of growth is expected to be in the transportation industry (U.S. Bureau of Mines 1977a). Except for gasoline additives, all other areas are expected to show modest growth. Leaded gasoline additives are currently being phased out because of the large amount of airborne emissions from motor-powered vehicles. Factors that could retard domestic growth in lead production include shortage of skilled labor, energy shortages, environmental restrictions, and the shortage of investment funds at moderate cost in a capital intensive industry. The latter two may have significant impact. Modifying existing smelters and refineries in order to comply with the new regulations established by the U.S. EPA and OSHA could cost the lead industry as much as $650 million by 1982 (Krammer 1978). It is possible that the stricter standards may force some producers to shut down their operations, which will necessitate the importation of a greater volume of lead.

Domestic reserves of high grade lead ore are primarily located in Missouri (72.5% of domestic reserves). The known worldwide reserves total 124 million kkg and are adequate to meet the world demand through the year 2000 (U.S. Bureau of Mines 1977a). A summary of the worldwide reserves is presented in Table 3-6.

3.5 SUMMARY

In 1976, domestic mine production of lead was about 518,000 kkg. The total primary U.S. smelter/refinery production was 597,000 kkg (domestic plus imported ores), while secondary production (scrap processing) contributed 526,000 kkg to the U.S. supply. Approximately 49% of the industrial supply was consumed in the manufacture of lead-acid storage batteries and the production of battery oxides. About 14% of the supply was consumed for the production of antiknock gasoline

TABLE 3-5. U.S. FORECASTS FOR LEAD DEMAND, 1976 and 2000

	Lead (kkg)			
	1976	Forecast Range, 2000		Probable
Production (Demand				
Primary	843,510	934,210	2,122,380	1,333,290
Secondary	526,060	616,760	1,251,660	780,020
End Uses				
Gasoline Additives	217,461	36,280	181,400	72,560
Transportation	710,181	1,106,540	2,312,850	1,514,690
Construction	45,350	45,350	136,050	63,490
Paints	96,142	81,630	154,190	117,910
Ammunition	66,211	54,420	136,050	81,630
Electrical	116,096	63,430	181,400	136,050
Other	118,129	90,700	272,100	126,980

Source: U.S. Bureau of Mines (1977a).

TABLE 3-6. SUMMARY OF WORLD LEAD RESERVES

	Reserve[a]	Other[b]	Total
North America			
United States	25.8	45.9	71.7
Canada	11.7	18.2	29.9
Mexico	4.1	5.0	9.1
Other	.7	1.1	1.8
Total	42.3	70.2	112.5
South America			
Brazil	2.4	2.2	4.6
Peru	3.2	4.1	7.3
Other	1.7	1.9	3.6
Total	7.3	8.2	15.5
Europe			
W. Germany	4.1	5.0	9.1
Bulgaria	2.7	2.7	5.4
Yugoslavia	2.7	2.7	5.4
Spain	3.0	3.4	6.4
Poland	2.3	3.2	5.5
Sweden	2.3	1.4	3.7
Other	5.6	5.3	10.9
Total	22.7	23.7	46.4
Africa			
Morocco	1.4	1.4	2.8
S.W. Africa	1.4	2.3	3.7
Algeria	.6	2.1	2.7
S. Africa	5.1	4.0	9.1
Other	1.3	2.4	3.7
Total	9.8	12.2	22.0
Asia			
U.S.S.R.	16.3	16.3	32.6
Peoples Republic of China	-	-	-
Iran	1.8	1.8	3.6
Other	4.0	3.3	7.3
Total	24.8	24.1	48.9
Oceania: Australia	17.1	14.7	31.7
World Total	124.0	153.1	277.0

[a]Measured and indicated.

[b]Includes inferred and hypothetical resources.

Source: Kirk-Othmer (1967).

additives. Other important use categories include red lead and litharge (5%), ammunition (4%), and solder (4%). The remaining 15% is divided among eighteen use categories. Each of the categories represents about 1% or less of the total supply, while about 9% is contained in industry stocks.

Of the identified releases of lead to the environment (355,000-360,000 kkg), about 53% was airborne and 46% was in the form of solid waste. Less than 1% of the total releases was identified as being released to surface waters or POTWs.

Of the estimated 190,000 kkg of lead released to the atmosphere, 93% was attributed to the use of gasoline antiknock additives. These releases are widely distributed; however, they are more concentrated in urban or heavily trafficked areas. Approximately 2% of the identified releases was attributed to fossil fuel combustion, and about 1% each to lead production, battery manufacture, copper and zinc smelting, and iron and steel manufacture.

The identified releases to the aquatic environment are relatively low (800-950 kkg). Of these, 53-63% was attributed to iron and steel production, 21% to lead production, 6% each to battery production and the nonferrous metal industry, and 4% each to the inorganic chemical and the pulp and paper industries. The remaining 2% of identified releases was attributed to pigment manufacture, the textile industry, and POTW discharges.

Solid wastes represent a large portion (165,000-168,000 kkg) of the total releases of lead to the environment. Lead production accounts for a large portion of these releases (34%) as well, and ammunition use could account for about 30%. Other important sources include solder, weights and ballast, bearing metals, and iron and steel production.

There are numerous uncertainties in the materials balance for lead. The releases from the production, use, and disposal of lead-acid storage batteries are largely unknown, although this is the largest single use of lead. In addition, releases from other uses, although minor, may be significant. The releases from several sources are largely unknown, or have only been roughly estimated; however, they may be significant on a local scale. The lack of data on which to estimate aquatic releases in general is of concern. Although they are likely to be low when compared with airborne releases and solid wastes, they may be significant on a local scale.

Urban runoff probably represents the major source to aquatic environment. Estimates are not shown in the materials balance (Table 3-1) because these releases represent an indirect source of atmospheric emissions. However, this source may represent as much as 21,000 kkg of lead discharging to surface waters and POTWs.

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4.0 FATE AND DISTRIBUTION OF LEAD IN THE ENVIRONMENT

4.1 INTRODUCTION

This chapter describes the levels of lead that have been observed in the environment and the environmental pathways that may result in these levels. Extensive data support these areas. Here, however, only the major topics will be discussed, including summaries of the available data.

4.2 DISTRIBUTION OF LEAD IN THE ENVIRONMENT

There is a large data base concerning the concentrations of lead in the environment. Nriagu (1978a,b) and Chow (1978) have reviewed the literature extensively. The following discussion is meant to address the key points and to briefly review concentrations of lead that are found in the United States.

4.2.1 Water and Sediment

4.2.1.1 Freshwater

Patterson (1965) estimated that the background level in surface waters during preindustrial times was 0.5 μg/l. Current global levels of lead in water are 1-10 μg/l (Livingston 1964).

Anthropogenic sources are industrial discharge, fallout, washout, and runoff, with the primary source being lead emitted to the air as a result of automobile use. Fleischer (1973) has estimated that these anthropogenic sources account for over 90% of the lead in U.S. surface waters.

Typical lead levels in U.S. surface waters are less than 25 μg/l. Lead concentrations were detected in 63% of the samples at a range from 1-55 μg/l with an average of 3.9 μg/l in a study of 749 locations (Durum et al. 1971) and Kopp (1969) reported a mean concentration of 23 μg/l in water sampled from 130 stations across the United States. This is a mean of the positive values and lead was detected in 19% of the 1580 samples.) Bradford (1971) reported an average concentration of 3 μg/l in California surface waters (ranging from <0.5 μg/l to 25 μg/l). Mathis and Cummings (1973) found a range of 1-18 μg/l in the Illinois River and Mancy (1971) reported a range of 0.6-3.3 μg/l in the Great Lakes. Levels of lead in surface waters and sediment are summarized in Tables 4-1 and 4-2.

4.2.1.2 Seawater

Seawater generally contains low levels of lead. Settle and Patterson (1980) estimate that prehistoric lead levels in the ocean were 0.0005 μg/l and report that present day levels are 0.005 μg/l. For nearshore potentially contaminated waters, Patterson (1974) reported levels of 0.014-0.08 μg/l off the Southern California coast. Patterson et al. (1976) found similar results on the east coast: 0.02-0.04 μg/l in Georgia, and 0.14 μg/l in the Long

TABLE 4-1. CONCENTRATIONS OF LEAD IN WATER

Description	Concentration Mean	Concentration Range	Reference
Background	0.5		Patterson (1965)
Global level		1-10	Livingston (1964
U.S.	3.9	1-55	Durum *et al.* (1971)
U.S.	23	2-140	Kopp and Kroner (1967), Kopp (1969)
Stream near smelter -- MO	300		Gale and Wixson (1979)
Surface waters -- CA	3	0.5-25	Bradford (1971)
Illinois River		1-18	Mathis and Cummings (1973)
Great Lakes		0.6-3.3	Mancy (1971)
Rainwater -- U.S. Urban	36		Lazarus *et al.* (1970)
Rainwater -- CA Urban	38		Chow and Earl (1970)
Rainwater -- NH Rural	13.4		Schlesinger *et al.* (1974)
Rainwater -- NB	4.3		Struempler (1976)
Snow -- NB Rural	7.8		Struempler (1976)
Seawater -- Background	0.0005		Settle and Patterson (1980)
Seawater -- Present day	0.005		Settle and Patterson (1980)
Nearshore contaminated (CA)		0.014-0.08	Patterson (1974)
Nearshore contaminated (GA)		0.02-0.04	Patterson *et al.* (1976)
Nearshore contaminated (L.I. Sound)	0.14		Patterson *et al.* (1976)
Groundwater -- MO		2.9-3.8	Proctor *et al.* (1974)
Groundwater -- CA	3	0.5-10	Bradford (1971)
Storm runoff -- NC	1500	100-12,000	Bryan (1974)
Storm runoff	5500		Newton et al. (1974)

TABLE 4-2. CONCENTRATIONS OF LEAD IN SEDIMENT

Description	Concentration (mg/kg) Mean	Range	Reference
Coastal sediment	87	1-912	Nriagu (1978a)
California Coast -- Surface	30-50		Chow *et al*. (1973)
California Coast -- Lower levels	10		Chow *et al*. (1973)
Great Lakes -- Surface	109		a
Great Lakes -- Lower level	28		a
Wisconsin Lakes -- Surface	918		b
Wisconsin Lakes -- Lower level	48.8		b
River sediment	23		Fitchko and Hutchinson (1975)
U.S. -- 1973-79	27-267		U.S. EPA (1980)

[a]Thomas (1976), Edgington and Robbins (1976), Kemp and Dell (1976).

[b]Peterson (1973), Iskander and Keeney (1974)

Island Sound, NY. Both Patterson (1974) and Settle and Patterson (1980) believe that most studies, before 1970, overstated the concentration of lead in ocean water by as much as an order of magnitude.

4.2.1.3 Rainfall

In some situations, rainfall appears to contain relatively high levels of lead. Lazarus et al. (1970) correlated the consumption of gasoline with concentrations of lead in rainwater collected nationally at 32 urban stations. They found an average level of 36 ug/l, which agrees with a study by Chow and Earl (1970) that reported an average concentration of 38 µg/l in rainwater collected at San Diego, CA. Schlesinger et al. (1974) measured lead concentrations in the precipitation of a rural area in New Hampshire. Their measurements, an average of 13.4 µg/l, showed lower levels than those found in urban settings. Struempler (1976) reported an even lower lead content, an average of 4.3 µg/l, in rainwater collected in rural areas of Nebraska in 1973; snowfalls in the same area were slightly higher, with a mean value of 7.8 µg/l.

4.2.1.4 Urban Runoff

Extremely high levels of lead have been reported in urban runoff. Bryan (1974) reported levels in Durham, NC runoff, ranging from 100 µg/l to 12,000 µg/l, with an average of 1500 µg/l. Newton and co-workers (1974) compared levels up to 5500 µg/l with a concentration of only 90 µg/l in an open field. Although these are unusually high levels for most water bodies, Kopp and Kroner (1967) reported levels ranging from 80-200 µg/l in the Mississippi River.

4.2.1.5 STORET Data

The STORET Water Quality System contains a large amount of monitoring data on lead. Because of the size of the data base, only detectable levels were used in the following analysis. These data comprise roughly 60% of all total lead observations for the period considered here -- 1970-79. Thus, the mean values discussed below are actually means of the detected values.

Lead concentrations in ambient waters appear to have declined in the United States since 1970. Figure 4-1 illustrates the trend of lead concentrations from 1970 to 1979. From 1970 through 1973, mean concentration decreased to 70 µg/l; to date, it has fallen below 40 µg/l. It is impossible to say whether these results reflect an actual decline or change in analytical methodology or detection limits.

To observe regional variations of lead concentrations, the data were disaggregated to the major basin level. Most major basins across the country reported mean values >50 µg/l, from 1970 to 1972; however, in

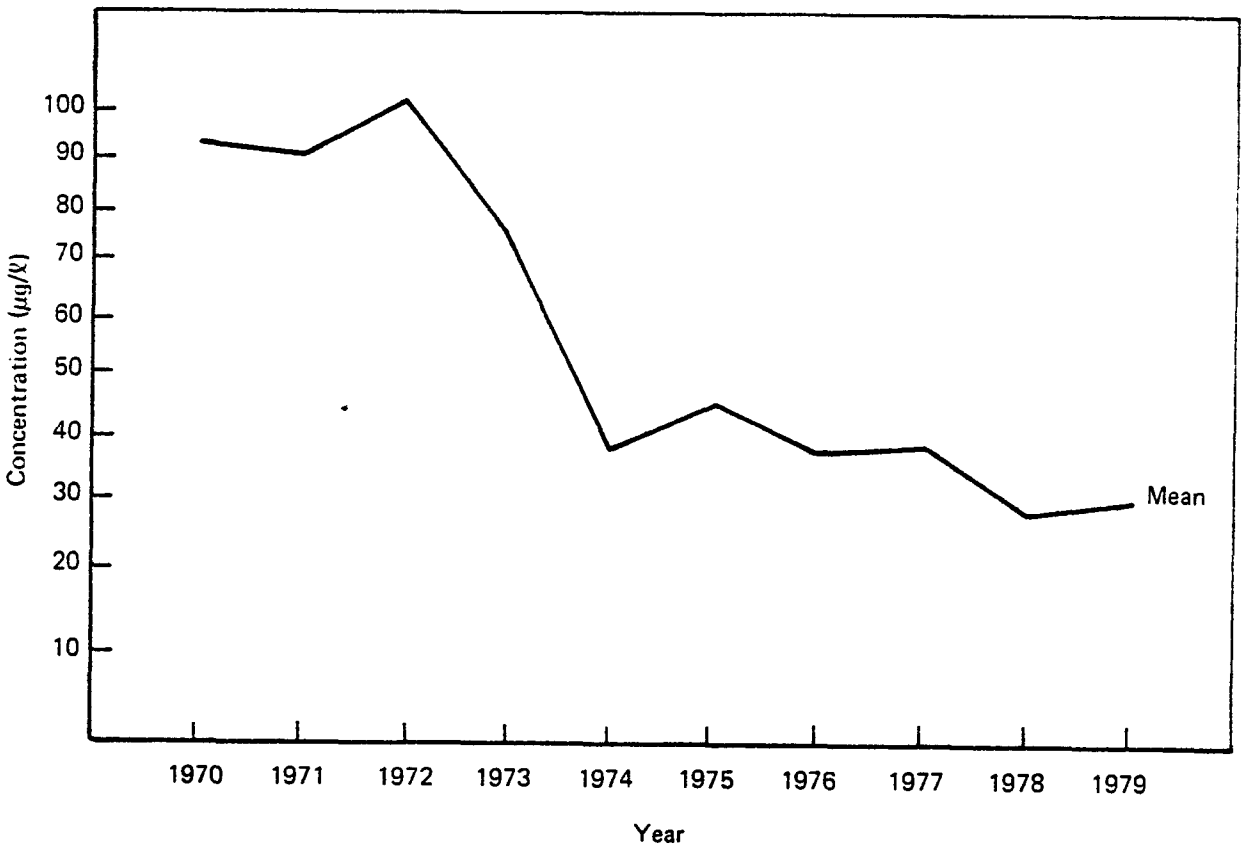


FIGURE 4–1 MEAN AMBIENT VALUES OF LEAD CONCENTRATIONS IN U.S. SURFACE WATERS, 1970-79 (DETECTED LEVELS ONLY)

more recent years, the conditions have altered. Since 1973, mean values for major river basins in the East, South and Midwest have been < 50 μg/l. The higher concentrations of lead appear in the western regions of the country, specifically in the Pacific Northwest and California basins. In the Pacific Northwest basin, mean concentrations, from 1970 to 1973, fluctuated between 133-336 μg/l. In 1976, concentrations decreased, resulting in a mean value of 127 μg/l. Mean levels have declined annually since 1976 to 69 μg/l in 1977, to 61 μg/l in 1978, and to 32 μg/l in 1979. Similarly, for the California basin, mean concentrations of lead in the early portion of the time period appeared in the range 80-228 μg/l. A decline has occurred annually from a mean level of 105 μg/l in 1976 to 43 μg/l in 1979.

The literature indicates higher concentrations of lead near highways, in urban areas, and, in general, where traffic is heaviest. Retrievals from STORET were used to contrast urban and rural lead concentrations. Areas in the East, Midwest, and West were selected and data were aggregated by county.

Retrievals from STORET were consistent with the literature. Mean lead concentrations in counties that were mostly urban, on the average, were fifteen times higher than the mean concentrations of rural counties. For instance, in New York, the urban county of Nassau was contrasted with Madison and Steuben counties of upstate New York. In Nassau county, mean concentrations were in the range of 13-65 μg/l, with maximum levels from 37 to 300 μg/l. In Madison and Steuben counties, the mean and maximum concentrations ranged from undetected to 14 μg/l and undetected to 37 μg/l, respectively. Similar results appeared for Los Angeles when contrasted with Fresno and Kern counties and for Cook county (Chicago, IL) contrasted with the rural counties of Livingston, Shelby, and Whiteside. Table 4-3 presents the urban and rural lead concentration results.

4.2.1.6 Lead in Sediment

In aquatic environments, lead is found at much higher concentrations in the sediment than in the water column. In coastal sediment, lead concentrations range from 1 mg/kg to 912 mg/kg with a mean value of 87 mg/kg (Nriagu 1978a). The highest concentrations remain in the surface layer; Chow *et al*. (1973) reported an average of 10 mg/kg in deep layers on the California coast compared with levels of 30, 29, and 50 mg/kg on the surface. The average concentration for the Great Lakes was 109 mg/kg at the surface and 28 mg/kg in the deeper layers (Thomas 1976, Edgington and Robbins 1976, Kemp and Dell 1976). The mean concentrations for a sampling of lakes in Wisconsin were 918 mg/kg in the surface layer and 49 mg/kg in the deeper layers (Peterson 1973, Iskander and Keeney 1974). The surface layers appear to contain three to five times the concentration of lead as deeper layers (see Table 4-2).

Fitchko and Hutchinson (1975) have estimated the average concentration of lead in river sediments to be 23 mg/kg. Nriagu (1978a) has

TABLE 4-3. MEAN AND MAXIMUM RANGES OF LEAD CONCENTRATIONS IN AMBIENT SURFACE WATERS OF URBAN AND RURAL AREAS

Urban/Rural Counties	Concentrations (μg/l)[a]	
	Maximum Range	Mean Range
East - New York		
Nassau (urban)	37-300	13-65
Madison (rural)	ND-4	ND-3
Steuben (urban/rural)	4-37	2-14
Midwest - Illinois		
Cook (urban	20-1700	15-71
Livingston (rural)	ND-40	ND-8
Shelby (rural)	ND-10	ND-1
Whiteside (urban/rural)	10-20	1-5
West - California		
Los Angeles (urban)	178-2400	53-309
Fresno (urban/rural)	ND-370	ND-89
Kern (urban/rural)	1-45	1-29

Note: ND = not detected.

[a] Ranges generally include detectable levels only, except where ND is indicated.

Source: U.S. EPA (1980).

estimated lead levels in polluted environments at about four times this value, 98 mg/kg.

In the STORET data base, lead concentrations in sediment have been recorded in only one-third of the major river basins, mainly in the East and Midwest. Over a 7-year period, 1973-79, maximum concentrations of lead in sediment ranged from 440-1000 mg/kg and mean concentrations from 27-267 mg/kg. In general, lead concentrations in sediment are two to three orders of magnitude higher than lead concentrations in ambient waters (U.S. EPA 1980).

4.2.2 Air

Nriagu (1978b) has an extensive discussion on the levels and forms of lead in the atmosphere; thus, they will only be discussed briefly here.

Background lead levels are present in the atmosphere (Table 4-4) as the result of releases from natural sources, including windblown dust, plant exudates, forest fires, vulcanism and radioactive decay (Nriagu 1978b). Using geochemical evidence, Patterson (1965) estimated the pre-industrial levels of lead in the atmosphere to be approximately 0.6 ng/m^3. This figure is an order of magnitude lower than the lowest level reported for the continental United States (8 ng/m^3). Chow et al. (1972) reported this level at 3800 meters at a relatively uninhabited site. The range of lead levels for remote areas of the continental United States is 0.1-10 ng/m^3 (Nriagu 1978b).

According to Nriagu (1978b), lead released to the atmosphere by natural sources accounts for only a small percentage, approximately 4%, with the remaining 96% accounted for by anthropogenic sources. In air, the primary anthropogenic sources of lead are internal combustion engines, lead smelting, and steel production.

Lead concentrations are greater in urban or more developed areas. Nriagu (1978b) reported an average range of 0.5-10 $\mu g/m^3$ in the developed areas, 0.1-1 $\mu g/m^3$ in more rural areas, and < 0.01 $\mu g/m^3$ in locations distant from developed areas. Measurements of concentrations in the immediate vicinity of traffic movement were usually found to exceed 10 $\mu g/m^3$. For the United States, urban lead concentrations ranged from a relatively low level of 0.14 $\mu g/m^3$ reported for Los Alamos by Tepper and Levin (1972) to a high of 7.5 $\mu g/m^3$ reported in mid-Manhattan by Chow (1973). Data collected in the U.S. EPA's National Air Sampling Network (NASN), during 1966 and 1967, showed average urban levels of 1.1 $\mu g/m^3$. Nonurban areas averaged 0.21 $\mu g/m^3$ near the city, .096 $\mu g/m^3$ at increasing distances from cities, and 0.022 $\mu g/m^3$ in remote rural areas (McMullen et al. 1970). These data indicate an average urban level almost ten times higher than concentrations found in rural settings.

The correlation between atmospheric lead concentrations and automobile usage is supported by data indicating increased levels paralleling

TABLE 4-4. CONCENTRATIONS OF LEAD IN THE ATMOSPHERE

Description	Concentration ($\mu g/m^3$) Mean	Range	Reference
Baseline-preindustrial	0.0006		Patterson (1965)
Lowest level -- Continental U.S. (White Mountains, CA)	0.008		Chow *et al.* (1972)
Global -- Urban		0.5-10	Nriagu (1978b)
Global -- Rural		0.1-1	Nriagu (1978b)
Global -- Remote		0.0001-0.01	Nriagu (1978b)
Boston -- Spring	3.9		O'Brien *et al.* (1975)
Boston -- Winter	5.4		O'Brien *et al.* (1975)
Montana -- Near smelter		0.4-4	U.S. EPA (1972)
Montana -- Distant from smelter		0.1-0.7	U.S. EPA (1972)
Idaho -- Near smelter	147.9	60-254	Ragaini *et al.* (1977)

increases in gas consumption. Chow (1973) examined both the 1961-1962 Tri-City Project conducted in Los Angeles, Philadelphia and Cincinnati and the 1968-1969 Seven-Cities Project conducted in the above cities and additionally New York, Chicago, Houston, and Washington, DC, and found significant increases in lead concentrations over that time period. Los Angeles had the highest increase, 56%, with smaller increases of 19% in Philadelphia and 17% in Cincinnati.

Seasonal trends in atmospheric lead levels based on both automobile usage patterns and climate conditions are also discussed in the literature. A 1972 study by O'Brien et al. (1975) shows a variation from 3.9 $\mu g/m^3$ in the spring to 5.4 $\mu g/m^3$ in the winter in Boston. Colucci et al. (1969) reported 4.9 $\mu g/m^3$ in the spring and 10.3 $\mu g/m^3$ in winter in the 1960-65 Los Angeles Study. These studies indicate a possible average seasonal variation of over 50%.

A large proportion of lead emitted in automobile exhaust settles quickly in the vicinity of the source. Nriagu (1978b) states that lead levels in the atmosphere generally decrease by about 50% between 10 and 50 meters from highways. Lead concentrations also decrease sharply with increased height from the street level. Schroeder (1974) reported levels of 14.3 $\mu g/m^3$ at 0.61 meter, 8.3 $\mu g/m^3$ at 1.5 meters, and 5.1 $\mu g/m^3$ at 0.2 meter.

In addition to the impact of automobiles in urban areas, local point sources also contribute to levels of lead in the atmosphere. The U.S. EPA (1972) measured lead levels in East Helena, MT, which contains an industrial smelter complex; in 1969, concentrations ranged from 0.4-4 $\mu g/m^3$ depending on the distance from the plant. The maximum daily concentration was 15 $\mu g/m^3$. In neighboring Helena, MT, in 1969, an average daily concentration of 0.1 $\mu g/m^3$ up to a maximum of 0.7 $\mu g/m^3$ was reported. In 1976, Ragaini and co-workers (1977) conducted a study in Kellogg, ID, near a lead smelting complex. Air samples taken at the town hall ranged from 60-254 $\mu g/m^3$, with a mean value of 147.9 $\mu g/m^3$.

4.2.3 Soil

All soils contain detectable levels of lead, ranging in concentration from <1 mg/kg to >100,000 mg/kg (10%) in lead ores. Averages in the literature range from 10 mg/kg (Bowen 1966) to 37 mg/kg (Cholak et al. 1961) for uncontaminated soils (see Table 4-5). For an average of all soils in the United States, Warren et al. (1971) estimated a lead content of 20 mg/kg. DeTreville (1964) presents data indicating a concentration of 16 mg/kg for the earth's crust, but with values as low as 0.04 mg/kg in alluvial soil. In an estimate derived from surveys of the literature, Nriagu (1978a) reported an average concentration of 18 mg/kg for the United States. The natural sources that contribute to this background level include weathering of rocks, volcanoes, and erosion of ore deposits.

Like lead in the atmosphere, the majority of anthropogenic lead in the soil results from the deposition of lead emitted from the combustion of leaded gasoline and, to a lesser degree, the emissions from industrial

TABLE 4-5. CONCENTRATIONS OF LEAD IN SOIL

Description	Concentration (mg/kg) Mean	Range	Reference
Uncontaminated soil	10-37		Bowen (1966), Cholak *et al.* (1961)
Earth's crust	16	Minimum-.04	DeTreville (1964)
All U.S. soils	20		Warren *et al.* (1971)
All U.S. soils	18		Nriagu (1978a)
Chicago Expressway -- <13.7 miles		Maximum-7600	Kahn *et al.* (1973)
Chicago Expressway -- 46 miles		Maximum-900	
Street dust	555		Rameau (1973)
Dust -- residential areas (IL)		656-3067	Schroeder (1974)
Dust -- commercial areas (IL)		1774-3549	Schroeder (1974)
U.S. city dust -- residential	1636		NAS (1972)
U.S. city dust -- commercial	2413		NAS (1972)
Urban soil -- U.S. cities	523	99-834	Chow *et al.* (1975)
Montana -- 1 mile from smelter	4000		U.S. EPA (1972)
Montana -- 2 miles from smelter	600		U.S. EPA (1972)
Montana -- 4 miles from smelter	100		U.S. EPA (1972)
Idaho smelter -- .7-12 miles		300-7600	Ragaini *et al.* (1977)
Soil in vicinity of a barn	2000		Ter Haar and Aronow (1974)
Soil in vicinity of a wooden house		450-6338	Ter Haar and Aronow (1974)
Cincinnati house		32-7620	Bertinson and Clark (1973)
Old house		440-490	Bogden and Louria (1975)
Orchard soil -- no pesticides		3-76	Chisolm and Bishop (1967)
Orchard soil -- pesticides with lead		7-360	Chisolm and Bishop (1967)

activities, such as lead smelting. A 40-year study of soils in areas of high traffic (Page and Ganje 1970) showed an increase in lead concentration from 17 mg/kg to over 50 mg/kg in 1968. Kahn and co-workers (1973) reported levels of lead in soil along a Chicago expressway as high as 7600 mg/kg at distances <13.7 meters from the highway and 900 mg/kg as far away as 46 meters. Before the use of automobiles, Nriagu (1978a) theorizes that the primary source of lead contamination was flue dust created by burning coal.

Lead in urban soils tends to be somewhat lower than that found in the immediate vicinity of highways. In a sample of nine U.S. cities, Chow et al. (1975) reported soil levels in inner city parks ranged from 99 mg/kg in a Houston park to 834 mg/kg in New York's Central Park. As a result of sampling 16 sites, the average concentration for these cities was 523 mg/kg.

High levels of lead are also found in urban dust. Sartor and Boyd (1972) found the following lead concentrations in dust from various sites: industrial (840 mg/kg), residential (1100 mg/kg), and commercial (1400 mg/kg). Rameau (1973) observed that the mean lead content of street dust was 555 mg/kg, with 90% consisting of particles < 250 μ. Although these are fairly wide ranges of concentrations, all city dust contains relatively high levels of lead, with residential areas lower than commercial areas. Schroeder (1974) reported 656-3067 mg/kg for residential areas, and 1774-3549 mg/kg for commercial areas in nine Illinois cities. Similar studies were conducted in several states. With the exception of Virginia and Kentucky, the average level of concentration in commercial areas is almost double the residential level. NAS (1972) compared 77 cities in the United States and found levels of 1636 mg/kg at residential sites compared with 2413 mg/kg at commercial sites.

Although many authors have noted a decrease in the soil concentration of lead with increasing distances from the roadway (Nriagu 1978a), lead concentration also appears to decrease with increasing depth in soil. Numerous authors, including Tolonen (1974), Ward et al. (1975), and Rolfe and Haney (1975), have reported that lead concentrations above background levels are uncommon below a depth of 10 centimeters.

Although the effects of automobile exhaust have a wide national impact, point source lead can produce extremely high local lead concentrations. Lead smelting operations probably have the most significant impacts on nearby soils. In Helena, MT, the U.S. EPA (1972) reported concentrations of 4000, 600, and 100 mg/kg of lead at distances of 1, 2, and 4 miles from a nearby smelter. Ragaini and co-workers (1977) studied a lead mining and smelting operation near the town of Kellogg, ID, where the majority of ambient lead originated in airborne emissions from the complex. One of the highest concentrations in the soil, 7600 mg/kg, was at the site closest to the complex, 0.7 mile, with other site observations varying from a fairly high reading of 2200 mg/kg 12 miles away to 300 mg/kg at a site 5 miles away. Unlike lead from automobile

exhausts, the data show that smelter emissions affect areas up to 25 kilometers away (Bolter et al. 1975, Koirtjohann et al. 1975).

Soil samples in the vicinity of lead-painted houses have been found to contain significant lead concentrations. Ter Haar and Aronow (1974) reported soil levels ranging from 450-6338 mg/kg next to a wooden house in Michigan and 2000 mg/kg near a Massachusetts barn. Bertinson and Clark (1973) reported lead levels ranging from 32-7620 mg/kg in the soil around a nineteenth century house in Cincinnati, and Bogden and Louria (1975) found levels, averaging 175 mg/kg before and 440-490 mg/kg after paint removal, in the soil surrounding an old lead-painted house.

Chisolm and Bishop (1967) have demonstrated the consequences of using pesticides containing lead; lead levels of 7-360 mg/kg were found in orchard soils compared with levels of 3-76 mg/kg in adjacent nonorchard soils.

4.2.4 Biota

Numerous authors have shown that lead concentrations in biota correlate with the concentration of lead in the surrounding environment. Thus, background levels for biota are related to those previously described for soil, water, and the atmosphere. In a study of pine needles in North America, Elias and co-workers (1976) reported lead concentrations of 10 μg/kg. It is believed that one-half of these concentrations comes from natural sources. Smith (1973) reported that grass growing in a relatively unpolluted environment contained lead levels of ~1-5 mg/kg. In an extensive study of naturally-occurring levels of lead in bird tissues, Bagley and Locke (1967) found concentrations in the range of 0.5-3.7 mg/kg. Settle and Patterson (1980) have estimated that natural lead levels in tuna, during prehistoric times, was ~.03 μg/kg. Studies of current effects of lead pollution have shown concentrations of lead in the biota at over 900 times these baseline levels (Settle and Patterson 1980).

A summary of selected levels of lead found in biota is contained in Table 4-6. In general, elevated levels are found near highways and point sources, such as smelters; in areas where lead-containing pesticides have been used; and in terrestrial birds that have consumed lead shot.

4.2.5 Limitations of Monitoring Data

In the evaluation of the monitoring data previously discussed, the results of Settle and Patterson (1980) must also be considered. These data show that background levels of lead, especially in tuna, have been overestimated by as much as a factor of 1000. These authors suggest that overestimates have occurred as a result of contamination in sample collections and analyses. Thus, the data presented in the previous

TABLE 4-6. CONCENTRATIONS OF LEAD IN BIOTA

Description	Concentration (mg/kg) Mean	Range	Reference
Vegetation			
Background level - pine needles	0.01		Elias et al. (1976)
Grass		1-5	Smith (1973)
Trees, shrubs, twigs -- Southeast	154	< 10-3000	Connor and Shacklette (1975)
Leaves -- Southeast	70	< 10-2000	Connor and Shacklette (1975)
Salt marsh-cordgrass -- MA		5.4-23.2	Banus et al. (1974)
Marsh plants -- dredge-spoil site	6.2		Drifmeyer and Odum (1975)
Leaf litter -- MO (lead smelter)		4,379-39,636	Gale and Wixson (1979)
Grass -- ID (smelter)		320-10,000	Ragaini et al. (1977)
Vegetables -- MO (lead belt)		8.8-114	Hemphill et al. (1971)
Vegetables -- control counties		<5-20.6	Hemphill et al. (1971)
Vegetables -- NJ (9 meters from road)		18-1742	Motto et al. (1970)
Vegetables -- NJ (76 meters from road)		8-212	Motto et al. (1970)
Terrestrial Animals			
Insects -- high traffic volume	~17		Price et al. (1974)
Insects -- low traffic volume	~ 3.8		Price et al. (1974)
Insects -- high traffic volume	8.6		Giles et al. (1973)
Insects -- low traffic volume	1.7		Giles et al. (1973)
Earthworms -- 3-48 meters from road		52.7-270	Gish and Christensen (1973)
Voles & mice -- lead treated orchards (liver)	23.4		
-- untreated areas	~4		Elfving et al. (1978)
Background level -- birds		0.5-3.7	Bagley and Locke (1967)
Duck wings		0.5-361	U.S. DI (1979)
Woodcocks -- U.S.	12.63	4.51-29.79	Scanlon et al. (1978)
Aquatic Animals			
Albacore, tuna, muscle (prehistoric period)	0.00003		Settle and Patterson (1980)
Albacore, tuna, muscle (present day)	0.0003		Settle and Patterson (1980)
Gastropods -- CA		2.6-11.8	Schwimer (1973)
Mollusks -- CA		2.2-931	Graham (1972)
Oysters -- East Coast		0.67-0.88	Kopfler and Mayer (1973)
New tailings ponds -- MO (crayfish)		28-69	Gale et al. (1973)
New tailings ponds -- MO (snails)		39-116	Gale et al. (1973)
Fish -- NY (lakes and streams)	.3-1.5	Max. 3	Pakkala et al. (1972)
Trout -- Cayuga Lake		0.004-0.02	Tong et al. (1974)
River fish - MT	3.85		Pagenkopf and Neuman (1974)

sections, related to background or control values, should be viewed with caution.

4.3 Fate of Lead in the Environment

4.3.1 Introduction

This discussion on the fate of lead in the environment is based on the results of discharges from processes that have been identified as significant contributors of lead to the environment (see Chapter 3.0). Emphasis is on the form of lead and its subsequent transport when released to the environment. Versar, Inc. (1979) has conducted a general overview of the environmental chemistry of lead, which has been used in the formulation of judgments concerning the direction and rate of transport lead assumes in an ecosystem. The literature available also supports these observations.

The major pathways of physical transport and qualitative rates of transport are designated in Figure 4-2. Atmospheric emissions (Pathway #1) include point sources, such as lead production, incineration, smelting, and coal combustion, that contribute to localized pollution; and dispersive sources, such as automobile exhausts, that contribute to the concentrations of lead found in urban air, urban dirt and dust, and urban runoff. Pathway #2 follows the flow of lead that originates from solid waste disposal areas and mine tailings. Because environmental controls may restrain further discharges to air and water, the quantity of lead disposed to land surfaces can be expected to increase. Lead discharges contained within industrial process effluents into local surface waters is reviewed in Pathway #3. The fate of lead in POTWs is described in Pathway #4.

A more general overview of all major pathways of anthropogenic sources of lead is shown in Figure 4-3. The major effect on the air compartment, which is the migration of groundwaters containing lead to nearby surface waters, has not been shown in this figure because: (1) the process is extremely slow, and (2) the current magnitude of this transport pathway is not sufficiently documented. In addition, the high concentration of lead in sediments with respect to the overlying water and the steep profile of lead concentrations in soils subject to contamination is not represented in this figure.

The following discussion is divided into two major sections: (1) 4.3.2, General Fate Processes, which discusses the fate of lead in its various forms when it reaches the air, water, or soil; and (2) 4.3.3, Major Environmental Pathways, which discusses the fate of lead as a result of specific sources.

4.3.2 General Fate Processes

4.3.2.1 Atmospheric Transport

The atmospheric transport of lead depends on particle size, the height of the release, geographical distribution, and the chemical form

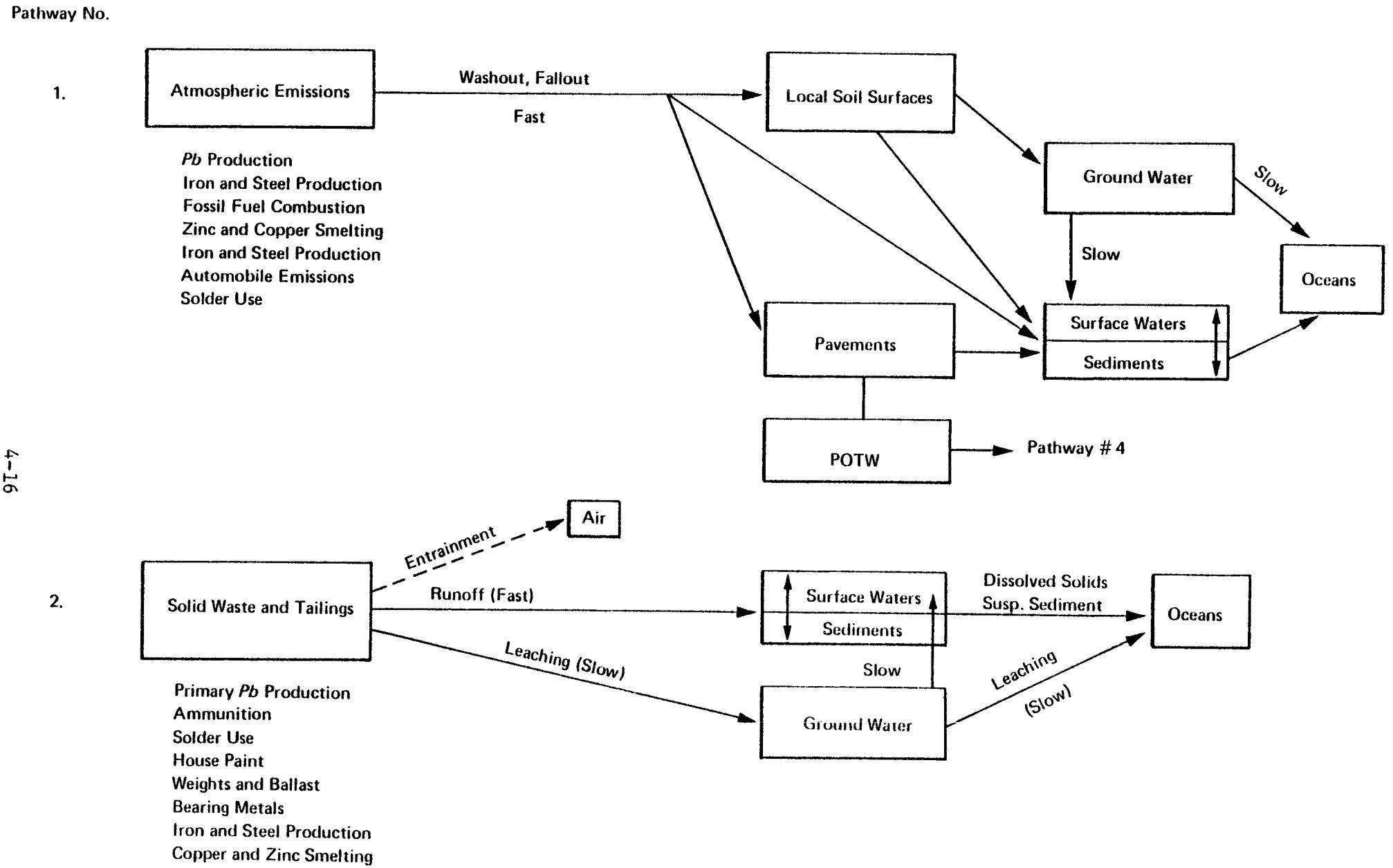


FIGURE 4–2 MAJOR ENVIRONMENTAL PATHWAYS OF LEAD EMISSIONS

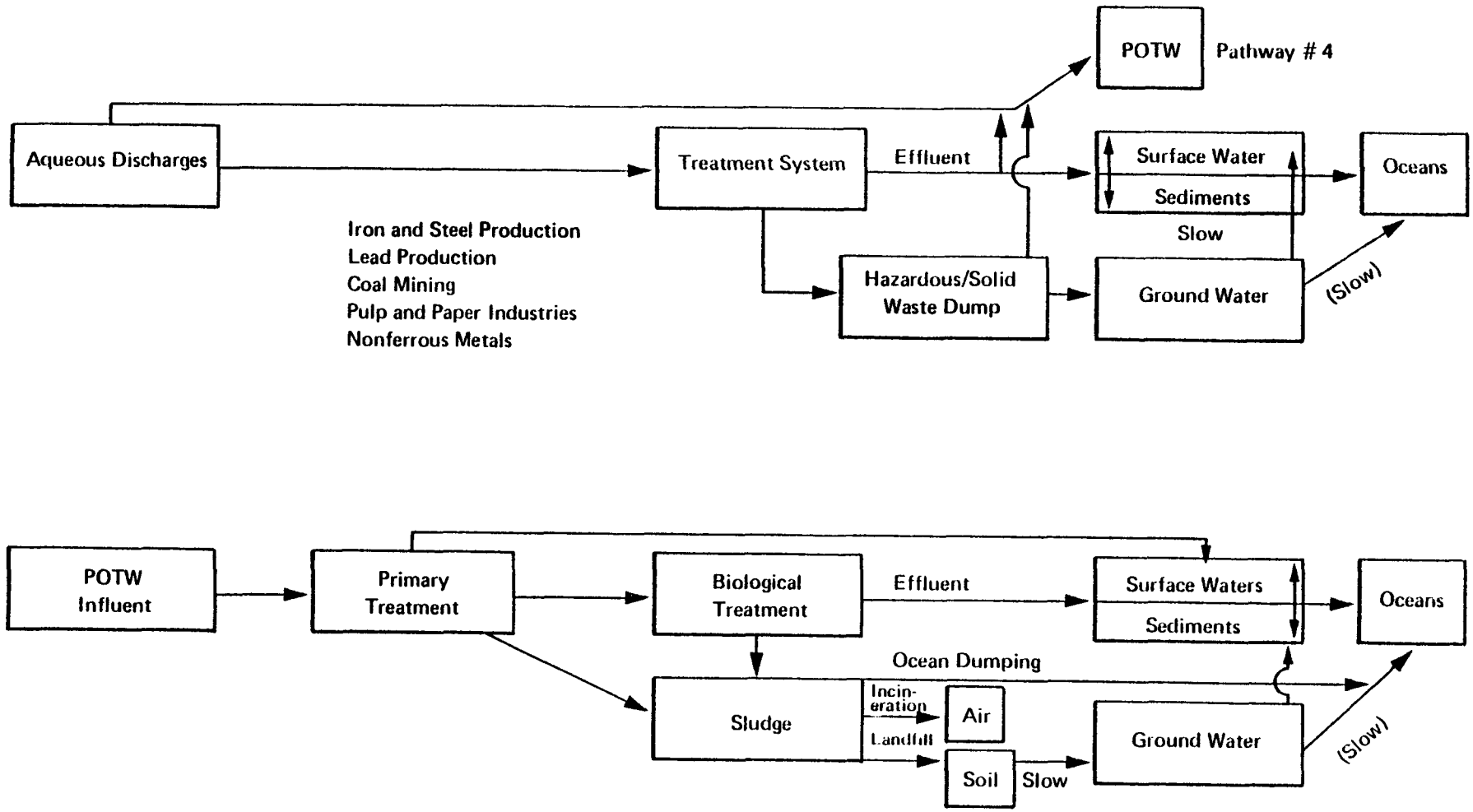
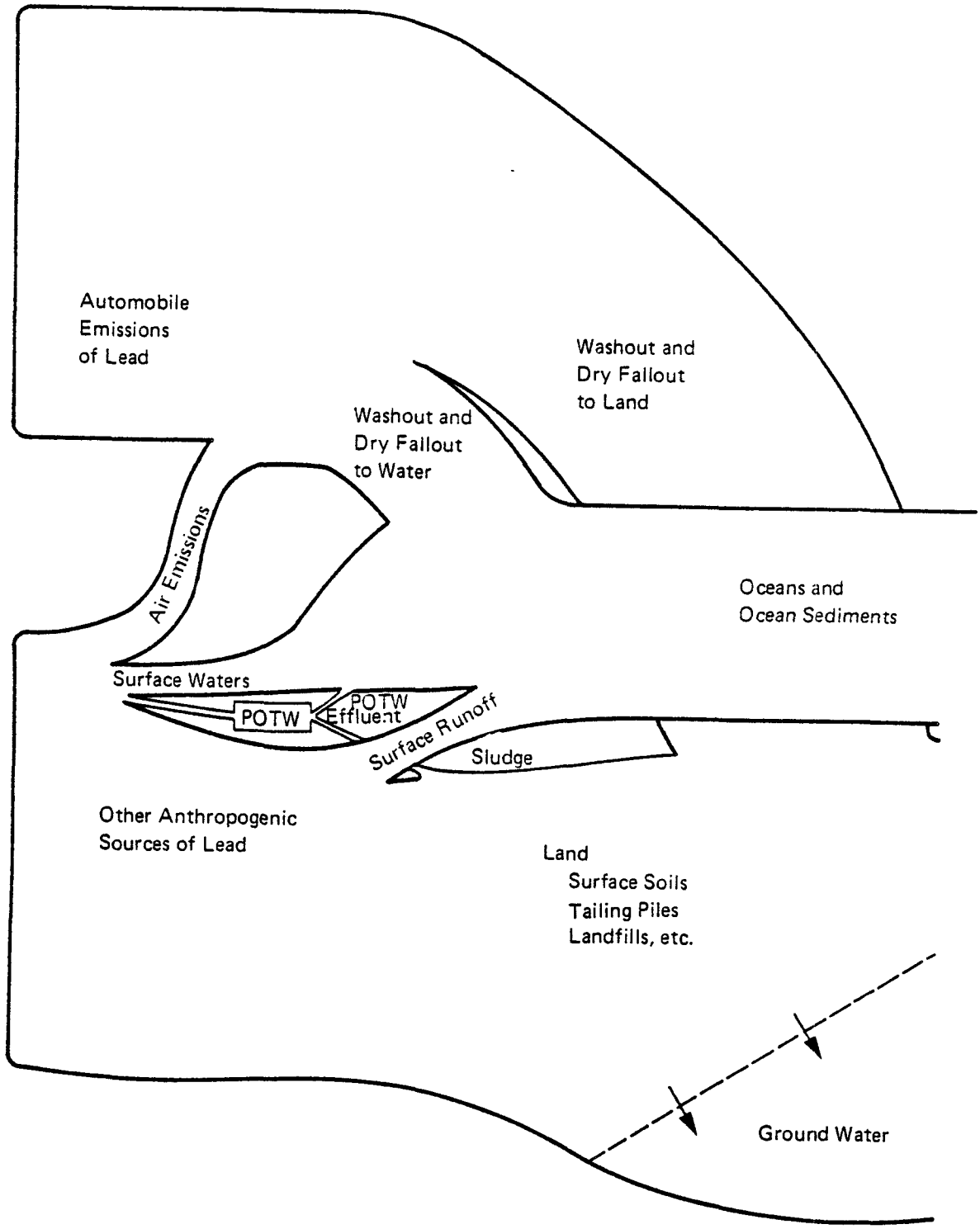


FIGURE 4–2 MAJOR ENVIRONMENTAL PATHWAYS OF LEAD EMISSIONS (Continued)



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

FIGURE 4–3 SCHEMATIC DIAGRAM OF MAJOR PATHWAYS OF ANTHROPOGENIC SOURCES OF LEAD RELEASED TO THE ENVIRONMENT IN THE UNITED STATES, 1976

of lead emitted. Although all these processes are significant, the chemical form probably has the least effect on transport and deposition. Larger particles, particularly >20 μm in aerodynamic diameter, are rapidly deposited. Atmospheric turbulence predominantly controls the deposition of particles <20 μm, by "forcing" the particles to the surface where they are deposited by impaction. Atmospheric parameters control the deposition velocity for particles <20 μm (deposition flux/air concentration), which is relatively independent of particle size and ranges from 0.1 to 10 cm/sec. The deposition velocity for a 20-μm particle ranges from 2 to 10 cm/sec, while particles >50 μm settle at velocities >10 cm/sec (Sehmel and Hodgson 1976).

The initial height of release is another significant factor in the determination of the distribution of airborne lead and its deposition patterns. Particles released near ground level are more likely to contribute to high ground level concentrations, which lead to human exposure, and are more likely to be deposited by impaction, which leads to deposition patterns that decay rapidly with distance from the source, such as those observed near highways and ore mines. It is much less likely that turbulent processes will bring particles released from elevated sources (particularly with diameters <20 μm) to ground level; thus, they will be dispersed over greater distances and will most likely be brought to the ground by precipitation with more widespread impacts. This is to be expected for smelter and coal emissions.

The geographical distribution further distinguishes the major source type. Although a considerably smaller source than automotive emissions on the national scale, ore mining, milling, and smeltering are concentrated in a few isolated major sources. This has led to acute localized impacts on isolated ecosystems. Automotive emissions distributed along every major thoroughfare may lead to lower resultant concentrations but a much greater risk of exposure to lead.

Finally, the chemical form of emitted particulates can affect deposition; lead sulfates and nitrates tend to be hygroscopic and may preferentially be rained out by in-cloud scavenging processes. Otherwise, the chemical form of a particulate is unlikely to affect its atmospheric transport and deposition.

4.3.2.2 Fate Processes in Aquatic Environments

Because the aqueous environmental chemistry of lead has been extensively discussed by Rickard and Nriagu (1978), only a brief discussion will be contained here.

Aqueous Complexation

The concentration of soluble lead in water is directly related to parameters, such as pH, the oxidizing potential of the water (indicated as pE), the presence of competing ions (Ca^{++}, Mg^{++}, Fe^{++}), and the existence of precipitating and complexing agents. According to the

model calculation of Vuceta and Morgan (1978), who used input parameters that simulated an aerated freshwater system, Pb(II) is present as the free metal ion below pH 7.1 and complexed with carbonate ion above that pH. Adsorption and complexation with hydroxide ions account for the remaining lead in the water column. Lead is strongly complexed by organic acids, such as humic and fulvic acids (Rickard and Nriagu 1978). Again, pH controls the equilibrium of complexation: at pH 3.5, the log of the equilibrium constant (log K) is 3.09, while at pH 5 the tendency to complex with fulvic acids increases as indicated by a log K of 6.13. At this pH, lead is exceeded only by copper in its ability to complex with organic acids. In interstitial waters, ∿80% of the total lead may be found complexed to organic molecules (Rickard and Nriagu 1978).

Sediment Adsorption

In natural freshwater systems, lead will be sorbed onto clay preferentially over calcium and potassium ions, with the tendency for sorption decreasing in the following order: kaolinite > illite > montmorillonite (Nriagu 1978a). Various parameters, such as surface area and the crystal type, dictate the adsorption of lead onto hydrous iron and manganese oxides. Although hydrous iron and manganese oxides exert less control over lead than clays and organic matter, lead is strongly adsorbed in comparison with other trace metals. Figure 4-4 indicates that lead is exceeded only by copper in its ability to be sorbed by silica and hydrous oxides (Vuceta and Morgan 1978). Adsorption to these surfaces is essentially irreversible; Rickard and Nriagu (1978) suggest the following mechanism:

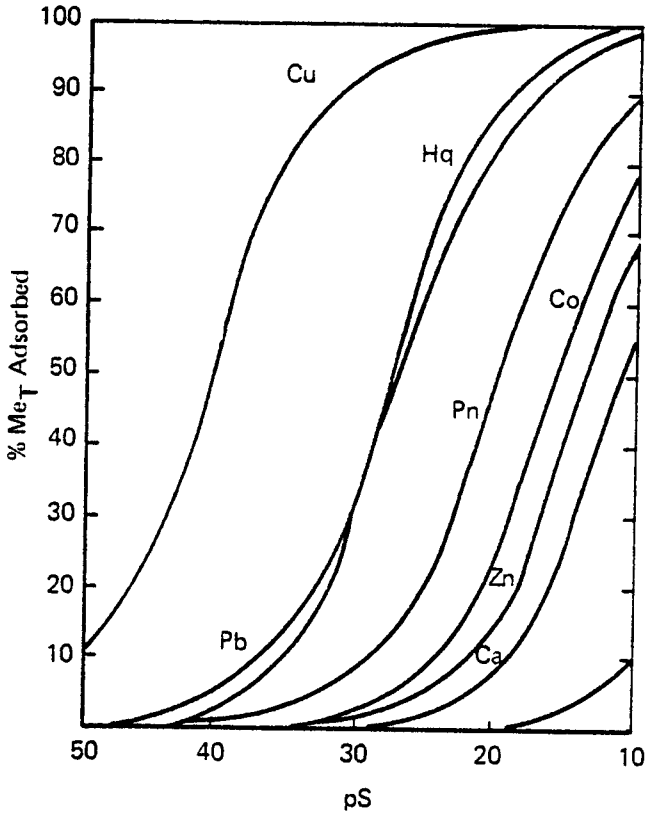
$$Pb^{+2} + MnO_2 + H_2O \longrightarrow \underset{\text{(quenselite)}}{PbMnO_2(OH)} + H^+$$

$$Pb^{+2} + Fe(OH)_3 \longrightarrow \underset{\text{(amorphous)}}{PbFeO(OH)_2} + H^+$$

Serne (1977) conducted several extraction techniques on the sediments of the San Francisco Bay. His results indicated that 45% of the lead was associated with organic matter and sulfides; 50% of the lead was inert to chemical attack, within clay lattices; the remaining 5% of the lead in the sediments was associated with iron and manganese oxides.

Transport of Lead in the Water Column

Perhac (1974) studied three streams in Tennessee to ascertain the nature of trace metal transportation. He found that lead was highly concentrated in suspended solids. Because the suspended solids were such a small percentage of the total solids in the water column, however, the bulk of the lead was transported in the dissolved state. Table 4-7 is a summary of these findings.



Note: (pH = 7, pE = 12, $pCO_2 = 10^{-8.5}$ atm., pCt–4.16) as a function of surface area of SiO_2 in ha/ℓ. pS = -log (SiO_2) ha/ℓ.

Source: Vuceta and Morgan (1978).

FIGURE 4–4 ADSORPTION OF HEAVY METALS IN OXIDIZING FRESH WATERS

TABLE 4-7. CONCENTRATION AND DISTRIBUTION OF LEAD IN THE WATER COLUMN

	Concentration Pb (mg/kg)	% of Mass in the Water Column[a]	% of Pb in Water Column[a]
Water column	0.015		
Dissolved solids	83	94.6	90.8
Colloidal solids	1683	0.17	1.2
Coarse particulates	278	8.96	8.0
Sediments	74.5	-	-

[a]these numbers are averages of 6 sites, and thus do not add to 100%.

Source: Perhac (1974).

Aquatic Biological Pathways

Natural levels of lead are commonly found in freshwater ecosystems and less commonly in marine systems. The metal does not appear to be an essential nutrient; however, it is present at background levels in organisms living in relatively nonpolluted areas. Most studies report tissue concentrations of lead measured in the environment without the specifications of the water concentrations that lead to these levels. These are described in Section 4.2. The subject of lead bioaccumulation has been discussed in detail by Bell et al. (1978), Phillips and Russo (1978), and Wong et al. (1978). The mechanism of bioaccumulation and the parameters that influence it are described briefly in the following section.

Parameters Affecting Uptake

The potential routes for exposure to and uptake of lead by aquatic organisms are ingestion and absorption across the derma and respiratory organs. For invertebrate filter-feeders, ingestion of suspended particulate matter that contain adsorbed or complexed lead is an important route (Armstrong and Atkins 1950). For higher trophic level species, ingestion of detritus vegetation and other animals contaminated with lead is an important route (Bowen and Sutton 1951). Because of the high lead concentrations associated with fish, for them, ingestion of crustaceans may be an important source (Hardisty et al. 1974). Several physiological processes may be involved in complexation of the lead with organic molecules (Schubert 1954), incorporation of lead ions (Williams 1953, Lehninger 1970), and uptake by exchange (Korninga 1952).

Several parameters, both biological and environmental, influence bioaccumulation of lead. Even under uniform exposure conditions, variability between species is important (Wong et al. 1978); however, more controlled assays are needed to determine its significance. In certain fish, bioaccumulation decreases with age, although it may be a result of the consumption of a lower fraction of zooplankton by younger fish (Schell et al. 1974). The calcium status of the exposed individual also influences uptake and retention of lead, which is reduced with increased Ca availability (Varanasi and Gmur 1978). In estuarine fish, the lead concentration in water, duration of exposure, temperature, and water salinity affect bioaccumulation (Sommers et al. 1976).

Bioconcentration Factors (BCF) and Biological Half-lives

Lead bioaccumulation levels and bioconcentration factors (concentration in tissue/concentration in medium) are presented in Table 4-8 for a variety of fresh and marine aquatic species, both invertebrates and vertebrates. The studies were conducted under a wide range of conditions and are not strictly comparable. Most organisms were exposed to lead dissolved in water; however, a few studies exposed species to suspended particulate lead or to contaminated sediment. In some cases, lead concentrations were measured in specific organs rather than in the

TABLE 4-8. LEAD BIOACCUMULATION LEVELS AND BIOCONCENTRATION FACTORS IN AQUATIC SPECIES

Species	Water Concentration (µg/l)	Tissue Concentration (mg/kg)	BCF (Approximate)	Reference
Oysters (*Crassostrea virginia*)	0.5-3.0 (Mobile, AL)	0.67-0.88[a]	200-1760	Kopfler and Mayer (1973)
"	25-200	9-115[a] (muscle)	4600	Pringle *et al.* (1968)
		28-368 (liver)	14720[a]	
Freshwater Clams (6spp.)	5.2-89.6 g/g (sediment) 0.021 (water)	up to 48[b] (body)	-	Anderson (1978)
Freshwater Clams (8 spp.)	(sediment in Mississippi)	11[a]	-	Price and Knight (1978)
Mussel (*Mytillus galloprovincialis*)	0.29-2.15 ng/g particulate lead suspended in water	3.9-9.5[b]	4400-13400	Marletta *et al.* (1979)
6 Invertebrate Species	32.0-565.0	up to 500[b]	1000-9000	Spehar *et al.* (1978)
Brook Trout (*Salvelinus fontinalis*)	119.0	68[b] (liver) 215[b] (kidney)	570 1800	Holcombe *et al.* (1976)
Brook Trout	3.0	4.2[b] (liver)	1400	Pagenkopf and Neuman (1974)
Brook Trout	12.0	56.0[b] (liver) 1.4[b] (muscle)	4600 100	Ray (1978)
Coho Salmon (*Oncorhynchus kisutch*)	0.21 lab	68[b] (gills)	300	Varanasi and Gmur (1978)
		50[b] (kidney)	200	
		10[b] (liver)	50	
Coho Salmon (*Oncorhynchus kisutch*)	150	1.5 (gills) 0.4 (liver)	10 4	Reichert *et al.* (1979)
Fish (*Gillichthys mirabilis*)	38	22[b] (gills)	580	Stenner and Nickless (1975)

[a] Wet weight.

[b] Dry weight.

[c] All values were calculated from given water concentration and tissue levels. Otherwise factor is as reported by the authors.

[d] *Pteronarcys dorsata*, *Hydropsyche betteni*, *Brachycentrus* sp., *Ephemerella* sp. *Physa integra* and *Gammarus pseudolimnaeus*.

total body. Tissue levels generally ranged from two to four orders of magnitude above water concentrations.

Preferential accumulation of lead in certain organs has been reported in various studies. In fish, the liver and kidney usually have higher concentrations than muscle and gill tissue (Leland et al. 1979, Ray 1978, Hardisty et al. 1974). The digestive tract sometimes contains high lead levels (Adams 1975), which implicates diet as an exposure route (Bryan and Uysal 1978). In mollusks, the muscle frequently contains high lead concentrations (Pringle et al. 1968, Gire et al. 1974).

Limited information on biological half-lives indicates that lead is retained in tissue for at least several months (Schulz-Baldes 1974). Half-lives for invertebrate species of 300 days in the soft parts of Mytilus edulis, 170 days in Mesidotal entoman and 50 days in Harmatoe were reported (Kauranen and Jarvenpaa 1972). Reickert and co-workers (1979) observed no increase of lead concentrations in the kidney or liver of the coho salmon during a five-week exposure to clean water following a two-week exposure to lead. A depuration rate of 0.48-0.91 mg/kg/day, however, was measured in the eastern oyster (Pringle et al. 1968), which indicates the presence of an effective excretion mechanism for eliminating the metal.

Tetraalkyllead compounds have been detected in fish tissue from various locations (Mor and Beccaria 1977, Harrison 1977, Chau et al. 1979). Rainbow trout bioaccumulate tetramethyllead as much as three orders of magnitude above water concentrations of the compound (Chau et al. 1979). No direct sources of tetramethyllead to the environment are known; however, certain sediment microorganisms are capable of methylating inorganic lead nitrate or trimethyllead acetate to tetramethyllead, a more mobile and biologically accessible form of lead (Summers and Silver 1978). It is unknown how significant this process is in aquatic sediments or its influence on biotic uptake. Most of the lead in anaerobic sediment is expected to be immobilized as lead sulfide in the presence of sulfur (Rickard and Nriagu 1978); therefore, it is generally unavailable for release into the water column.

Biomagnification in Food Chains

It does not appear that lead concentrations in aquatic biota increase at higher trophic levels (Anderson et al. 1978, Drifmeyer and Odum 1975, Namminga et al. 1974, Mathis and Cummings 1973); rather, the exposure pathway is a more important factor in lead bioaccumulation, as described previously. In fact, secondary consumers often have lower concentrations of lead than lower food chain members (Wong et al. 1978). Phytoplankton and zooplankton have been reported to have high lead concentrations in comparison with other species in the same ecosystem (Namminga et al. 1974). Herbivores and detritivores are likely to accumulate higher concentrations than carnivores (Leland and McNurney 1974). An explanation for preferential uptake by these organisms is the strong association of lead with sediment and organic matter and low levels in the water column.

4.3.2.3 Fate Processes in Terrestrial Environments

Movement in Soil

Lead is found in the following ores: galena (PbS), plattnerite (PbO_2), cerussite ($PbCO_3$), and anglesite ($PbSO_4$). The earth's crust averages approximately 15 µg/g of lead. The chemistry of lead in soils is dominated by adsorption and precipitation reactions, which are functions of the soil texture, cation exchange capacity (CEC), pH and in part, phosphorous content (P).

Several researchers have proposed regression equations to determine the quantity of lead sorbed per gram of soil. For instance, the quantity of lead sorbed as a function of the above mentioned parameters is determined by the following equation (with a correlation coefficient of 0.997):

$$Pb = 34.3 + 0.0774P + 5.358\ pH + 5.337\ CEC$$

where Pb is in µmoles/g soil (Zimdahl and Hassett 1977).

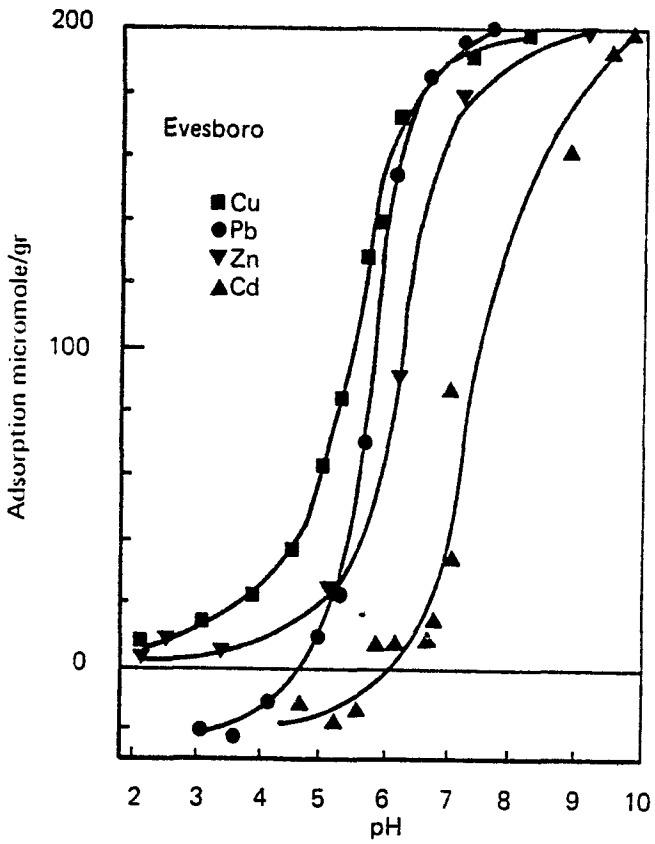
As indicated in Figure 4-5, lead is adsorbed quite strongly by soils, exceeded only by copper. The lead ion is adsorbed above pH 5. Below this pH level, precipitation as PbO_2 and organometallic complexation are the control mechanisms of Pb^{+2} in solution (Zimdahl and Hassett 1977). Lead has a strong tendency to sorb onto soil particulates as indicated from the results of most soil column leaching studies. A literature review by Zimdahl and Hassett (1977) revealed one study in which only 17 µg/g soluble Pb was present in the soil solution three days after application of 2784 µg/g $PbNO_3$. The remaining lead had been sorbed. Another study applied the equivalent of seven years rainfall to a soil column containing 460 µg/g Pb. Only 1.6% of the applied Pb was leachable. Jennett et al. (1977) also found that neutral water was not capable of desorbing lead, or causing Pbs to come into solution. A recent study, Stevenson and Welch (1979), determined the extent of downward migration that occurred within 6.5 years of lead applications ranging from 0 to 3200 kg/ha Pb. Although Figure 4-6 illustrates that the increased levels of Pb can be observed to a depth of about 60 centimeters, the initial lead had been applied to a depth of 15-30 centimeters. Below a 60-centimeter depth, the values approached background levels.

Because lead is readily sorbed onto soil particles, the movement of lead with runoff is likely. Entrainment of soil particles is also a possible route of lead transport.

Terrestrial Biological Pathways

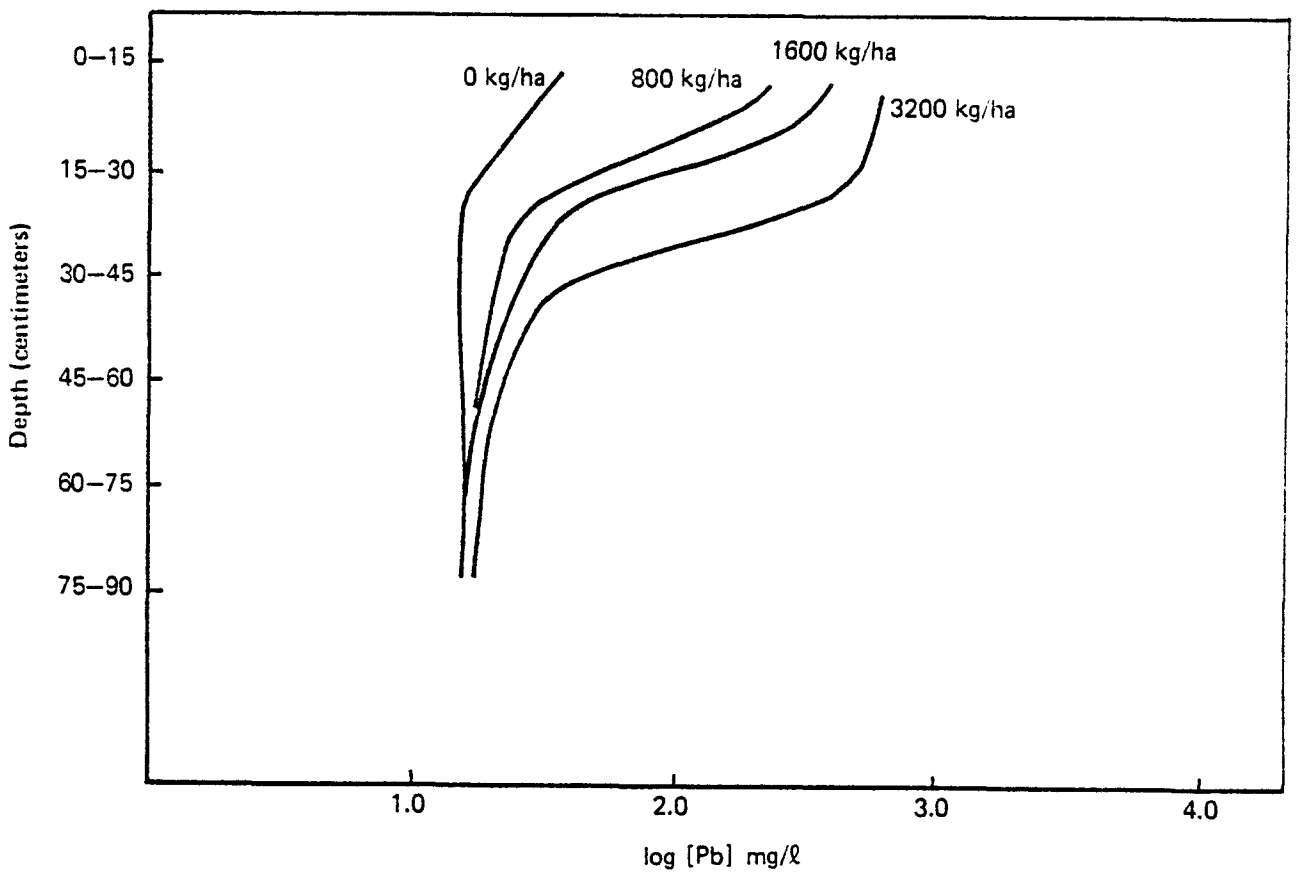
Parameters Affecting Uptake

Plants are exposed to lead by aerial deposition on leaves, stems, or bark, and by root uptake from the soil. Aerosol lead can accumulate



Source: Huang et al. (1977).

FIGURE 4–5 ADSORPTION OF HEAVY METALS ON SOIL MINERALS AND OXIDES



Source: Stevenson and Welch (1979).

FIGURE 4–6 DOWNWARD MOVEMENT OF LEAD IN SOIL

in particulate form on plant foliage. Although there is controversy concerning the extent to which plants can accumulate lead from aerosols, it is generally thought to be minimal (Bell et al. 1978), primarily because of the insolubility of lead (U.S. EPA 1977). It is difficult to obtain data on plant accumulation of lead because of the problems associated with the differentiation between lead deposited on and lead absorbed by the foliar tissue (Bell et al. 1978). Studies of plant uptake near automobile exhaust indicate that leaves have an extremely small likelihood of absorbing both the soluble and particulate forms of lead (Koeppe 1977). The amount of lead that can be washed from leaves has been reported to be 50% with a range of 8-80%. The amount removed depends on the leaf surface; lead retention is higher in plants with a rough or hairy surface (Peterson 1978).

Rooted plants are not considered good sensors of airborne lead levels and attention is being given to the use of bryophytes. Little and Martin (1972) reported that elm and hawthorne leaves could only retain 17% of the total aerial lead fallout. They indicated that 30-90% of the fallout on the foliage usually exists as a superficial coating that can be easily washed off by rain, wind, etc. (Rickard and Nriagu 1978).

The soil environment is an important influence on the amount of lead accumulated. The soil factors that can result in a maximum binding of lead, and thus a reduced plant uptake, include a high cation exchange capacity, pH, organic matter content, and phosphorous. Other factors that influence uptake are soil temperature, calcium availability, soluble silicon, soil texture, and the geological background of the soil. Zimdahl and Koeppe (1977) found that the concentration of lead in oats and alfalfa increased with a decrease in pH and organic matter, and that the addition of phosphorous reduced the uptake of lead. However, the association of lead and organic matter has not always appeared consistent (U.S. EPA 1977a).

Still, it is difficult to determine the actual amount of lead taken up by plant roots because much of the lead is bound on the plant surface as crystalline or amorphous deposits. Appreciable uptake of lead occurs from hydroponic solutions, which do not have complicated binding parameters (Koeppe 1977). Warren (1978) concluded that no simple relationship exists between the lead content of vegetables and the soil in which they are grown and that each vegetable has a characteristic lead accumulation ability. In general, the overall mean plant/soil ratio of lead was 8%. However, not all vegetables growing in lead-contaminated soil will exhibit elevated levels of lead (Peterson 1978).

The amount of lead absorbed by vegetation and moved into above-ground portions depends on the species, soil type, and many environmental factors.

The amount translocated is generally low. The highest lead concentrations are usually associated with the organ that has the greatest surface to volume ratio and the roughest or most pubescent surface, generally the roots (Peterson 1978, Smith 1976). In general, there appears to be a 7- to 10-fold decrease in lead concentration from root to foliage and a similar decrease from foliage to grain (Zimdahl and Koeppe 1977).

Bioconcentration Factors

A few bioconcentration factors (BCF) for different species of vegetation are calculated in Table 4-9. The high degree of variability reflects species differences as well as differences in exposure sources. Yopee and co-workers (1974) have recommended that the maximum permissible level of lead to which crop plants are exposed should not be >2.0 mg/kg when soluble in soil solution.

Numerous studies have investigated lead bioaccumulation in terrestrial invertebrates, such as earthworms (Gish and Christensen 1973, Gullvag 1978) and grasshoppers (Scanlon 1978), and in vertebrates, such as small mammals and birds (Clark 1979, Roberts *et al*. 1978, Haschek *et al*. 1979). Some of the measured tissue concentrations are reported in Section 4.2, Distribution of Lead in the Environment. Unfortunately, it is very difficult to relate environmental media concentrations to the extent of lead uptake in terrestrial animals because of the many exposure pathways possible (e.g., ingestion of contaminated food, inhalation, dermal absorption from soil, etc.). No laboratory studies were found that might have provided insight into exposure levels and bioaccumulation in terrestrial animals.

4.3.3 Major Environmental Pathways

4.3.3.1 Pathway #1 -- Atmospheric Transport

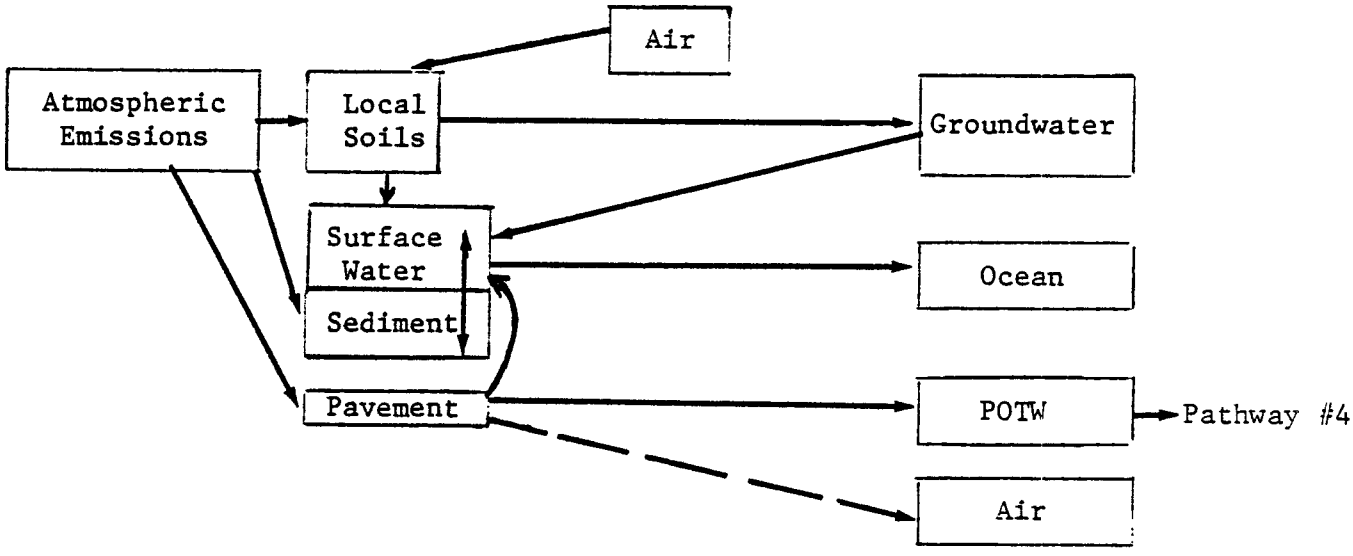


TABLE 4-9. LEAD ACCUMULATION BY VEGETATION

Species	Source	Exposure (mg/kg)	Accumulated (mg/kg)	Calculated BCF	Reference
Field Studies					
Oak roots	Lead smelter	Soil: 6376	2400	.36	Jackson and Watson (1977)
.4 km away		Soil: 208	6.0	.02	
2.0 km away		Soil: 140-190	3.0	.08	
control		Soil: 13.6-25		.08	
Mixed Species					
Fall	Lead smelter	Soil: 52 dry wt	Roots: 208 Unwashed Tops: 326	Roots: 4	Peterson (1978)
Spring	Lead smelter	Soil: 88.6	Roots: 309 Unwashed Tops: 823	Roots: 3.49	Peterson (1978)
Fall	Control	Soil: 13	Roots: 12.7 Tops: 25.8	Roots: .98	Peterson (1978)
Spring	Control	Soil: 18.9	Roots: 22.3 Tops: 37.6	Roots: 1.18	Peterson (1978)
Lettuce	Highway 47,100 car/ 24 hrs 30 ft from road	Air:4.5 μg/m^3	Leaves: 24	Whole plant: 48/138.5= .35	Bell *et al.* (1978)
		Soil: 134	Roots: 24		
Potato	Highway 47,100 car/ 24 hrs 30 ft from road	Air:4.4 μg/m^3	Leaves: 87	Whole plant: 120/138.5= .87	Bell *et al.* (1978)
		Soil: 134	Roots: 33		
Laboratory Studies					
Barley	Laboratory	Soil:800	Roots: 800 dry wt Tops: 3 dry wt	Roots: 1.0	Zimdahl and Koeppe (1977)
Apple leaves	Potted soil & colored news-print	567 (dry) (2nd yr)	7.71 dry wt (5.50 control)	0.01	Elfving *et al.* (1979)
Corn tops	4 weeks in lab soil without optimum pH, CEC & phos-phorous for binding pH	2000 soil	200-400 dry wt	0.1-0.2	Koeppe (1977)
	Hydrophonic for 3 days	100 μg Pb/ml	Roots:10,600 dry wt Tops: 390 dry wt	Roots:106 Tops: 3.9	Koeppe (1977)

Sources

Pathway #1 describes the fate of lead entering the atmosphere as a result of the combustion of gasoline with lead alkyl additives; the storage and distribution of leaded gasoline; mining, milling, and smelting of primary lead ores; zinc and copper smelting; fossil fuel combustion; and iron and steel production. Each of these sources is unique with respect to the chemical and physical form of the emitted lead and the geographical and vertical source distribution. Except for the evaporation of tetraethyl and tetramethyl lead from leaded gasoline, most atmospheric emissions of lead are in the particulate form.

Automotive Emissions -- Antiknock Additives

The lead alkyl gasoline additives, tetraethyllead (TEL) and tetramethyllead (TML), may be released by evaporation during storage or distribution. The release of these additives appears to be the predominant mechanism rather than combustion, since the highest concentrations of organoleads have been reported in samples taken near a busy gas station (0.59 $\mu g/m^3$) and in an underground parking garage (1.8-2.2 $\mu g/m^3$) (U.S. EPA 1977a). Organolead concentrations comprised 10 and 17%, respectively, of the total airborne lead. Laveskog (1971) also observed small amounts of vapor phase tetraalkyllead compounds in automobile exhaust, where they are likely to be quickly adsorbed onto exhaust particulates (Harrison and Laxen 1978). In urban air, organoleads typically comprise 1-4% of the total airborne lead (Harrison and Perry 1977).

Harrison and Laxen (1978) recently investigated the chemistry of TEL and TML in ambient air. They concluded that like the atmospheric chemistry of other hydrocarbons, homogeneous gas phase reactions predominate in the breakdown of these compounds. Their results indicate that daytime decay rates for TML range from 3-29%/hr, and for TEL, 17-93%/hr. The stable lead compounds formed by these decay processes have not been identified.

Automobile Exhaust

The U.S. EPA (1977a) has effectively summarized data on the particulate sizes associated with exhaust from leaded gasoline combustion in automobiles. Under cruise conditions, automobile exhaust contains lead in extremely small aerosol sizes. The reported mass median diameter is generally 0.5 μm or less. Chamberlain et al. (1975) report even smaller sizes (0.015 μm). Particles <0.1 μm rapidly coagulate via Brownian motion to form particles 0.1-0.5 μm in diameter. On the other hand, during cold start or acceleration, the particle sizes are considerably larger, with mass median diameters in the range of 2-10 μm. Haibibi (1973) and Ter Haar et al. (1972) found that the mass median diameter increased with vehicle mileage. Ter Haar and co-workers (1972) estimated that approximately 35% of the Pb burned over the lifetime of

a car is emitted as fine particulate (<0.5 m) and 40% is emitted as particulates (>0.5 m). Thus, actually only 75% of the lead burned is released to the atmosphere.

The predominant chemical form of lead in automobile exhaust is PbBrCl. During acceleration, with hotter exhaust conditions, the PbBrCl deposited in the exhaust system is released either by scouring or vaporization.

Biggens and Harrison (1980) hypothesize that the reaction of PbBrCl leads to the release of HBr and HCl in acid sulfate droplets (H_2SO_4 and NH_4HSO_4). This mechanism may account for the observed decrease in Br/Pb ratios in aged urban aerosols, and may produce relatively insoluble $PbSO_4$, which is a significant component of urban lead aerosols that has been shown to increase in concentration in aged exhaust aerosols (Ter Haar and Bayard 1971) and road dusts (Biggins and Harrison 1980, Olson and Skogerboe 1975).

In rural environments affected by automotive exhaust, but presumably exposed to lower concentrations of SO_2, sulfates, and photochemical precursors, Ter Haar and Bayard (1971) observed that the predominant lead compounds were PbBrCl, $PbCO_3$, PbO_x, and $(PbO)_2PbCO_3$, with lesser contributions from $PbCl_2$ and $PbSO_4$.

Wet/Dry Deposition

Automobile exhaust is emitted very close to the roadway, which is an important factor in the substantial dry deposition near the roadway. Large particles (> 20 μm) are expected to be deposited on the roadway or within a few meters. Smaller particulates will also experience large deposition rates by impaction near the roadway as a result of their high groundlevel concentrations. Nonetheless, a significant fraction, probably on the order of 20-50% of the emitted lead, is associated with fine particulates that are carried as aerosols away from the source and comprise the bulk of the urban lead aerosol observed at greater distances (< 100 meters) from heavily travelled roads (U.S. EPA 1977a).

The significant fraction (probably 20-50%) of automotive lead that is transported from the immediate source is expected to be deposited, possibly at great distances from the source, by precipitation. The long-term impacts may be significant, particularly in mountainous regions that receive large rainfall. These areas scavenge effectively both water and lead from the air.

In the northeast, this rain is likely to be acidic (ph <5.6), which would enhance the solubility of Pb by exacerbating the impacts on surface waters. For example, Hirao and Patterson (1974) found that nearly all of the lead entering an isolated High Sierra mountain watershed was of anthropogenic origin, which was dominated by emissions from combustion of leaded gasoline. This lead was delivered to the watershed via snowfall (92%) and dry deposition on foliage (8%). Ninety-eight percent of the lead entering the watershed each year is held there and is primarily

associated with organic matter in the uppermost soil horizons. The reported deposition rate (0.9 $mg/m^2/yr$) is one of the lowest observed worldwide (Nriagu 1978a). Thus, at great distances from sources of automotive lead, wet deposition is the predominant air-to-surface pathway.

This is not to suggest that washout of automotive lead near roadways is not important. Indeed, washout rates near roadways are much greater than those observed in the High Sierra: Atkins & Kruger (1968) observed wet deposition rates of 40 $mg/m^2/yr$ in Palo Alto. However, washout was only 24% of the total deposition in Palo Alto.

Deposition on Soils

Once lead is deposited, it is held strongly in the soil, usually in the upper 1-5 centimeters. The understanding of the chemical forms and fate of lead in soils is limited because of insufficient analytical data. Olson and Skogerboe (1975) found that 50-70% of the lead in roadside soil samples existed as $PbSO_4$. Other compounds identified included $PbO \cdot PbSO_4$, lead oxides, metallic lead, and PbS. The conversion of PbBrCl to $PbSO_4$ may occur either in the atmosphere prior to deposition, or in the soil. Biggins and Harrison (1980) essentially confirmed these findings, identifying also $PbSO_4 \cdot (NH_4)_2$, Pb_3O_4, and $2PbCO_3 \cdot Pb(OH)_2$. Elemental lead was associated with sites where vehicle cold-starts occur. $PbSO_4$ is one of the more soluble of these compounds, although all are relatively insoluble at typical soil pHs. Lead is generally associated with organic matter in the soil (Siccama et al. 1980). All studies find that lead is strongly associated with the solid phase in the soil environment.

This fact, combined with the observed concentration of lead in the upper soil horizons, implies that erosion and washoff of soil particles by surface runoff is the major pathway for surface water contamination by lead deposited from the atmosphere. Bertine and Mendeck (1978) studied lake sediments in the vicinity of New Haven, CT in order to examine contamination of surface waters by lead in urban areas, where the primary lead inputs are from automotive exhaust. In this particular watershed, lead contamination from the late 1800s to 1920 was predominantly related to coal consumption; after 1920, automotive lead was the predominant input. Kleinsman and co-workers (1977) analyzed Pb fallout and runoff in New York City and suggested that automotive sources contribute a major fraction of the lead input to the New York harbor.

Dry deposition of lead may also occur on vegetative surfaces. The lead on these surfaces is generally easily washed off; and it is unlikely that significant plant uptake occurs.

Smelters, Coal and Oil Combustion and Iron and Steel Production

Lead emissions from coal and oil combustion, smelters, and iron and steel production are similar in that most of these emissions are

released from elevated point sources. In general, baghouses or electrostatic precipitators control particulate emissions from smelters and many fossil-fuel utilities. These precipitators release only the smaller particulates. Furthermore, because of the condensation process that forms lead particulates at high temperatures, lead tends to be concentrated in the finer particulates, which are generally <1 μm. Consequently, these particulates are widely dispersed and are deposited predominantly by precipitation.

The chemical characterization of these sources are expected to differ, although the precise chemical characterization of lead containing aerosols from these sources is unknown. Corrin and Natusch (1977) cite unpublished studies that show PbS, $PbSO_4$, and elemental lead as the predominant lead species in smelter baghouse exhaust. Nriagu (1978b) indicates all of the above lead species, plus $PbCO_3$, $PbO \cdot PbSO_4$, $PbO \cdot PbSO_4$, Pb included in other metal oxides, and lead silicates as possible chemical forms in smelter exhaust.

Apparently, lead in particulates is preferentially adsorbed onto the surface of the smaller silicate particles or included in soot particles (Corrin and Natusch 1977). Nriagu (1978b) also suggests the presence of PbO_x, $PbSO_4$, $Pb(NO_3)_2$, and $PbO \cdot PbSO_4$.

Lead from smelters or fossil-fired utilities is likely to be quite insoluble, although the lead sulfates, which may be a major component of smelter exhaust, are relatively more soluble than the other forms.

Deposition on Soils

The effects of smelter emissions have been intensively studied. Jennett et al. (1977) performed a study of a forest ecosystem contaminated by lead emissions in the New Lead Belt of southeastern Missouri. Smelter emissions were found to result in significant deposition of lead at least as far as two miles from the smelter. Basically, fugitive dusts generated during ore transport and handling are the predominant cause of extremely high deposition rates within 1000 feet of the smelter. Almost one-half the particulate lead in the smelter stack emissions is associated with particles <1 μm diameter. On the other hand, fugitive dusts show a mass median diameter of about 7 μm, with 40% >10 μm.

Deposited lead is concentrated in the upper one inch of the soil profile where the surface is covered by leaf litter. In locations without surface leaf litter, several inches of the soil profile were contaminated. Exposed plant parts (grasses, tree leaves, lettuce, etc.) are seriously contaminated by lead, while roots and tissues are relatively uncontaminated. This tendency of lead to be associated with surface vegetation and leaf litter (as observed in roadside environments) causes contamination of surface waters by runoff and soil erosion. In stormwater analyses, Jennett and co-workers (1977) demonstrated surface water contamination by showing relatively constant low levels of dissolved lead throughout two storms, while levels of suspended lead increased

dramatically in phase with stormwater flow and suspended solids content. These sediments are subsequently deposited in man-made lakes, which drain the contaminated watershed (Jennett et al. 1977).

The Walker Branch watershed of Oak Ridge, TN is predominantly affected by coal-fired power plants. Studies by Andren et al. (1975) are apparently the only observations of lead deposition that focus on coal-derived lead. The observed deposition rates of 60 $mg/m^2/yr$ are typical of those reported universally and summarized by Nriagu (1978b). As expected for emissions from a coal-fired utility with tall stacks, ~67% of the lead deposition was by wet deposition.

Mining and Milling of Lead Ores

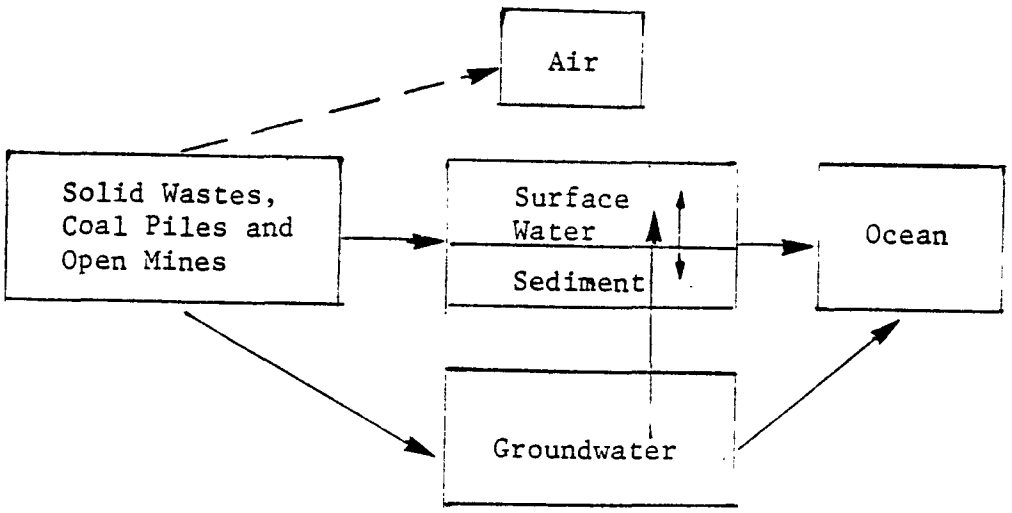
Mining and milling of lead ores create fugitive dusts comprised primarily of PbS, but also containing $PbCO_3$, $PbSO_4$ $Pb_5(PO_4)_3Cl$, lead oxides, and lead silicates. The particle sizes are large and the sources are at ground level. Under these conditions, one would expect extremely localized concentrations and depositions, based on Jennett and co-workers' (1977) observations in the vicinity of mines, mills, and on haul roads. The PbS particulates are insoluble and will be subject to runoff during storms. Foraging species may become significantly contaminated (Jennett et al. 1977).

Summary

The principle source of atmospheric lead emissions is from automobile use. The fate of lead depends on the size distribution of the lead particulates, which, in turn, is a function of the operating conditions of the car. Cold starts, and stop and go traffic, promote large particle sizes and immediate deposition; cruise conditions form smaller lead aerosols, which are subject to wider dispersion. The height of lead release causes the greatest impact near the road. A significant 20-50% remains airborne, however. Lead emissions from mining and smelting operations cause localized high concentrations of lead. Smelters and other sources, which have higher stacks, allow for greater dispersion of lead releases.

The deposited lead forms are largely insoluble. Lead accumulates in the top few inches of leaf litter and soil. Runoff and erosion introduce lead into surface waters as a suspended solid, which result in accumulation of lead in the sediment.

4.3.3.2 Pathway #2 -- Solid Wastes, Tailings, and Municipal Landfills



Sources

In general, solid wastes containing lead are created by mineral ore processing, iron and steel production, copper and zinc smelting, and the production and use of various lead-containing products. Pathway #2 describes the fate of lead in the environment as a result of these sources. The solid wastes result from the overburden of mining and low-grade portions of mineral ore deposits. Tailings, which are highly concentrated in minerals, are produced as a final waste product of mineral concentrating operations. Municipal waste landfills accumulate objects containing lead, such as objects covered with lead-containing pigments, or solder. Although lead batteries are the most obvious sources of lead to municipal landfills, they are not a major contributor to lead concentrations in municipal dumps, because car batteries are generally recycled.

Mining of lead has been principally restricted to the state of Missouri. The Old Lead Belt was found in the southeast portion of the state and is currently inactive. The New Lead Belt (also referred to as the Viburnum Trend), in the southwest portion of the state, has been first in world lead production since 1970 (Wixson 1978). Since 1873, lead mining has resulted in 1550 million metric tons of tailings, and 3875 million metric tons of combined tailings and waste (Martin and Mills 1976). Disposal of these wastes in the 19th and early 20th centuries was without regard to environmental considerations, and thus erosion and weathering contributed to adverse environmental impacts. Currently, tailings are used to create dams for the establishment of tailing ponds.

The lead species in solid wastes and tailings depend on the nature of the ore. Galena (PbS) is most commonly mined and is the most frequently encountered form of lead in mine tailings. Other lead species include PbO, $PbCO_3$ and $PbSO_4$ (Zimdahl and Hassett 1977, Jennett 1979).

Acid Mine Drainage

Tailings and solid wastes from mineral mining aid in the formation of mineralized acid drainage. The impact of acid mine drainage to local surface waters largely depends on the alkalinity, or buffering capacity, of the waters upstream or downstream of the point of discharge. The streams channelling the Old and New Lead Belts are quite buffered and of a neutral to slightly basic pH (Zimdahl and Hassett 1977, Wixson 1978). Thus, rarely has lead in tailing effluents been a problem in local streams. (Zimdahl and Hassett 1977). Most of the lead entering the streams is associated with suspended solids in the form of PbS, $PBCO_3$, PbO and $PbSO_4$.

Fate Processes in Streams

Little evidence substantiates that surface mineralization in the Lead Belts of Missouri has elevated levels of lead in the surface waters. As stated earlier, most of the lead in the water column is in the undissolved, particulate form. Lead in the Old Lead Belt enters the streams principally via runoff and leaching through tailings (Wixson 1978). In the New Lead Belt region, the velocity of the streams is high enough to prevent the suspended particulates from settling out.

Eventually, the streams discharge into Clearwater Lake, which is about 30 miles from the last few mine discharges, and is dammed at one end. The lead concentrations in Clearwater Lake sediments range from <3 mg/kg at the point of stream entry to 60 mg/kg near the dam (Zimdahl and Hassett 1977). Lakes with long narrow arms, such as Clearwater, experience scour and washout during storms. The sediments are therefore transported to the base of the dam. In this specific lake, the lake sediments act as a temporary sink for heavy metals. Opening the dam during periods of high in-flow, effectively removes the metals from the lake sediments, thereby accounting for the low levels of lead found in Clearwater Lake sediments (Wixson 1978, Zimdahl and Hassett 1977).

Coggins and co-workers (1979) investigated the drinking water quality of the reservoir in the area engaged in lead and silver mining. Lead concentrations were in the range 50-69 μg/l in the water column. Lead in the sediments averaged 150 mg/kg (ranging from 30-650 mg/kg), which was principally adsorbed onto the clay fraction of the sediments. Other metals were also associated with iron and manganese hydrous oxides, or complexed with organic matter; for the most part, lead was not.

Groundwater Contamination from Tailings and Mining Activity

Mink (1972) investigated the groundwater of the Coeur d'Alene mining area in Idaho. Evidence indicated that the effluent of a tailings pond was not responsible for the high levels of lead found in the groundwater. The aquifer pollution was a result of contact with tailings from old abandoned mining operations. Solubilization of the heavy metals in the tailings by the high water table resulted in concentrations of 6% lead in the upper aquifer.

Fly Ash Ponds

Disposal of fly ash generated from coal combustion has created concerns about the leaching potential of metals associated with the fly ash. Theis et al. (1978) studied the extent of groundwater contaminations from a fly ash pond. Analysis of the fly ash indicated that Pb was the third most abundant heavy metal (after arsenic and zinc) in the fly ash. In the groundwater leachate, lead was generally found in the lowest concentration of the metals tested, at about 40 μg/l, 100 feet from the pond. Precipitation as the hydroxide and carbonate, rather than sorption on the iron and manganese oxides controlled lead concentrations (Theis et al. 1978). The degree of precipitation is a function of pH. Theis et al. (1978) show that abrupt precipitation of lead results at an approximate pH of 6.5, thereby reducing the concentration of soluble Pb from $10^{-4.5}$ moles/l at pH 6 to 10^{-6} moles/l at pH 7.

Municipal Landfills

Roulier (1975) conducted a study using soil from two sites to examine municipal solid wastes disposal, and subsequent metal leaching. At one site, conditions favored metal attenuation, and, at the other landfill, a "worst case" scenario was simulated.

The first site collected leachate from 1.390 kilograms of municipal refuse under anaerobic conditions in a soil column. The concentration of lead was below the detection limit of 0.5 mg/l and fell into the category of "least generally mobile."

The second study used soil columns packed with quartz sand and pure clay, and pure leachate was slowly passed from municipal landfills through the columns. Under these conditions, 99.8% of the lead was attenuated by the soils, exceeding zinc, cadmium and mercury. The controlling mechanism as in the fly ash study was precipitation at neutral to alkaline pH ranges, and sorption to clays.

Data from other landfills show lead concentrations ranging from 0.1-2 mg/l, with less than 0.1 mg/l as a typical value (U.S. EPA 1977b). Another study of 12 landfills ranging in age from 0.25 to 16 years showed a mean lead concentration in the leachate of 0.92 mg/l (range 0.1-3.2 mg/l) (Chian and DeWalle 1977); this is slightly under an order of magnitude greater than the above study.

Lead in Soils from House Paint

Although lead from house paints contributes to localized high levels of lead in the soils immediately surrounding a lead-painted house, on the whole, this is not a significant source of lead to the land. Ranges of 1000-2000 mg/kg in the soil have been reported (Bogden and Louria 1975). Even though the concentration of lead in house paints is now controlled, older wooden houses may still be covered with leaded paint. Natural weathering and sanding and scraping houses prior to repainting are ways

in which lead is transferred to the soil. Bogden and Louria (1975) analyzed the soil lead concentrations 15 and 30 meters away from a repainted house, at the time of scraping the leaded paint, and one year later. The lead concentration in the paint chips was 17%. The resultant soil concentrations are shown below:

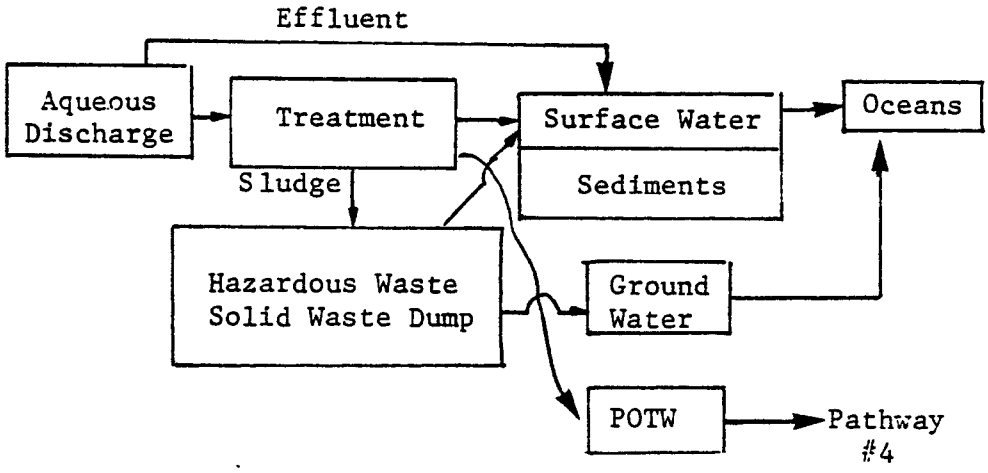
Lead Content at Various Distances from the House

Time Period	Concentration (mg/kg) 15 meters	30 meters
August 1973	185	165
August 1974	440	490
Control Soils	130-220	

Summary

Thus far, the surface waters of the New Lead Belt do not show elevated levels of lead. It is believed that the highly buffered, neutral-to-basic nature of the streams channelling the New Lead belt prevents the lead from solubilizing or settling out. Frequent opening of the Clearwater Lake dam scours lead that settles onto the lake sediments. Groundwater contamination from tailing ponds and fly ash ponds does not appear to be a significant source of lead, although it has occurred in some cases. Lead, in groundwater leachate is controlled by precipitation as the hydroxide or carbonate, provided that the pH is not acid. Municipal waste leachate studies categorize lead in the least generally mobile metal group, where adsorption to clays and precipitation control its movement.

4.3.3.3 Pathway #3 -- Aqueous Industrial Discharge



Sources

Sources of aqueous discharges of lead to the environment include lead production, coal mining, and industries such as lead battery manufacture, paper and pulp and nonferrous metals production. Pathway #3 describes the fate of lead as a result of aqueous industrial discharge. The lead battery industry is a major user of secondary lead and recycles the wastewater to recover additional lead. Wastewater treatment, as exercised by the lead battery industry, consists of a settling tank, neutralization unit, in which lime, ammonia, or sodium hydroxide is added to increase the pH; a scrubbing unit, which removes dissolved carbon dioxide by aeration; and, finally, another settling tank, to which coagulants are added to flocculate and precipitate the suspended materials. Eventually, the sludge from both settling tanks is collected, dewatered, and processed to recover the high lead content. The effluent from the second settling tank is discharged to local surface waters or POTWs (Chloride Overseas 1971). Fochtman and Hass (1972) found that the lead content of several lead battery effluents ranged from 0.5 to 3.0 mg/l dissolved lead, and from 5 to 35 mg/l suspended lead.

The paper and pulp industry also discharges lead to water. This discharge is principally a result of the lead-containing pigments on the paper. Concentrations of lead in paper coating and glazing effluents range from 0.05 to 1000 mg/l. After treatment and dilution, the average lead concentration in the final effluent of 17 paper and pulp mills was 16 μg/l (U.S. EPA 1979).

Distribution to Surface Waters

Mathis and Cummings (1973) and McIntosh and Bishop (1976) have studied the distribution of lead discharged into surface waters. The Mathis and Cummings (1973) study used the Illinois River as its environmental system. The river is known to receive both municipal and industrial discharges. When compared with nonindustrial use (rural) rivers, the average lead concentrations in the sediments of the Illinois River (28 mg/kg) was 1.6 times that of the rural streams (17 mg/kg). The water column contained an average of 0.002 mg/l lead.

McIntosh and Bishop (1976) researched the level of pollution in a small eutrophic lake, which was the receptor of direct industrial discharges primarily from metal plating and urban runoff. The metal plating was discontinued in 1974; McIntosh and Bishop (1976) examined the changes in the lake in the tabulation of the data below. These values represent averages, and thus the columns do not necessarily add:

	Lead concentration (μg/l)	
Water Column	1974	1975
Dissolved	24	9.4
Suspended	15.2	6.6
Total	28.6	14.4

Of the 54 water samples collected from Little Center Lake, dissolved lead was detected (limit = 2.0 ug/l) in only 50% of the samples and suspended lead in only 18% of the samples. The lead, however, is quite evident in the lake sediments. The top ten centimeters contained between 450-500 mg/kg Pb dry weight. The highest concentration of lead in the lake sediments was near the street sewer outfall, from which suspended lead quickly settled out.

Sludge Disposal

The sludge generated by industrial effluent treatment is normally disposed of in a solid or hazardous waste dump, or treated to recover the lead, as in the battery industry. A properly designed landfill site should prevent significant translocation of lead, which has a tendency to precipitate in an insoluble state. Groundwater contamination is not likely in a landfill or settling pond as the evidence in Pathway #2 suggests.

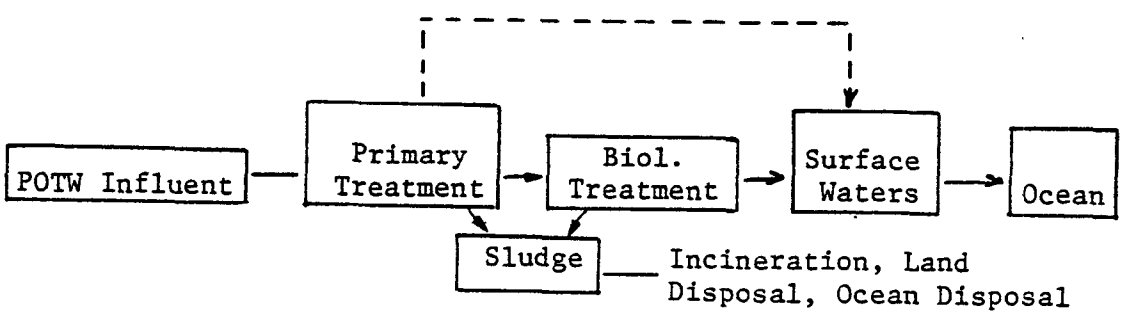
Ultimate Sinks

The sediments are the major sinks for lead discharged with industrial effluent into surface waters. Treated industrial effluents generate a waste sludge into which most of the lead partitions. Disposal of the sludge at a hazardous or municipal landfill will generally prevent further translocation of lead in the environment, with the exception of runoff.

Summary

The manufacture of lead batteries could conceivably result in large quantities of lead discharged into the environment. However, waste effluent treatment practices, which have been effective since the early 1970s, have allowed little lead to be discharged via this route. Studies of stream and lake systems receiving industrial discharge indicate that the sediments concentrate most of the lead. Waste effluents treated prior to discharge generate sludges that are disposed of in a hazardous waste site. Mobilization is not generally expected to occur from this site.

4.3.3.4 Pathway #4 -- Publicly-owned Treatment Works



Treatment Scheme

Pathway #4 describes the fate of lead in wastewaters that are introduced into a publicly-owned treatment work (POTW). The inflow to the POTW may be a combination of industrial and commercial effluents, domestic wastes, and surface runoff. Consequently, the nature of the influent is quite varied; however, typical influent lead concentrations will be ~0.1 mg/l (Arthur D. Little, Inc. 1979).

The degree to which lead is removed from the raw wastewaters, and thus the concentration of lead in the discharged wastewaters, depends on the type of treatment involved. The U.S. EPA (1977c) provides a summary of data from 269 municipal treatment plants in the United States that use various treatment methods. The data for lead are summarized in Table 4-10.

Lead partitions into the sludge during treatment. Oliver and Cosgrove (1974) found that lead immediately precipitates when introduced to sewage. The metal exceeds others in this capacity.

Sludge Disposal

Sludge disposed onto land may go to a sanitary landfill, or be spread for the purpose of amending the soil. The form of lead in sludge is not exactly known. Sommers and co-workers (1976) found that lead sulfides, phosphates, and hydroxides were not detected in sludges containing relatively high concentrations of lead.

Sludge routed to municipal landfills was described in the section on Pathway #2. Sludge that is incinerated will contribute to the concentrations of lead in the atmosphere. The fate processes will be similar to those described in the discussion on Pathway #1.

Surface Water Discharge

The behavior of lead discharged with POTW effluents into local surface waters will be similar to that already described for aqueous pathways (Pathway #3). Morel and co-workers (1975) have provided a detailed study of the fate of lead discharged by the Joint Water Pollution Control Project (JWPCP) of the Los Angeles County Sanitation District; these discharges may be generally representative of POTW discharges to the ocean.[1] Three equilibrium models were applied to the sewage to determine the speciation of heavy metals present in the sewage. Lead was present entirely as PbS in all models regardless of the input of organic-complexing agents or adsorption surfaces.

Although lead was found in the fairly insoluble sulfide form in the effluent (370 mgd effluent, discharged through submarine outfalls at a

[1]The wastes, containing both domestic and industrial wastes, contain high levels of lead (250 mg/l) after primary treatment.

TABLE 4-10. EFFLUENT DATA FROM U.S. MUNICIPAL TREATMENT PLANTS USING VARIOUS TREATMENT METHODS

Type of Treatment	Effluent Data (Means)	
	% Removal of Pb	Pb Concentration (mg/l)
Primary	24	.136
Biological (all types)	38	.092
Activated sludge	39	.067
Trickling filter	37	.116
Biological with chemical addition	39	not available
Tertiary	44	not available

Source: U.S. EPA (1977c).

depth of 60 meters), the studies indicated that the combined processes of dilution and oxidation resulted in substantial solubilization of lead (as well as other metals); this increases the residence time of lead in the water and allows it to be transported greater distances.

Summary

The concentration of lead in POTW effluent, and the effectiveness of its removal depends upon the initial influent concentration and type of treatment enacted. Most of the lead partitions into the sludge during treatment. Discharge to marine systems causes solubilization of lead, which prohibits "hot-spots" because of dilution. In freshwaters, lead is expected to largely partition into the sediments.

4.4 OVERVIEW

4.4.1 Atmospheric Levels and Pathways

The atmosphere receives the greatest portion (53%) of the total environmental releases of lead; approximately 93% of these releases are a result of automobile emissions. Transport of lead in the atmosphere depends on particle size, chemical form, and the distribution and height of the release. Particles larger than 20 μm are rapidly deposited. Although automobile exhaust contains lead in extremely small aerosol sizes, with a mass median diameter of 0.5 μm or less, large particles may be released during cold start or acceleration. These larger particles are deposited onto the roadway, or within a few meters. Smaller particles also are deposited, to some extent, near the roadway by impaction; however, some fraction is carried a distance (<100 m) from heavily travelled roads and may be deposited by washout. Still, precipitation may carry some of the particles a considerable distance; thus, more remote areas may be contaminated.

Lead releases of smelter, fossil fuel combustion, and iron and steel production are primarily from elevated point sources and are generally less than 1 μm in size. Consequently, these particulates are widely dispersed and are primarily deposited by precipitation. Smelter emissions result in significant deposition of lead at distances up to two miles from the smelter. Fugitive dusts account for higher deposition near the source. Airborne releases from mining and milling operations will largely be in the form of fugitive dusts, resulting in localized deposition.

The deposited lead forms from all atmospheric sources are largely insoluble. Lead accumulates in plants, leaf litter, and soil. Runoff and erosion introduce lead into surface waters as a suspended solid, which results in sediment concentration.

The monitoring data for lead generally reflect these fate pathways. The range of lead levels in air for remote areas of the continental United States is 0.0001-0.01 $\mu g/m^3$. In contrast, urban areas show levels of

0.5-10 $\mu g/m^3$, which are considerably higher, primarily because of automobile use. The monitoring data show rapid deposition; and it has been estimated that lead levels in air generally decrease by about 50% between 10 and 50 meters from the highway.

Elevated atmospheric lead levels are also found in the vicinity of point sources, such as smelters, in the range of 0.4-4 $\mu g/m^3$. Considerably higher levels have been reported, however.

4.4.2 Aquatic Levels and Pathways

Direct sources of lead to water are largely unidentified; however, they include such sources as iron and steel production, lead production, and coal mining. Still, indirect sources, such as urban runoff and atmospheric deposition, are significant sources to aquatic systems.

Lead reaching surface waters is likely to be strongly sorbed onto suspended solids and sediments. Because lead in the sediment is strongly sorbed, it is unlikely to be desorbed as a result of a physical disturbance. Changes in the water chemistry, for example, pH, could result in an increased solubilization of lead. Lead found in the water columns is expected to be strongly complexed by organic molecules. Lead can bioconcentrate in aquatic organisms up to 2-4 orders of magnitude above water concentrations. It appears to be fairly persistent in aquatic biota, with a lifetime of at least several months. Little evidence suggests biomagnification of lead in aquatic food chains.

Typical levels of lead in U.S. waters are less than 25 $\mu g/l$. Levels of lead in seawater are considerably lower, on the order of 0.005 $\mu g/l$. Lead concentrations in surface waters were higher in urban areas than in rural areas.

Sediments contain considerably higher levels of lead than surface waters. Coastal lead sediment contains approximately 100 mg/kg, while the average lead in river sediments was estimated to be about 20 mg/kg. Higher concentrations were found in STORET data, with mean concentrations ranging from 27 to 267 mg/kg, during 1973-79.

4.4.3 Terrestrial Levels and Pathways

A large amount of lead (47%) enters the environment annually in the form of solid waste, primarily from lead production and the disposal of lead-containing products. In addition, a large amount of lead reaches the soil as a result of atmospheric deposition.

Lead transported to soil is quite strongly sorbed, and, under most conditions, is not subject to leaching. The movement of lead with the erosion of soil particles is likely, however. Entrainment of soil particles is also a possible route of lead transport. In addition, uptake of lead into plants can occur, although only a small portion of

the total lead in any soil is available for plant uptake. Bioconcentration factors are generally less than 1, although they may be slightly higher in the roots.

Because of the characteristics of the New Lead Belt, the production of lead in that area has apparently not resulted in elevated levels of lead from tailings or solid wastes. The waters in the area are highly buffered and neutral to basic. In addition, control of a dam in the area prevents lead accumulation in the sediment of the immediate area. Groundwater contamination from tailing ponds and fly ash ponds does not appear to be significant for lead, although it has occurred in some cases.

Lead is common in municipal waste, because it is found in numerous domestic and commercial products. However, its movement is controlled by adsorption to clay and precipitation.

Monitoring data for lead in soils largely reflect airborne deposition patterns. An average concentration for lead in U.S. soils appears to be about 20 mg/kg in uncontaminated soils. Elevated concentrations are found in the vicinity of highways (up to 7600 mg/kg), and, in general, in urban areas where a range of 100-800 mg/kg lead in soil is found. Elevated concentrations are also found in the vicinity of smelters (up to about 8000 mg/kg), in the vicinity of houses that at one time were painted with lead-containing paints, and in old orchard soils. Urban dust is also found to contain high levels of lead. Concentrations of about 1000-1600 mg/kg are found in urban residential areas and 1400-2400 mg/kg in commercial areas.

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5.0 HUMAN EFFECTS AND EXPOSURE

5.1 HUMAN TOXICITY

5.1.1 Introduction

The human health effects of lead have been extensively and intensively investigated; thus, several comprehensive reviews of lead toxicosis are available (WHO 1977, U.S. EPA 1977, Nriagu 1978). None of the literature, however, has demonstrated any natural function of lead in the body nor any beneficial effects. However, its pervasive nature in the environment makes some degree of exposure inevitable. This chapter is focused primarily on toxicity data that are most useful in the assessment of human exposure to lead and the consequences of such exposure.

The heme-hematopoietic system, the kidney, and the nervous system are the three organ systems that are major targets of lead toxicosis. Subtle neuro-behavioral impairments, such as difficulty in task performance, have also recently been reported at blood lead levels at which no other symptoms of lead toxicity are seen. These findings have resulted in some uncertainty in the established maximal permissible daily intake level for lead. In 1972, a joint FAO/WHO committee established a provisional tolerable weekly intake of 3 milligrams for adults (Mahaffey 1977); a value of 0.3 milligrams lead is currently considered the maximum daily (2.1 mg weekly) permissible intake from all sources for children (King 1971). Lead concentrations of 80 μg/100 ml of blood or 200 μg/l of urine traditionally have served as biologic threshold limit values in determining the "safe" levels of occupational exposure.

5.1.2 Metabolism and Bioaccumulation

The absorption and metabolism of lead depend on a number of factors, such as particle size, physicochemical form of lead, route and extent of exposure, as well as several other factors, such as age and nutritional status.

5.1.2.1 Absorption

Absorption of lead can occur through three exposure routes: oral, inhalation, and dermal. In adults, uptake of lead from the gastrointestinal tract ranges from 5 to 10% of intake (Kehoe 1961, Rabinowitz *et al*. 1974). *In vitro* studies with rats indicate lead crosses the intestinal barrier by a passive diffusion process linked to some degree to the concomitant movement of water (Coleman *et al*. 1978). Kehoe (1961) conducted an extensive series of balance studies in human volunteers for periods ranging from several months to nine years. He found that intake of a total of 0.6 mg/lead/day for one year resulted in a barely detectable increase in the lead content of urine and no demonstrable increase in

blood lead concentration. A total dose of 1.3 mg/lead/day, however, resulted in a progressive increase in urinary excretion of lead and in the lead content of blood and other tissues. Goyer and Mushak (1977) estimate that every 100 milligrams of dietary lead contributes about 10(6.4-15) milligrams of blood lead per 100 milliliters in adults.

More recently, attention has been focused on the increased absorption of dietary lead by infants and young children (Alexander et al. 1973, Roels et al. 1978, McCabe 1979). Alexander and co-workers (1973) reported increased absorption of lead (53%) from the gastrointestinal tract in eight normal children between the ages of 3 months and 8 years, although they examined a small amount of children. Similarly high absorption rates have been noted in young experimental animals (Rosen and Sorell 1978, Momcilovic 1978, 1979).

Animal studies have also revealed several dietary factors that can influence gastrointestinal absorption. Two critical nutritional requirements for a growing mammal are the supply of calcium for bone growth and the supply of iron for red blood cell production. A reduction of either calcium or iron in the diet can result in an enhanced absorption of lead (Granick et al. 1978, Six and Goyer 1972). Iron deficiency in infants may also result in pica and, thus, predispose children to increased lead exposure. Other factors, such as high intake of dietary fat, deficiencies of certain vitamins and minerals, ethanol ingestion, stress, strain of animal, etc., can affect absorption and the resulting toxicity of lead in experimental animals (Barton and Conrad 1978, Bushnell et al. 1979, Levander 1979, McCabe 1979, Mykkanen et al. 1980).

Studies in humans indicate that 30$\pm$10% of lead inhaled is deposited in the lungs (WHO 1977, Tsuchiya 1979) and that the amount of lead retained will vary considerably depending on the size of the particle and the depth and frequency of respiration. There is no evidence that lead accumulates in the lungs or that all lead retained is eventually absorbed or transferred to the gastrointestinal tract (Tsuchiya 1979). An indirect but useful measure of lead deposition and retention in the airways can be made from blood lead levels. A general consensus is that an air lead level of 1 μg Pb/m^3 will increase blood lead concentrations in adults by approximately 1 μg Pb/100 ml whole blood (Mahaffey 1977, WHO 1977). Such a relationship has not been defined for children.

Absorption through the skin is only important in the case of organic compounds of lead; because of their lipid-solubility, organic lead compounds penetrate skin to a significant degree (Barry 1978). Inorganic lead salts do not readily penetrate intact skin. In an experiment that involved the application of lead acetate to the foreheads of eight male volunteers, Moore and co-workers (1980) discovered that between 0 and 0.3% of the dose was absorbed.

5.1.2.2 Distribution and Retention

Absorbed lead is transported by blood and initially distributed according to blood distribution patterns; subsequently, it is redistributed to various compartments. Under conditions of continuous intake over a prolonged period of time, a near steady state is achieved with respect to intercompartmental distribution. The kinetics of lead distribution and accumulation in humans have not been well defined. Rabinowitz and co-workers (1976), however, have examined the distribution of lead (^{204}Pb) under dynamic conditions in five healthy men. The data obtained could be explained on the basis of a three-compartment model. The first pool, blood, contains 1.7-2.0 milligrams lead with a lifetime of 27-40 (mean 35) days. The second pool consists mainly of soft tissues. It contains 0.3-0.9 milligrams lead and has a lifetime of 30-55 days. The third pool, composed of hard tissues (mainly skeleton), contains most of the body burden of lead.

The accumulation of lead begins during fetal life. Lead is readily transferred across the placenta and has been detected in the human fetus as early as 12-14 weeks of gestation (Rosen and Sorell 1978). Among normal individuals, the total body burden of lead increases from approximately 0.2 mg at birth to values up to 200 mg during a person's lifetime; this increase is limited almost entirely to an increasing storage of lead in the calcified matrix of the bone (Chisolm and Barltrop 1979). Barry (1975, 1978) estimates the total lead content of the body may reach 119 mg in women, 165 mg in men without occupational exposure to lead, and 522 mg in men with occupational lead exposure.

The concentration of lead in blood (PbB) is of prime importance in the determination of recent lead exposure. Lead circulating in the blood is principally (~90%) bound in a slow diffusable form in the erythrocytes; the remainder, the diffusable fraction, is in the plasma (Butt et al. 1964, Posner 1977). Recent data suggest that most adult populations in the United States have mean PbB of about 10-20 μg/100 ml; approximately 3.5% of the adult population may have levels above 30 μg/100 ml (NRC 1980).

Lead levels in erythrocyte and plasma of 75 pregnant women and their infants at delivery were measured by Cavalleri and co-workers (1978). No significant differences were found in maternal and fetal values. The mean lead erythrocyte concentrations were 26.4 and 25.4 μg/100 ml in mothers and newborns, respectively. Plasma values were 0.66 and 0.62 μg/100 ml, respectively. Similar PbB values were noted in 500 Belgian mothers and their newborn infants (Roels et al. 1978).

Robinson and co-workers (1958) reported PbB values of 7-28 μg/100 ml in 9 neonates, 5-31 (median 15) μg/100 ml in 28 infants up to 6 months of age, and 3-54 (median 27) μg/100 ml in 75 children from 6 months to 13 years in age. In adults, normal PbB values range from 5-40 μg/100 ml (Goldwater and Hoover 1967) with no significant difference in PbB values of smokers (19.9 μg/100 ml) and nonsmokers (19.0 μg/100 ml) (McLaughlin and Stopps 1973).

Of 176 children between the ages of one to 9 years living within 20 miles of a lead smelter in Idaho, Walter et al. (1980) discovered that 174 had PbB levels exceeding 40 μg/100 ml; 105 of these children had PbB values >60 μg/100 ml. In contrast, Baker et al. (1977) reported PbB levels within normal limits in children living within 3 kilometers of a lead smelter.

Lead also concentrates in the teeth, particularly in secondary dentine; however, it appears to be firmly bound and unavailable for release. At present, there is no general consensus on what constitutes a normal or acceptable level of lead in teeth (Barry 1978).

5.1.2.3 Elimination

About 90% of ingested lead is eliminated, unabsorbed, via the feces (Kehoe 1961). Absorbed lead is principally excreted via the urine (76%) with lesser amounts in gastrointestinal secretions (16%) and miscellaneous routes of excretion, such as breast milk, sweat, exfoliated skin, hair, and nails (8%) (Rabinowitz et al. 1973). Normal levels of lead in urine are in the 30-40 μg/100 ml range (McLaughlin and Stopps 1973, Rabinowitz et al. 1973). Human breast milk contains < 5-12 μg/l (Hammond 1977).

5.1.2.4 Metabolism of Organolead Compounds

Organolead compounds, i.e., tetraethyllead and tetramethyllead, are readily absorbed from the gastrointestinal tract, lungs, and skin (Cohen 1979). They are dealkylated to trialkyl lead compounds by mixed function oxidases in the liver (Bolanowska 1968, Grandjean and Nielsen 1979). The trialkyl form accumulates in nonosseous tissues, particularly the liver, kidney, and brain. In rats, the half-life in liver and kidney is approximately 40 days for trimethyllead and 15 days for triethyllead (Hayakawa 1972). Tetramethyllead is also absorbed through skin and dealkylated to the trialkyl form more slowly than tetraethyllead (Grandjean and Nielsen 1979, Cremer 1965).

In a study of twenty-two individuals living in the vicinity of Copenhagen, Nielsen et al. (1979) reported a median concentration of 0.014 μg trialkyllead/g brain tissue in these individuals who had no occupational exposure to organic lead. This quantity accounted for approximately 20% of the total lead content of the brain.

5.1.3 Human and Animal Studies

In many respects, the toxic effects associated with lead exposure are similar in both experimental animals and humans. Therefore, where considerable human data are available, the primary focus was placed on humans and more specifically on data that are most useful in the assessment of acceptable limits of human exposure to lead. In areas of scant or nonexistent human data, supplemental animal data are presented in detail.

5.1.3.1 Carcinogenesis

Animal Data

Oral or parenteral administration of rather high doses of some lead salts have produced benign and malignant renal tumors in rodents. However, lead toxicosis and high mortality were also associated with these studies. Zollinger (1953) was the first to observe renal tumors following long-term subcutaneous injection of rats with lead phosphate; 19 of 29 rats (65.5%) that survived treatment for longer than 10 months developed renal tumors. Total doses ranged between 120 and 680 milligrams of lead.

More recently, lead-induced renal tumors have been confirmed in both rats and mice fed diets containing 0.1 and 1% basic lead acetate (Van Esch and Kroes 1969, Oyasu et al. 1970, Van Esch et al. 1962, Mao and Molnar 1967) and in rats fed 1% lead acetate (Boyland et al. 1962, Goyer and Rhyne 1973).

In addition to renal neoplasms, Zawirska and Medras (1968) observed tumors of the testes, adrenals, thyroid, pituitary and prostrate glands in rats fed diets containing lead acetate (3 mg/rat/day for 2 months; 4 mg/rat/day for 16 months. Oyasu et al. (1970) reported gliomas (11.8% versus 0.3% in controls) as well as renal tumors (76%) in male Sprague-Dawley rats fed 1% basic lead acetate in the diet for approximately one year.

Renal changes but no neoplasms were found in male hamsters fed 0.1 or 0.5% basic lead acetate for two years (Van Esch and Kroes 1969) and in male rats fed diets containing 0.1% lead arsenate or 0.1% lead carbonate for two years (Fairhall and Miller 1941).

Co-carcinogenic activity of lead salts has been demonstrated in rats and hamsters. Hinton and co-workers (1979) observed a co-carcinogenic interaction between lead and FBPA (N-[4'-fluoro-4-biphenyl] acetamide) resulting in the development of renal adenocarcinoma in rats. This was demonstrated by decreasing the latency period, increasing the tumor yield, and increasing the percentage of tumor-bearing rats. However, similar results were not observed in the liver, in fact the addition of 1% lead (as lead acetate) to the FBPA-diet provided early protection from the onset as well as retardation of later development of hepatocellular carcinoma.

In another study, intratracheal administration of 1 mg lead oxide with 1 mg benzo(a)pyrene per week for 10 weeks resulted in lung adenomas in 11 of 26 (42%) Syrian hamsters within 60 weeks. One adenocarcinoma of the lung was also observed. These tumors were not noted in hamsters given the same dose of either lead oxide or benzo(a)pyrene alone (Kobayashi and Okamota 1974).

A single carcinogenicity study with organic lead noted that subcutaneous injection of 0.6 milligrams of tetraethyllead given as 4 equally divided doses to Swiss mice between birth and 21 days of age

resulted in malignant lymphomas in 1 of 26 males and 5 of 41 females between 36 and 51 weeks. The incidence of this lesion in controls was 1 of 39 in males and 0 of 48 in females (Epstein and Mantel 1968). The significance of this finding, however, is difficult to assess because lymphomas occur spontaneously in this strain of mice.

Observations in Humans

Although human data are scant, no evidence suggests that lead is carcinogenic to humans (IARC 1972). In a study of 425 former employees of an accumulator factory, Dingwall-Fordyce and Lane (1963) found no evidence of an elevated incidence of malignant neoplasms. A significant excess number of deaths was noted in 170 workers with high lead exposures (100-250 μg/lead/100 ml urine) over the past 20 years; however, mortality predominantly resulted from vascular lesions of the central nervous system rather than neoplasia.

In another study, no suggestion of a relationship between lead exposure and death from cancer was found in 442 orchardists who at one time sprayed fruit trees with lead arsenate (Nelson et al. 1973).

In more recent studies (Cooper and Gaffey 1975, Cooper 1978), no consistent association between the incidence of cancer deaths and either length of employment or estimated lead exposure could be found in a cohort of 7032 heavily-exposed workers employed for more than one year from 1946-70 as smelter or lead battery workers (67% PbB $\geq$ 40 μg/100 ml, 20% $\geq$ 70 μg/100 ml). Further study of an additional 491 deaths in the same population during the period 1970-75 also resulted in no correlation between lead exposure and incidence of cancer (Cooper 1980).

With respect to organolead, a single report by Robinson (1974) noted no increased cancer rate in a 20-year follow-up of 592 workers exposed to tetraethyllead. A 20-year latency period, however, is insufficient to exclude carcinogenicity and may well underestimate the true incidence of cancer.

Thus, experiments with small rodents indicate that the addition of 0.1 or 1% basic lead acetate to the diet of rats and mice or 1% lead acetate to the diet of rats is carcinogenic, resulting in an elevated incidence of renal tumors. An increased frequency of adrenal, thyroid, brain, pituitary, and prostate tumors has also been associated with exposure of rats to lead acetate; however, these data require confirmation. Lead salts have also been shown to exhibit co-carcinogenic activity in rats and hamsters. No indication of tumors was reported following exposure to lead carbonate or lead arsenate; however, this evidence cannot be held as conclusive. Although human data are scant, no evidence suggests that lead is carcinogenic in humans. The equivalent human dose to dietary levels producing renal tumors in laboratory animals is 550 mg/day elemental lead (IARC 1972), which is far in excess of the maximum tolerated dose of lead for humans.

5.1.3.2 Mutagenesis

The evidence on a possible association of lead exposure and chromosomal aberrations in humans is inconclusive and contradictory. Chromosomal aberrations have been reported in some workers in lead industries (Deknudt et al. 1973, Bauchinger et al. 1976). These studies, however, involved mixed exposures (Pb, Zn, Cd), which may be a contributing factor to the positive findings. However, an increased frequency of aberrations has been reported in in vitro studies with human lymphocytes exposed to lead acetate (10^{-4}-10^{-6} M) in culture (Schwanitz et al. 1970, Beek and Obe 1974, Obe et al. 1975).

Numerous other observations have essentially been negative. O'Riordan and Evans (1974) found no significant increase in chromosomal aberrations in shipbreakers exposed to lead oxide fumes; the study population had PbB values ranging from 40 μg to >120 μg/100 ml. Bauchinger and co-workers (1972) found no abnormalities in the chromosomes of policemen with PbB levels elevated 20-30% above control values. Deknudt et al. (1977) noted a random increase in the incidence of rings and dicentric chromosomal aberrations in lymphocytes of some smelting plant workers; however, no correlation could be established between the number of aberrations and PbB, age, or length of exposure. PbB values ranged from 44 μg to 95 μg/100 ml. Negative results were also noted in human lymphocytes exposed in culture to 10^{-6}-10^{-2} M lead acetate (Schmid et al. 1972).

Thus, whether exposure to lead can induce chromosomal abnormalities in humans remains an unanswered question because of the lack of sufficient data on the issue.

Data from animal studies are also conflicting. Muro and Goyer (1969) noted that chromosomes from bone marrow cells of mice fed 1% lead acetate in the diet for two weeks showed an increased number of chromatid aberrations (gaps and fragments). These aberrations involved single chromatids, which suggest that injury followed DNA replication and thus could have been produced in culture. However, mice exposed to 1800 mg lead acetate/liter in drinking water for one year exhibited no increased chromosomal aberrations in either spermatocytes or marrow cells (Leonard et al. 1973) and no increase in aberrations was noted in Chinese hamster cells exposed in vitro to various concentrations of lead acetate (Bauchinger and Schmid 1972).

DiPaolo and co-workers (1978) noted that relatively high concentrations of lead acetate enhanced transformation of Syrian hamster cells by a simian adeno-virus (SA7) in vitro. Enhancement ratios of 3.1, 1.9, 1.4, 1.55, and 1.0 were recorded for hamster embryo cells exposed for 18 hours in culture to 200, 100, 50, 25, or 0 μg/ml lead acetate, respectively. Subcutaneous injection of transformed cells into either Syrian hamsters or nude mice produced fibrosarcomas.

With respect to organic lead, negative findings were reported in a dominant lethal study with tetraethyllead in mice (Kennedy et al. 1975). while exposure of Drosophila melangoster to this compound resulted in an increased occurrence of nondisjunction and chromatid breaks (Ahlberg et al. 1972, Ramel 1973).

In summary, the available experimental data do not provide unequivocal proof of the mutagenic activity of lead. Results are often conflicting and do not allow a satisfactory evaluation of the genetic risks of lead.

5.1.3.3 Adverse Reproductive Effects

Animal Data

Several animal studies indicate that sublethal lead exposures may impair normal reproductive ability. Impotence and prostate hyperplasia were observed in sexually mature male rats exposed to lead acetate for 30 days; blood lead levels were 14-26 μg/100 ml. Testicular damage with inhibition of spermatogenesis occurred in rats with PbB of 50-100 μg/100 ml. Similarly exposed female rats exhibited irregularities of the estrus cycle at PbB 14-30 μg/100 ml. Ovarian follicular cysts developed in females when PbB reached 50 μg/100 ml (Hilderbrand et al. 1973). A subsequent study (Der et al. 1974), however, was unable to replicate these findings in Sprague-Dawley rats.

In another study, Krasovskii and co-workers (1979) noted decreased sperm mobility and increased acid phosphatase activity in the gonadal tissue of rats fed 50 μg lead/kg (as lead acetate) daily for 20-30 days. Small disruptions in the permeability of vessels and dystrophic changes in the Leydig cells were also reported. No effects were seen in another group of rats identically treated with 1.5 μg lead/kg/day.

Similarly, Balb/c^+ male mice fed 0.1% lead acetate in the diet for 16 months exhibited no impairment of fertility, which was judged by their ability to impregnate untreated females. The incidence of abnormal sperm in these treated mice was comparable with control values. A second group of mice fed 1% lead acetate in the diet exhibited, within 8 weeks, a doubling in the mean number of abnormal sperm compared with controls. However, no consistently significant reduction in sperm count or sperm motility was seen. Unfortunately, males at this treatment level were not mated with virgin females to establish the effect on fertility (Eyden et al. 1978).

Sexual maturation also appears to be disrupted by lead exposure. Kimmel et al. (1976) reported a dose-related delay in puberty (vaginal opening) in female rats exposed from conception to 25, 50, or 250 mg/l lead acetate in drinking water, which, at least for the lowest dosage group, was not secondary to reduced body weight. In a later study utilizing the same treatment regimen, Kimmel et al. (1980) indicated

that delayed puberty did not affect the ability to conceive, to carry a normal litter to term, or to deliver the young. No significant increases in resorptions, malformations, or postnatal deaths were produced. Gray and Reiter (1977) noted a similar delay in female mice administered 5 mg lead/ml in drinking water at parturition; no delay in vaginal opening was noted in pair-fed control mice.

In pregnant animals, large amounts of lead can prevent implantation and cause embryonic death. In addition, a few studies have demonstrated a teratogenic effect following an acute dose of lead, although malformations have not been observed in most experimental investigations.

Gale (1978) observed a variable embryotoxic response to lead in different strains of hamsters. Lead nitrate (50 mg/kg) was injected intravenously to five inbred strains (MHA, LSH, LHC, CB, and PD4) and one outbred strain (LVG) of pregnant hamsters early on the eighth day of gestation. The MHA and CB strains were relatively resistant to lead exposure; however, the other strains exhibited increased resorptions (80-92%), tail bud abnormalities, hydrocephalus, and skeletal defects.

In rats, intravenous injection of aqueous solutions of 0, 5, or 25 mg $^{210}Pb(NO_3)_2$/kg on day 9 of gestation produced no effects at the low dose, but resulted in 43.5% resorption and 17.7% incidence of stunting and external malformations in fetuses in the 25 mg/kg group examined on day 20. Dams similarly injected on day 15 of gestation exhibited no overt toxicity at the low dose, but 100% resorption occurred at the 25-mg/kg level. Injection of 25 mg/kg on day 15 was associated with petechial hemorrhages in fetal brain within 90 minutes of dosing with more massive hemorrhaging consistently noted 24 hours post-dosing (Hackett et al. 1978).

Gerber and co-workers (1978) noted pregnant C57Bl mice fed diets containing 0.5 or 0.25% lead (as lead acetate) exhibited decreased pregnancy incidence, increased embryonic death, and retarded growth in surviving embryos. Maisin et al. (1975) noted similar findings in mice fed 0.1 or 0.5% lead in the diet during gestation. In contrast, Kennedy et al. (1975) reported no effects on the number of fetuses resorbed, the number of viable fetuses, or the incidence of terata in either mice or rats. In this study, 7.14, 71.4, or 714 mg/lead/kg was administered to mice and rats on days 5-15 and days 6-16 of gestation, respectively.

Urorectocaudal malformations were produced in the surviving offspring of rats injected intravenously with 50 mg lead nitrate/kg on day 9 of gestation. Malformations noted include absence of tail (21%), absence of genitalia (23%), imperforate anus (12%), and sirenomelia (16%). None of these abnormalities was present in untreated controls. These malformations were not observed in rats injected with the same dose on day 16 of gestation; however 40% of survivors were hydrocephalic (McClain and Becker 1975). Tail abnormalities, ranging from stunting to the complete absence of the tail, have been reported in hamsters following injection of dams with various lead salts (Ferm and Carpenter 1967). In addition,

treatment of chick embryos with lead salts has been shown to produce hydrocephalus and meningoceles (Butt et al. 1952, McLaughlin et al. 1963), as well as cardiac abnormalities (Gilani 1973).

With respect to organic lead, the inability of the fetus to metabolize tetraelkyllead compounds to their trialkylform appears to protect the fetus. No terata have been observed in either rats (Odenbro and Kihlstrom 1977, Kennedy et al. 1975) or mice (Kennedy et al. 1975) exposed in utero to tetralead compounds. Increased resorption of fetuses, growth retardation, and incomplete bone ossification were noted in rats exposed to either tetraethyl- or tetramethyl-lead; however, these incidences occurred only as a result of doses that produced distinct maternal toxicity (McClain and Becker 1975).

Observations in Humans

Lead exerts a profound, adverse effect on the fetus and interferes with the reproductive ability of both men and women at PbB levels of 30-40 μg/100 ml (NRC 1980). Several stages of the reproductive process are vulnerable to lead. They are the sperm and/or egg prior to conception, the embryo during pregnancy, and the neonate.

Historically, lead exposure has been linked to disturbances of menstruation and elevated incidences of miscarriages and stillbirths in women working in lead industries. This was particularly the case during the latter half of the 19th century. Recently, Panova (1972) reported that women working in a printing plant for 1-12 months at ambient air levels of < 7 μg Pb/m^3 had a higher incidence of ovulatory dysfunction (mainly anovulatory cycles) compared with a control group. In a critical review of this study, however, Zielhuis and Wibowo (1977) noted the difficulty in evaluating Panova's conclusions because of the design of and the presentation of data. More importantly, no consideration appears to have been given to the dust levels of lead, which is an important consideration in print shops.

Lead absorbed into the bloodstream of pregnant women crosses the placenta and enters the blood of the fetus. Umbilical cord blood lead levels are similar to those found in mother's blood (Cavalleri et al. 1978). Because the fetus develops rapidly, it is particularly vulnerable to intrauterine exposure to lead. Furthermore, calcium and iron deficiencies, which are commonly observed in pregnant women, cause the mother to have a higher risk of lead toxicosis.

Both Lane (1949) and Nogaki (1958) suggest that miscarriages can occur in women with only modest exposure to lead; and Wibberley et al. (1977) recently noted a striking increase in the level of placental lead associated with stillbirths. Fahim et al. (1976) reported elevated incidences of pre-term deliveries (13 versus 3 in controls) and early fetal membrane rupture (17 versus 0.41 in controls) in women living in a lead mining area of Missouri compared with women living in an urban environment in Missouri. One confusing aspect of this study, however, is the unexplained similarity in PbB levels in all mothers.

There are also reports on the effects of lead on the male reproductive function. Lancranjan et al. (1975) reported evidence of decreased fertility in occupationally-exposed men. These disturbances included alterations in spermatogenesis (increased frequency of asthenospermia, hypospermia and teratospermia) in men with PbB of 50-80 μg/100 ml. No effects were noted in the 23-41 μg/100 ml PbB range. Reduced sexual potency, testicular pain, and a reduced number of spermatozoa have been reported in workers exposed to tetraethyllead (Vurdelja et al. 1967, Neshkov 1971).

No data suggest that lead is teratogenic in humans. One report (Palmisano et al. 1969) suggested a possible link between maternal consumption of lead-contaminated whiskey with neuromuscular abnormalities and failure to thrive in a ten-week-old infant whose mother had lead poisoning concomitant with alcoholism.

Thus, several studies indicate that exposure to high levels of lead may impair normal reproductive ability in experimental animals (e.g., testicular damage, irregularities of the estrus cycle, disruption of sexual maturation). In pregnant animals, lead is embryotoxic; and, at least in some species, lead induces terata of the urorectocaudal region subsequent to acute lead exposure. A number of investigators, however, have observed no increase in the incidence of terata in experimental animals exposed to lead during gestation.

Lead also exerts significant adverse effects on the reproductive function in humans; however, most available data are related to high occupational exposure and therefore are difficult to extrapolate to normal exposure levels. No evidence suggests that lead is teratogenic in humans.

5.1.3.4 Other Toxic Effects

No beneficial effects of lead have been found. Because of its accumulation in bone tissue, even a low daily intake of lead can eventually produce toxic effects. Although other systems may be adversely affected, in humans, as in experimental animals, the three principal target organs for lead are the erythroid cells of the bone marrow, the kidney, and the central and peripheral nervous systems. The inhibitory effects of lead on erythropoiesis are reversible; however, severe acute or chronic lead poisoning may be followed by irreversible injury to the kidney and nervous system.

Hematopoietic System

Exposure to lead results in derangement of the hematopoietic system with disruption of hemoglobin synthesis considered the critical or first adverse effect of lead exposure. Lead interferes with hemoglobin synthesis at a number of steps: (1) it causes partial inhibition of several enzymatic steps in the biosynthesis of heme; (2) it impairs the uptake and use of iron; and (3) it impairs globin synthesis in the developing erythroid cells of the bone marrow.

Heme is formed in the mitochondria and is an essential component of the cytochrome system and cell respiration. Heme also serves as the prosthetic group of hemoglobin, the protein that transports oxygen from the respiratory system to the cells of the body. The biosynthesis of heme is a multi-step process. At least two of these steps are considered directly inhibited by lead. These are: (1) the transformation of δ-aminolevulinic acid (ALA) into porphobilinogen, catalyzed by δ-aminolevulinate dehydratase (ALAD); and (2) the insertion of iron in protoporphyrin IX to form heme, catalyzed by ferrochelatase (heme synthetase) (Hammond 1977, Chisolm 1978). Lead also affects other steps in the process of heme synthesis, such as δ-ALA synthetase and coprogenase. However, these effects may be secondary results of feedback derepression rather than a direct effect of lead (Hammond 1977).

Although specific inhibition of ferrochelatase by lead is generally accepted as the cause of the accumulation of protoporphyrin in the erythrocytes, lead probably also inhibits the availability of iron for coupling with protoporphyrin (WHO 1977). Globin synthesis in the red blood cells is also apparently impaired; however, the mechanisms that cause reduced globin synthesis are unknown (WHO 1977).

In general, clinical anemia does not occur until PbB levels are >80 μg/100 ml (Muir and Bridbord 1977). The available data (Tola et al. 1973) suggest, however, that mild anemia with a small reduction in blood hemoglobin may occur in adults at or slightly above the dose levels associated with a minimal increase in urinary excretion of ALA (PbB 50 μg/100 ml. Children appear to be more sensitive to lead anemia than adults; reduction in hemoglobin may occur at PbB of ~40 μg/100 ml (WHO 1977). An increased rate of erythrocyte breakdown (decreased erythrocyte life) is often, but not consistently, seen in cases of anemia as a result of lead poisoning (WHO 1977).

Indicators of the critical effect of lead on hemoglobin synthesis include rapid depression of ALAD activity, an associated increase in urinary ALA, and a delayed rise in free erythrocyte protoporphyrin (FEP) in blood (Chisolm 1978).

Because lead acts directly on the circulating erythrocytes, the rapid depression of ALAD activity most closely correlates with the concentration of lead in the blood. There is an inverse linear relationship between the logarithm of ALAD activity in erythrocytes and the concentration of lead in whole blood over a PbB range of 5-95 μg/100 ml. The average no-effects PbB threshold for inhibition of erythrocyte ALAD activity is about 10-20 μg/100 ml (Tola et al. 1973). Nordman and Hernberg (1975), however, have reported an inhibition of ALAD at PbB of 8.4 μg/100 ml, which raises questions concerning the existence of a no-effect level. The toxicological implications of ALAD inhibition are not adequately known.

Significant inhibition of ALAD is associated with the accumulation and excretion of excess ALA in urine and a rise in blood FEP. The

relationship between ALA in urine and PbB is not linear; the increase of ALA in urine becomes marked when PbB is >40-60 μg (Tsuchiya 1979). The rise in FEP and the depression of ALAD activity are almost equally as sensitive as indices of lead exposure. FEP elevation is delayed by approximately two weeks because it reflects inhibitory effects occurring in erythroid cells in the bone marrow (Hammond 1977). Women and children appear to have an earlier and more rapid increase in FEP than men for the same levels of PbB. Blood lead threshold for the FEP response is ~15-20 μg/100 ml for adult females and children and ~25-30 μg/100 ml for adult males (Zielhuis 1975). The reasons for these differences are not entirely clear; however, they may be related to differences in iron stores, hormonal factors, and growth rate (Roels et al. 1978).

Renal System

The nephropathic effects of lead intoxication are profound. Two stages of lead-induced nephropathy are: (1) proximal tubular morphologic changes accompanied by a reversible tubular dysfunction, which is seen mainly with short-term exposure; and (2) interstitial fibrosis, which is generally considered to be of a slow, progressive nature and which eventually causes contracted kidneys and chronic renal failure (Goyer 1979, Payne and Saunders 1978).

Acute lead nephropathy in children is characterized by dysfunction of the proximal renal tubules (Fanconi's syndrome) manifested as glycosuria, hypophosphatemia with phosphaturia and generalized aminoaciduria (Granick et al. 1978). Pueschel et al. (1972) reported generalized aminoaciduria in 8 of 43 children with PbBs of 40-120 μg/100 ml and slight lead-related neurological signs. Similar symptoms have been observed in occupationally-exposed adults (Clarkson and Kench 1956, Goyer et al. 1972); however, PbB levels were not reported. In adults, amonoaciduria is probably uncommon at PbB levels < 70 μg/100 ml (U.S. EPA 1980). Changes in the proximal tubules include the formulation of intranuclear inclusion bodies of lead-protein complexes and alterations in mitochondrial structure and function (Goyer and Mushak 1977). Treatment with chelating agents will reverse a minoaciduria along with the functional and morphological changes associated with lead toxicity (Goyer and Mushak 1977).

It is uncertain if acute lead nephropathy, treated or untreated, influences the development of any form of chronic nephropathy without continuous lead exposure. An Australian study indicated that long-term exposure to lead early in life could result in chronic nephropathy in adulthood (Emmerson 1968). On the other hand, Tepper (1963) was unable to find evidence of chronic nephropathy in young American adults 10-20 years after childhood lead poisoning. A recent study in rats (Fine et al. 1979) also reported no indications of progressive, interstitial nephropathy or alterations in renal growth or development, despite an acute increase in the body burden of lead in growing rats.

Long-term exposure to lead may result in the development of irreversible functional and morphological renal changes. The second stage of lead nephropathy has the morphologic appearance of an interstitial nephritis. Interstitial fibrosis becomes progressively severe with tubular atrophy and eventually reduced glomerular filtration progressing into renal failure (Goyer 1979).

Reduced glomerular filtration occurs at relatively low levels of lead exposure (Weeden et al. 1975, Cramer et al. 1974, Lilis et al. 1979). Weeden and co-workers (1975) reported reduced glomerular filtration rates in four of eight men occupationally exposed to lead (PbB 48-98 μg/100 ml). Lilis et al. (1979) noted a sizable reduction in the glomerular filtration rate (age-adjusted) of 255 secondary lead smelting workers. A significant increase in blood-urea-nitrogen and serum creatinine levels, which correlated with the duration of lead exposure, were also documented. A total of 18, 37, 37, and 7% of the test population had PbB levels in the >80, 60-79, 40-59 and <40 μg/100 ml, respectively.

Thus, proximal tubular dysfunction can occur in both children and adults and are generally noted after short-term exposure. Kidney disease associated with chronic lead exposure has not been adequately studied and is difficult to detect, i.e., blood urea nitrogen and serum creatinine become elevated only when two-thirds of the kidney function is lost. Little is known about dose-response relationships; however PbB >70 μg/100 ml for a prolonged period are believed to give rise to chronic irreversible nephropathy (Goyer 1979).

Central and Peripheral Nervous Systems

Among the most devastating effects of increased lead absorption are the effects on the central and peripheral nervous systems. Toxic levels of lead in the central nervous system (CNS) can interfere with neurotransmitters or the levels of essential metals as well as inhibit various enzyme systems, some of which are responsible for the energy metabolism of the brain (Granick et al. 1978). Manifested as encephalopathy, effects on the CNS are seen more frequently in children than adults. This may be attributed to several factors; e.g., children absorb more lead from the diet than adults; the brain is especially vulnerable to insult during the period of rapid neurodevelopment; and behavioral patterns, such as pica, may predispose children to higher lead exposure. In addition to age, the severity of encephalopathy also depends on the intensity and duration of exposure. Major features of encephalopathy are: dullness, hyperkinetic or aggressive behavior, headaches, muscular tremors, hallucinations, and, in severe cases, convulsions, mania, paralysis, and coma (WHO 1977). The minimal level of lead exposure associated with lead encephalopathy in children is not clearly known; however, it is estimated to be 80 μg/100 ml PbB (WHO 1977).

Arnvig and co-workers (1980) reported that nine men with occupational exposure to lead in a battery plant exhibited PbB levels of

58-82 μg/100 ml. The authors conducted numerous psychological tests on these men and observed severe deficiencies in memory tests, concentration and attention, and psychomotor performance. Although they did not evaluate a control group, the authors feel that serious abnormalities were identified.

Recently, however, several studies have raised questions about the subtle impairment of the cerebral function in children at sub-encephalopathic blood lead levels (40-80 μg/100 ml) (Landrigan et al. 1975, De la Burde and Choate 1972, 1975, Perino and Ernhart 1974). These studies, however, have been subject to criticism because of flaws in the experimental design, such as overlapping of lead exposures between test and control populations, insensitivity of behavioral tests, complications of nutritional and socioeconomic status, etc. General findings of a few key studies are discussed below; however, the interested reader is urged to review the detailed analyses of the neurological and behavioral effects of lead by Repko and Corum (1979), Needleman (1980), and the U.S. EPA (1977).

De la Burde and Choate (1972, 1975) compared children with a history of pica and elevated PbB (>40 μg/100 ml) with control children with no history of pica. The lead-exposed children showed mental impairment, irritability, and poor fine motor coordination. Although the groups were matched for race, age, sex, and socioeconomic variables, no lead assessments were made for the control group.

A controversial study by Landrigan et al. (1975) reported neurological dysfunction in 46 symptom-free children (3-15 years) with moderately elevated blood lead concentrations (mean 48 μg/100 ml) when compared with children with PbB <40 μg (mean 27 μg/100 ml). The lead-exposed group showed lower scores (Wechsler Intelligence Scale) and poorer results in the finger-wrist tapping test than controls. However, no differences were exhibited between the groups in full-scale I.Q., verbal I.Q., or hyperactivity behavior ratings. No conclusive evidence of lead as a causative factor was presented. Perino and Ernhart (1974) also concluded that neurobehavioral deficits occurred at PbB levels as low as 40 μg/100 ml.

Diminished learning ability (I.Q. drop of 4-7 points) and behavioral changes, such as diminished attention span in children, have also been associated with increased concentrations of lead in teeth (Needleman et al. 1974).

Conversely, several studies have reported negative results between lead-exposed groups and controls (Kotok 1972, Lansdown et al. 1974, McNeil et al. 1975). These studies, however, have the same type of flaws as the studies that suggest positive correlations between low lead levels and neurological deficits in children.

Despite problems inherent in these studies, sufficient evidence indicates that subtle neurobehavioral effects do occur in children

exposed to sub-encephalopathic levels of lead. The minimal level of lead exposure, duration of exposure, and the age of susceptibility, however, cannot be clearly defined with any degree of certainty. The World Health Organization (1977) concluded that the probability of noticeable brain dysfunction increases in children from PbB levels ~50 μg/100 ml.

Subtle behavioral changes have also been documented in experimental animals (Shih and Hanin 1978, Laporte and Talbott 1978, Dietz et al. 1978, Bushnell and Bowman 1979). Effects noted include changes in time perception (Dietz et al. 1978), learning ability (Laporte and Talbott 1978, Bushnell and Bowman 1979), motor activity (Reiter et al. 1975), and avoidance behavior as adults (Krigman 1978). Lin-Fu (1976) cited several animal studies that suggest hyperactivity might result from moderate lead exposure via mother's milk.

Hernberg et al. (1967) and Landrigan and Baker (1976) have also documented the effects of lead exposure in the peripheral nervous system of both adults and children. Peripheral neuropathy affects somatic motor neurons and is characterized by the loss of nerve fibers (axons and myelin sheaths) and, to some degree, segmental demyelination (Krigman 1978). Classic symptoms are wrist drop and lead "colic," a reflection of the effect of lead on the autonomic innervation of the gut (Granick et al. 1978). A number of studies has also documented the occurrence of slowed nerve conduction with an approximate PbB >50 μg/100 ml (Landrigan and Baker 1976, Hernberg et al. 1967, Lilis et al. 1977).

5.1.4 Overview

5.1.4.1 Ambient Water Quality Criteria -- Human Health

An ambient water quality criterion of 50 μg/l lead has been established to protect human health (U.S. EPA 1980). The criterion is based on on calculated level that is protective of the effects of lead on heme synthesis, the "critical effect" selected by the U.S. EPA (1980) for establishing standards. Because children are more sensitive to the effects of lead than adults, the U.S. EPA has established that the maximum safe blood lead level (PbB) for any given child should be lower than the threshold for a decrement in hemoglobin (PbB = 40 μg/100 ml). The Center for Disease Control and the American Academy of Pediatrics recommended a PbB of 30 μg/100 ml. However, the U.S. EPA (1980) has estimated that if the geometric mean PbB was kept at 15 μg/100 ml, 99.5% of all children would have PbB $\leq$ 30 μg/100 ml.

Non-air sources contributing to PbB levels have been estimated to be 10-12 μg/100 ml. It is believed that dietary intake is the main contributor to this PbB level (U.S. EPA 1980). U.S. EPA (1980) assumed that food and the available drinking water ($\leq$10 μg Pb/l) are the principal contributors to a PbB level of 12 μg/100 ml, and calculated that the consistent consumption of drinking water at the present lead standard (50 μg Pb/l) would contribute an additional 3.4 μg/100 ml to PbB levels.

This would yield a PbB of 15.4 μg/100 ml, attributable to food and drinking water. A value approximately the same as the maximum geometric mean PbB value that would maintain 99.5% of the children in the United States at a PbB level <30 μg/100 ml. However, this blood level (15.4 μg/100 ml) does not take into consideration contributions from air.

5.1.4.2 Other Considerations

Lead has no natural or beneficial function in the body and even a low daily intake of lead can eventually produce toxic effects. Children appear to be particularly susceptible to the effects of lead, which probably reflects the marked difference in lead absorption between children and adults. In general, young children absorb 3-10 times more lead than adults. Between 5 and 10% of ingested lead and 30% of inhaled lead are absorbed by an adult. Lead salts do not readily penetrate intact skin.

Absorbed lead is transported by the blood; and, under steady-state conditions, is believed to be distributed into three compartments: blood, soft tissues, and the calcified matrix of bone. The concentration of lead in blood (PbB) is of prime importance in the determination of recent lead exposure. Most adults in the United States have mean PbB of 10-20 μg/100 ml. Approximately 90% of the lead body burden is stored in bone. The effects of lead, however, are more closely related to the concentration of lead in critical tissues, such as the brain and the kidney. Although other systems may be adversely affected, the most prominent effects of lead are noted in the erythropoietic system, the nervous system, and in the kidney.

Lead exposure results in derangement of the heme-hematopoietic system, with disruption of hemoglobin synthesis being the first observed adverse effect of lead exposure. In general, clinical anemia does not occur until PbB values are >80 μg/100 ml; however, mild anemia may occur with PbB levels of ~40 μg/100 ml in children and at or slightly above 50 μg/100 ml in adults.

The effects of lead exposure on the central and peripheral nervous systems include encephalopathy, interference of neurotransmitters, and inhibition of various enzyme systems responsible for energy metabolism in the brain. Encephalopathy is rare in adults but more frequent in children. The lowest reported PbB effect levels are 80 μg/100 ml for children and 100 μg/100 ml for adults. Recently, subtle neuro-behavioral impairments have been reported at PbB levels for which no overt symptoms of lead toxicity are seen. Although data are somewhat contradictory, the evidence suggests a slight cognitive impairment and possible behavioral effects in individuals with PbB concentrations persistently greater than 40 μg/100 ml. Below this value, some effects are indicated; however, the evidence, to date, is inconclusive.

The nephropathic effects of lead intoxication are profound. Proximal tubular dysfunction can occur in both children (PbB = 40-120 μg/100 ml) and adults (PbB >70 μg/100 ml) and is generally noted after short-term exposure. Prolonged exposure, resulting in PbB values >70 μg/100 ml, may cause irreversible functional and morphological renal changes.

Although human data on the carcinogenicity of lead are scant, no evidence suggests that lead is carcinogenic to humans. Many of the animal studies that indicate carcinogenic activity for some lead compounds are not suitable for extrapolation to humans because of the inappropriate routes of exposure. Feeding experiments with rodents have shown that the addition of 0.1 to 1% basic lead acetate to the diet of rats and mice or 1% lead acetate to the diet of rats is carcinogenic, resulting in an elevated incidence of renal tumors. Lead salts have also been shown to be co-carcinogenic in rats and hamsters. The equivalent human dose to dietary levels producing renal tumors in laboratory animals is 550 mg of elemental lead/day, which is far in excess of the maximum tolerated dose of lead in humans. No carcinogenic activity has been noted in occupationally-exposed persons with PbB >40 μg/100 ml.

High occupational exposures to lead have resulted in profound adverse effects on fetuses and have interfered with the reproductive ability of both men and women at PbB levels of 30-40 μg/100 ml. No evidence indicates that lead is teratogenic in humans. In pregnant animals, lead is embryotoxic and, at least in some species, induces terata of the urorectocaudal region with doses of 50 mg/kg. Several investigators, however, have observed no increase in the incidence of terata in experimental animals exposed to similar levels of lead during gestation. Exposure to high lead levels, however, does impair normal reproductive ability in experimental animals (e.g., testicular damage, irregularities of the estrus cycle, disruption of sexual maturation).

Evidence for a possible association of lead exposure and chromosomal aberrations in humans is inconclusive and contradictory. Chromosomal aberrations have been reported in some workers in lead industries; these studies, however, involved exposures to multiple agents that may confound reported findings. Numerous other observations have been essentially negative. Data from animal studies are also conflicting.

Thus, a multitude of adverse health effects are associated with lead intoxication. Blood lead levels in the 30-40 μg/100 ml range are the lowest effect levels reported to date. Some evidence indicates adverse effects below this value; however, to date, the evidence is inconclusive. Therefore, the lower limits of lead exposure at which toxic effects may occur cannot be quantified accurately.

5.2 HUMAN EXPOSURE

5.2.1 Introduction

The effects of lead on humans have been studied for years as described in the previous section. In order to deal with this problem, numerous regulatory mechanisms have been initiated; i.e., setting an air quality standard, a drinking water standard, and a limit on lead content in paints (NRC 1980). In addition, lead screening programs have been established for children, and some states have enacted laws intended to deal with this hazard.

Although "undue" exposure to adults appears limited primarily to occupational settings, exposure to lead is prevalent among children. In 1973, 11.1% of 277,347 children tested in the lead screening program showed blood lead levels (PbB) >40 μg/100 ml. In 1974 and 1975, this percentage decreased to 6.4 and 6.5, respectively. In 1976, when the level defined as an elevated blood lead level was reduced to 30 μg Pb/100 ml, 8.7% of the 500,463 children screened exceeded this level. In addition, 2.7% of the children exceeded 50 μg/100 ml in the blood (Center for Disease Control 1977). Studies of large populations of children have shown that PbB levels show lognormal distributions with geometric means for city children of 20-30 μg/ml (Billick et al. undated-a, Angle and McIntire 1979).

One of many problems associated with the quantification of human exposure to lead is the establishment of a background exposure. Although a PbB of 25 μg/ml is not considered excessive in terms of the lead screening program, it is probably in excess of mean PbB levels in rural areas (Angle and McIntire 1979) and of historical levels (Settle and Patterson 1980). These authors have suggested that typical American skeletal concentrations of lead are 500 times higher than ancient Peruvian skeletons. Thus, "normal" levels of lead do not necessarily represent acceptable levels.

An additional problem in the quantification of human exposure to lead is that PbB is commonly used as an indication of exposure. Though this measure can be used, to some extent, in the assessment of risk, PbB levels indicate relatively recent exposure (Needleman et al. 1979). Also, it is difficult to identify the source of exposure, except through regression analyses or similar statistical methods. Although other indices have been used to estimate exposure (i.e., hair, teeth), especially in a relative sense, they do not correlate as well with effects as PbB values.

One of the most difficult problems in the quantification of human exposure is inherent in the analytical techniques, and laboratory and sample contamination. This has been noted numerous times in the literature. Keppler (1970) reported the results of interlaboratory analyses for lead in blood and found that results from 60% of the laboratories

were unacceptable. More recently, Settle and Patterson (1980) have discussed this problem in the analysis of albacore. They reported a 1000-fold error in the lead concentration reported by the National Marine Fisheries Service (NMFS) when it dissected, handled, and analyzed the specimen. Settle and Patterson (1980) attributed a 20-fold error to improper sample preparation and a 50-fold error to improper analyses. These authors suggest that such errors are widespread and have led to an underestimation of exposure as a result of anthropogenic sources.

This section will attempt to identify the routes of exposure to lead for humans. In addition, attempts will be made, based on the literature, to identify the major routes of exposure in varying situations. It is particularly difficult to assess the routes of exposure for children because certain behavior patterns are not well studied or quantified; e.g., the composition of the diet and behavioral aspects (i.e., pica, contact with dust).

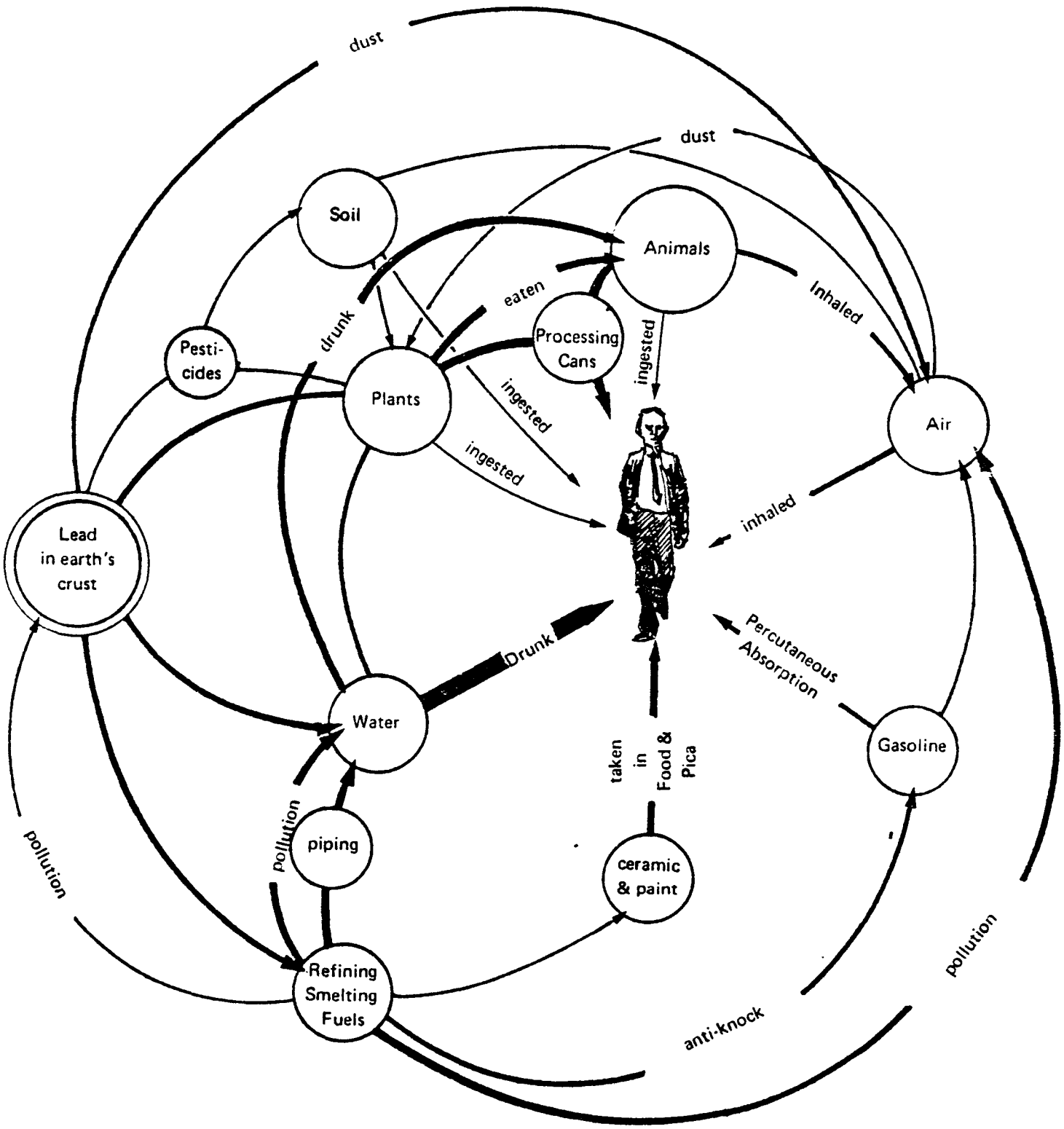
The known exposure routes for humans are summarized in Figure 5-1. Note that the routes are numerous and complex. Although food is generally considered the largest source of lead in exposure to adults, none of these routes can be considered minor, because both drinking water and air can result in more significant exposures in some situations. Similarly, the consumption of paint chips is considered the largest exposure route of lead for children; however, other routes, including contact with dust and dirt, may be significant. In general, exposure through percutaneous absorption, as well as exposure to alkyl lead compounds, are thought to be important only in occupational settings (WHO 1977). Thus, these two exposure routes will not be discussed here.

The following sections will consider exposure of humans to lead through food, drinking water, air, the ingestion of nonfood substances, and miscellaneous sources. Because these subjects have been reviewed extensively (WHO 1977, Mahaffey 1978, U.S. EPA 1977, Tsuchiya 1979, NRC 1980), these reviews have been used here to avoid duplication of effort and have been supplemented with pertinent work from the recent literature.

5.2.2 Populations Exposed Through Food

5.2.2.1 Pathways of Exposure

Intake of lead in food is thought to be the primary pathway for adults not employed in lead-related industries and for children without pica (Mahaffey 1978). Sources of lead in food are numerous and the pathways from the original source to the diet are sometimes complex (see Figure 5-1). In general, lead can either be taken up through surface deposition on the plant or through the roots (see Chapter 4.0). Foods of animal origin (meats, unprocessed milk, eggs) are generally lower in lead (Mahaffey 1978). Sources of lead in the diet include contamination resulting from lead-soldered cans, past use of lead arsenate pesticides, deposition on soil or plants from such sources as



Source: Moore (1979).

FIGURE 5–1 POPULATION EXPOSURE ROUTES FOR LEAD

automobile and smelter emissions, contamination of moonshine whiskey, leaching from improperly glazed earthenware, and uptake from lead in cooking water (Mahaffey 1978, Moore 1979).

Although each of these routes has been important in certain instances, and, in some cases, fatal, the contamination of food through the use of lead solder in cans represents the most widespread source of lead in the diet. In 1975, the Federal Drug Administration (FDA) estimated that canned foods comprise 11-12% of a person's diet after 1 year of age; however, the lead resulting from these foods comprises 30% of the average dietary intake of lead. More recently, the FDA has said that lead-soldered cans are used to package 10-15% of all food, and they contribute about 14% to the total lead ingested (Anonymous 1979a). Settle and Patterson (1980) have estimated that canned foods comprise about 20% of the diet, and that 50% of the lead in the American diet originates from lead-soldered cans. According to Settle and Patterson (1980), the lower percentage estimated by the FDA may be a reflection of their overestimation of uncontaminated food sources.

Mitchell and Aldous (1974) examined the contents of 256 metal cans and reported that 62% of them contained lead at concentrations 100 μg/l, 37% >200 μg/l, and 12% >400 μg/l. In contrast, only 1% of products in glass or aluminum containers had lead concentrations >200 μg/l. The same authors conducted a survey of milk and reported that bulk milk contained a mean lead concentration of 40 μg/l. Other authors found that a normal level for milk was 2-10 μg/l lead (Harding 1978). However, canned evaporated milk contained a mean lead concentration of 202 μg/l. The difference can be attributed to contamination in processing and from the lead solder. Similar results have been reported in baby food, with a mean concentration of 202 μg/l for canned-food items, and a mean concentration of 35 μg/l for bottled foods (Mitchell and Aldous 1974). Settle and Patterson (1980) found that tuna in lead-soldered cans contained 1400 μg/kg lead (wet weight); however, tuna packed in a die-punched unsoldered can contained 7 μg/kg lead. These results did not represent a survey of levels in tuna.

It is apparent that lead-soldered cans represent an important source of lead in the diet; however, the contribution from this source is highly variable. To complicate matters further, the FDA reported that higher concentrations of lead were found in foods stored in opened cans than in unopened cans (Anonymous 1979b). Thus, persons who store foods in opened cans (lead-soldered) may be exposed to higher levels of lead in foods than some studies have indicated.

Automobile exhaust, as a source of lead deposition on plants and soil, is important in some areas where traffic volume is higher. For example, Rabinowitz (1974) estimated that in Southern California, 60-70% of lead in oat tops and 80% of lead in lettuce leaves were attributed to automobile exhaust. The absorption of lead in air depends highly on the type of crop. In addition, elevated concentrations in plants are observed

within 100-200 meters of the highway (see Chapter 4.0). Still, it is not possible to identify what portion of the diet contains lead as a result of automobile emissions, even though it is evident, in some cases, that this portion could be high.

Food contamination resulting from other sources of lead, such as smelters, can also be important. Elevated levels in plants appear to be spread over a wider area from these sources (see Chapter 4.0). As an example of levels resulting from a smelter area, Roberts et al. (1974) found mean levels of 2.9 mg lead/kg (fresh weight) in lettuce leaves from a smelter area, as compared with mean levels from the urban control area of 0.9 mg/kg lead. On the other hand, no difference was observed in lead levels in tomatoes from the two areas. Leafy vegetables, however, retain larger amounts from air deposition (Rabinowitz 1974). Again, it is difficult to identify what portion of the diet of persons living near smelters can be attributed to lead emissions.

Past uses of lead arsenate as a pesticide have resulted in accumulation of lead in the soil (see Chapter 4.0). This source of lead can result in food contamination in crops cultivated in areas where lead arsenate was used. Elfving and co-workers (1978) have reported elevated concentrations of lead (over controls) in carrots and millet grown on old orchard soils (7.1 and 6.8 mg/kg lead dry weight). However, the soil in these cases contained 218 mg/kg lead. Considerably higher concentrations of lead have been reported in other orchard sites.

The number of people exposed to high levels of lead as a result of beverages contaminated because of improperly glazed earthenware and distilling and uptake from cooking water is probably small compared with the pathways described above. However, they can be extremely serious, and, in certain situations, they are sometimes fatal. The storage of acidic foods in earthenware has resulted in clinical lead poisoning (Mahaffey 1978); however, no estimates of intake have been made from such incidents. On the other hand, lead contamination of distilled alcoholic beverages are somewhat better studied. Ball and Sorenson (1969) estimated that 70 million gallons of "moonshine" are produced annually. Sandstead and co-workers (1970) found that 30% of the moonshine samples tested contained lead concentrations >1 mg/l. Consumption of 1 l/day would result in an intake of 1 mg/day of lead.

Commercially available wines also contain lead averaging ~0.2 mg/l (Mahaffey 1978, U.S. EPA 1977). Exposure to lead from this source could result in an intake of 0.2-1 mg/day, depending on the amount of wine consumed (1-5 liters).

The uptake of lead by food cooked in contaminated water has not been quantified; however, Moore (1979) reported that most foods show a positive uptake of lead from contaminated cooking water. Because most waters contain concentrations <0.05 mg/l, this exposure pathway is not expected to be significant.

5.2.2.2 Total Dietary Intake -- Adults

Over the past 30-40 years, various authors have estimated dietary intake of lead in adults. WHO (1977) and Mahaffey (1978) provide a detailed review of these studies; thus, they will not be discussed in detail here. In addition, because of analytical problems, some of the older estimates may be suspect.

Tepper (1971) estimated an average intake of 137 μg/day. Kolbye et al. (1974) estimated average intakes of 57-233 μg/day, depending on how the trace and undetected values were dealt with. Gross (1979) reporting the results of the Kehoe lead balance experiments, found an overall average dietary intake of 180 μg/day (159 μg/day, using median data). These estimates of dietary intake represent the mean or typical intake. Thus, it is apparent that the "average" adult consumes 100-200 μg lead/day in food. However, Gross (1979) reported a maximum intake (mean for an individual) of 334 μg/day.

Intake does vary greatly between individual and on a daily basis. Up to 50% of this intake may be a result of lead solder in cans. The remaining intake cannot be allocated to particular sources; however, it probably results from contamination during processing and the other sources described above.

In addition, persons living near areas of high lead emissions may ingest even higher doses of lead. Kerin (1972) found that the persons living near a lead smelter ingested 640-2640 μg/day in the diet, probably as a result of the contamination of local crops.

5.2.2.3 Total Dietary Intake -- Infants and Children

Kolbye and co-workers (1974) estimated dietary intake of 6-month-old children eating adult table food and infant foods. These authors found that the average intake ranged from 100-140 μg/day. Stewart and Skeberdis (1975) found that infants from 1-12 months consumed 93 ± 36 μg/day. Boppel (1975) estimated that a 6-month-old infant (7.2 kg) fed commercial baby food would have a lead intake of 45 μg/day. Breast-fed infants are probably exposed to somewhat lower levels of lead (Moore 1979, Walker 1980), in general, because breast milk has been shown to contain 6-59 μg/l lead. Canned milk or formula would contain higher concentrations of lead, perhaps in the range of 200 μg/l as reported by Mitchell and Aldous (1974) for evaporated milk. Walker (1980) found a range of 50-500 μg/l in canned infant formula. Formula diluted with water would generally contain less than 50 μg/l. However, concentrations may be much higher in formula diluted with contaminated water, similar to concentrations reported for electric kettles. The use of these products in preparing infant formula has resulted in severe lead poisoning (Wigle and Charlebois 1978).

Older children may be exposed to somewhat lower levels of lead in food. Kolbye and co-workers (1974) estimated a dietary intake of

75 μg/day for a 2-year-old child. Mahaffey (1976) studied children aged 6-47 months. In 1975, she found a daily intake ranging from 12-505 μg Pb/day with a mean intake of 110 μg/day, and a median of 76 μg/day. In 1976, a similar study showed a range of 11-719 μg/day, with a mean intake of 115 μg/day and a median of 95 μg/day. Lead intake for the highest decile was 316 μg/day (Mahaffey 1976).

The specific sources associated with lead in the diet of children are unknown. However, lead solder in cans is likely to be at least as significant a source for children as it is for adults (up to 50% of dietary intake). For an infant consuming canned formula, this source, as well as contamination during processing, would result in 100% of the dietary intake.

5.2.3 Populations Exposed Through Drinking Water

5.2.3.1 Pathways of Exposure

Surface and groundwaters in the United States generally contain low concentrations of lead (see Chapter 4.0). Similarly, 25% of the 2595 tap water samples contained undetectable levels of lead. However, 41 of these samples contained concentrations >50 μg/l, perhaps serving around 2% of the population (U.S. DHEW 1970). Lead service lines, plumbing, solder, and storage containers can result in contamination of drinking water. In general, these lead concentrations are related to pH and hardness of the water, the length of the household lead pipe, the amount of lead in the pipe, and the position of the pipe. A low pH is generally considered the most important factor that contributes to high concentrations of lead in drinking water.

The presence of a low pH and soft water is well known in the Boston and Seattle areas. Greathouse et al. (1976) reported that drinking water from 25% of the households sampled in Boston exceeded 50 μg/l, and Dangel (1975) reported that 24% of the water in Seattle exceeded this level.

Cambridge and Somerville, Massachusetts also have had high levels of lead in drinking water; 14.5 and 30% of the households surveyed showed water lead levels of greater than 50 μg/l. It has been estimated that about half of the buildings in these cities have some lead service pipe. The initiation of pH adjustment in 1975, however, reduced the lead levels in households that had exceeded the standard to less than about 20 μg/l in Cambridge (Karalekas et al. 1976).

Other cities with levels of lead greater than 50 μg/l have also been reported, including Worcester, Massachusetts (O'Brien 1976); Bennington, Vermont (Taylor 1977), and New Bedford, Massachusetts (Karalekas et al. 1978). Rural water supplies have also been identified as having elevated lead levels in drinking water, such as Chesterfield County, South Carolina, where 10.3% of the 217 samples taken exceeded 50 μg/l (Sandhu et al. 1977).

The prevalence of elevated lead levels in drinking waters in the United States today is difficult to evaluate for several reasons. One, the use of lead pipes is not well documented. In a survey of water utilities reported by Donaldson (1924), 51% of the 539 cities surveyed indicated that they used lead or lead-lined services to some extent. It is unlikely that the situation has changed drastically since that time. Patterson and O'Brien (1979) reported that nearly half of the 100 largest cities in the U.S. distribute corrosive water, and many have some portion of lead pipes. Thus, it appears that there is a potential for a large portion of the population to receive lead at levels greater than 50 μg/l in drinking water. However, such approaches as pH adjustment and treatment with zinc orthophosphate are being used to control corrosion (Karalekas et al. 1976).

5.2.3.2 Drinking Water Exposure -- Adults

Drinking water is sometimes included in the estimates of dietary exposure described above. Often, this is not well documented, however. for the purposes of this report, a consumption of 2 l/day water will be assumed for adults, in addition to the dietary consumption of lead. Thus, most adults would ingest <20 μg/day lead in drinking water. Although the population sizes are unknown, a small proportion of the population would be exposed to 100 μg/day from this source, and an extremely limited subpopulation would be exposed to 2 mg/day in drinking water.

5.2.3.3 Drinking Water Exposure -- Infants and Children

A drinking water consumption of 1 l/day will be assumed for infants and children, thus reducing the intakes estimated above by one-half. Therefore, the respective subpopulations would receive <10 μg/day, 50 μg/day, and 1 mg/day in drinking water.

5.2.4 Populations Exposed Through Inhalation (Air, Dirt, and Dust)

5.2.4.1 Pathways of Exposure

The major source of lead in the atmosphere is automobile emission (see Chapters 3.0 and 4.0). Local contamination of air around smelters, battery manufacturers, etc. also results in human exposure. Adults are directly exposed through inhalation of ambient air. The exposure pathway for children is far more complicated because deposited particulates result in contamination of dust and dirt. Intentional or inadvertent ingestion of lead from this source is not easily quantified and will be discussed later in this section.

Concentrations of lead reported in air have been discussed in Chapter 4.0. The U.S. EPA (1977) also provides a discussion on this subject. For the purposes here, various concentrations typical of certain situations have been assumed (see Table 5-1).

TABLE 5-1. HUMAN EXPOSURE TO LEAD THROUGH INHALATION

Subpopulation	Conc ($\mu g/m^3$)	Inhalation Adults[a] (μg/day)	Inhalation Child[b] (μg/day)
Smelters, battery operations, etc.	10	200	40
Urban			
Outdoors	1-4	13-53	2.7-10.8
Indoors[c]	0.3-1.4	2-9.4	0.4-1.8
TOTAL		15-62	3.1-12.6
Rural (suburban)			
Outdoors	0.1-1	1.3-13	0.3-3
Indoors	0.03-0.3	0.2-2	.04-0.4
TOTAL		1.5-15	.34-3.4
Remote	<.02	0.4	0.08

[a]Inhalation of 20 m^3/day assumed for adults spending 1/3 time indoors, 2/3 outdoors.

[b]Inhalation of 4 m^3/day for 1-year old child spending 1/3 time indoors, 2/3 outdoors.

[c]Concentrations assumed to be 1/3 outdoors.

Source: See Section 4.2.2 for concentrations of lead in air.

5.2.4.2 Inhalation Exposure -- Adults

Inhalation estimates of lead for adults have also been described in Table 5-1. A respiratory flow of 20 m^3/day was assumed. Potential exposure appears to be highest in urban areas and near lead-related industries.

5.2.4.3 Inhalation Exposure -- Infants and Children

Inhalation for a one-year-old child was assumed in Table 5-1. The patterns of exposure are the same as those seen in adults. Older children would experience increasingly high total exposure to lead.

5.2.5 Populations Exposed Through Other Routes

5.2.5.1 Cigarettes

Lead has been reported in cigarettes at concentrations up to 39 mg/kg (Cogbill and Hobbs 1957). This has been primarily attributed to past uses of lead arsenate (Mahaffey 1978, WHO 1977). WHO (1977) estimated, assuming a 2% transfer rate from mainstream smoke, that a person smoking 20 cigarettes/day would inhale 1-5 μg/day.

5.2.5.2 Dirt and Dust

Lead reaches dirt and dust through various pathways (see Figure 5-1). Of particular importance are automobile and smelter emissions and losses from painted surfaces that result in soil contamination. Monitoring data (see Chapter 4.0) have shown that levels approaching 8,000 mg/kg in soil can result in the vicinity of such sources. Household dust and street dust also exhibit elevated concentrations. Old orchard sites and manufacturing sites may also have high levels of lead.

These levels are probably not of great significance for adults; however, children are susceptible to this exposure route through ingestion of dirt and dust, and mouthing of contaminated hands and other objects. Lepow et al. (1974) examined ten children with chronically high PbB levels. These authors determined that nine of the ten children studied exhibited excessive mouthing. Three of the children had their hands or nonfood items in their mouths during 50% of the observation time. The mean lead in the samples from the childrens' hands was 2400 μg/g. This study estimated that if a small child playing in dirt with a lead concentration of only 1200 μg/g mouthed his fingers 10 times/day, ingesting 10 mg of dirt/mouthing, this child would ingest 120 μg/day of lead. The same calculation using household dust (with a concentration of 11,000 μg/g) would result in an exposure of 1100 μg/day (Lepow et al. 1974). Unfortunately, little is known about such behavior patterns of children. These estimates, however, do not appear unreasonable and may be common in areas with contaminated soil and dust.

5.2.5.3 Paint

The ingestion of paint chips by children is commonly thought to be the most prevalent route of lead exposure for children in the United States today. Paints used before World War II contained lead at levels greater than 1% (10,000 mg/kg). In 1974, a marketplace survey found that 70.8% of the oil-based paints and 96.1% of the water-based paints sampled contained < 0.06% lead, which is the recommended maximum permissible level in paint (Committee on Toxicology 1976). However, numerous old buildings still exist with chipped and peeling paint that contain higher levels of lead. Gilsinn (1972) estimates that 7 million housing units are old enough to contain high levels of lead.

This problem is especially severe for children having pica. It appears common in children, especially 1-to-3-year-olds; estimates suggest that one-third to one-half of the children in this age bracket have pica. Unfortunately, little is known about the amount of paint ingested. However, only small amounts (a 1-milligram chip) of paint containing 1% lead are required to result in an exposure of 1 mg/day (Mahaffey 1978). Such an exposure would probably be common in children having pica and who are exposed to lead at such levels.

5.2.5.4 Other Routes

Numerous other exposure routes for lead are generally considered to be minor. For example, ingestion of newsprint, curtain weights, lead paint on kitchen utensils, decorative decals, and lead stearate used in PVC pipes can all result in exposure to lead (WHO 1977, Mahaffey 1978). These exposure routes are expected to be important only in isolated situations and have not been included here.

5.2.6 Blood Levels Associated with Various Subpopulations

Bell and co-workers (1979) report a survey of the literature shows PbB levels in rural or urban areas in the range of 9-24 μg/100 ml with most values ∿16 μg lead/100 ml blood. Gross (1979) reported an overall mean PbB level in a sample of adults of 25 μg/100 ml, and individual mean levels ranged from 18-40 μg/100 ml. WHO (1977) estimated that each 100 μg of oral intake from dietary sources contributes about 6-18 μg of lead/100 ml of blood. Thus, as expected, dietary intake accounts for most of the lead in the blood of adults.

In addition, numerous studies have been conducted to determine the importance of air concentrations in contributing to lead in blood. These studies are reviewed in U.S. EPA (1977) and Bell *et al*. (1979) and will not be discussed in detail here. In total, these studies appear to indicate that the contribution of air lead to blood lead ranges from 0.6-2.0 μg/100 ml in blood/μg lead/m^3 in air. In general, blood lead concentrations do not correlate well with air concentrations because the effects are masked by intake from other sources, primarily food. However, urban levels of blood lead have been shown to be

significantly higher than suburban levels (Tepper and Levin 1972). The geometric mean blood lead (age and smoking adjusted) for urban areas was 18 μg/100 ml and 16 μg/100 ml for rural. Assuming a log-normal distribution, <5% of the urban population would have PbB levels >30 μg/100 ml; and <0.5% of the rural population (U.S. EPA 1977).

A relationship between blood lead and distance from highways has also been observed. Daines and co-workers (1972) found that mean PbB levels for black women living 3.7, 38.1, and 121.9 meters from a highway were 23.1, 17.6, and 17.4 μg/100 ml, respectively.

Levels of blood lead in persons living near smelters are also elevated. Landrigan et al. (1975) found that 16% of the individuals over 19 living near the smelter in El Paso, Texas had PbB levels >40 μg/100 ml.

In addition, relationships between blood lead and levels in drinking water have been observed. Moore and co-workers (1977) found that the mean PbB level rose as the cube root of the "first draw" of water. Thus, they estimated that PbB levels at water concentrations of 50 and 100 μg/l would be about 20 μg/100 ml and 22 μg/100 ml, respectively. Although this may represent a significant difference, the example indicates that drinking water does not contribute greatly to lead exposure in adults, except at very high concentrations.

In general, elevated PbB levels (>15 μg/100 ml) have been related to such factors as residency in urban areas, near highways, and near smelters. Blood levels of lead >40 μg/100 ml in adults are found in these areas.

Blood levels of lead in children are consistently higher than those of adults in the same environment. Lead screening programs detect about 40,000 children annually with PbB levels >30 μg/100 ml. However, this figure represents only a proportion of those children in the United States who actually exceed this level. McFeatters (1976) reports that the U.S. Department of Housing and Urban Development estimates that at least 600,000 U.S. children have high levels of lead in their blood. The preceding discussion indicates that the largest route of exposure for children with pica is the ingestion of paint, dirt, and dust. The source of this contamination is primarily paint in rural areas, automobile emissions in urban areas, and smelter or other industrial emissions near sources. Numerous studies have been conducted to determine the relative importance of these various routes and sources. Again, these have been described extensively in U.S. EPA (1977) and Bell et al. (1979) and will only be discussed briefly here.

The distance of residence from a highway has been correlated with PbB levels of children. Caprio and co-workers (1974) found that >57% of the children living within 100 feet of roadways had PbB levels >40 μg/100 ml. More than 27% of those living 100-200 feet from the

highway and 31% of those living greater than 200 feet away had similar levels. Although this study indicated a localized effect on PbB levels, the pathway of exposure could have been inhalation or ingestion of dirt and dust.

Billick and co-workers (undated-a) examined 170,000 records from 1976 of children's PbB levels in New York. These authors found that blacks had higher PbB levels than whites or Hispanics. In addition, a peak in PbB levels was observed during the summer each year. The highest PbB levels were found in the 2-to-4-year-old age bracket. In addition, a general downward trend was observed in PbB levels during these years. This was attributed to the lead screening program, decrease in paint exposure, and changes in environmental lead exposure. The air lead concentrations, ethnic group, and age were highly significant factors in determining PbB levels and explained 63-65% of the variation in PbB levels.

In a follow-up study, Billick and co-workers (undated-b) evaluated lead in gasoline as an indicator of exposure. They found that gasoline lead (as measured by sales) is a highly significant variable. They developed a model that explained 75% of the variation in PbB level by age, race, season, and leaded gasoline sales. Using the model, these authors estimated the geometric mean concentration for children in the absence of gasoline lead to be 10.1 μg/100 ml for the first quarter. In comparison, the yearly geometric mean for black, Hispanic, and white children of the same age was 21.2, 19.2, and 19.3 μg/100 ml, respectively. These results suggest a reduction of about 50% in childrens' PbB levels in urban areas with the phasing out of lead in gasoline. The ambient air contributed little to the variation in blood, after gasoline lead had been considered. These authors suggest that inhalation was only secondary to ingestion of dirt and dust.

Angle and McIntire (1979) investigated the correlation of childhood PbB level with lead in air, soil, and house dust. A multivariate analysis showed that PbB levels for preschool children was positively correlated with house dust and soil (11% of the variance) and PbB levels in children 6-18 was significantly correlated with air, soil, and house dust (21% of the variance). However, 38% of the variation was a result of the difference between samples from an individual.

The U.S. EPA (1977) concluded that observable increases in PbB levels occur at soil or dust lead exposures of 500-1000 mg/kg. In a summary of soil lead/blood lead relationships on children, these authors found a mean increase of 3-6% in lead blood for a two-fold increase in soil lead levels.

Lead paint is a well known source of elevated blood levels in children (U.S. EPA 1977, WHO 1977, Bell _et al_. 1979). In general, however, the contribution of paint to levels in blood is difficult to distinguish from the contribution of lead in soil and dust, especially in urban areas. Cohen and co-workers (1973) found that the mean PbB

level for 230 children (1-5 years old) in two rural counties was 22.8 ± 11.0 μg/100 ml. More than one-half of the children lived in houses more than twenty-five years old, one-quarter of which had flaking paint or plaster. The presence of lead in paint or plaster is not the only condition that must be met to increase levels of lead in the blood; the paint must also be peeling or chipping. In addition, for exposure to occur, it must be ingested. As a result, it is difficult to correlate lead in paint with PbB levels in children (U.S. EPA 1977).

Children with high PbB levels are also concentrated around industrial sources of lead, especially smelters. Most recently, Walter and co-workers (1980) studied PbB levels in the areas surrounding a primary lead smelter in the Idaho Silver Valley. These authors found that 98.9% of the children surveyed in 1974 (ages 1-9) that resided closest to the smelter had PbB levels >40 μg/100 ml and 59.7% had levels >60 μg/100 ml. A multiple regression analysis for each year of age showed that air lead was the most significant variable influencing PbB level. The next most important variable was levels of lead in household dust, primarily in children younger than four. Soil lead was also a significant factor in determining blood lead. Pica was significant at age 2 years, and 25% increase in blood lead was predicted in a child with pica. In addition, sex, and occupation of the parent were significant variables. These authors also noted that inhalation is probably not the major pathway of absorption. Lead was apparently deposited and ingested.

A follow-up survey conducted in 1975 showed that PbB levels were somewhat reduced, probably because of emigration, treatment of children with high PbB levels, increased cleanliness, replacement of highly contaminated topsoil, and reduction of lead emissions (Walter et al. 1980). Today, almost all children have PbB levels <60 μg/100 ml, and most have levels <40 μg/100 ml (Anonymous 1979a).

Similar results, although less severe, have been reported from the lead smelter in El Paso, Texas. About 70% of the children 1-4 years old living near this smelter had PbB levels >40 μg/100 ml, and 14% had PbB levels >60 μg/100 ml (Landrigan et al. 1975).

Baker and co-workers (1977) examined lead absorption in children in nineteen U.S. towns with primary nonferrous smelters and compared them with children of the same age in towns without smelters. This included all primary lead, zinc, and copper smelters, with the exception of the smelters in El Paso, Texas, Kellogg, Idaho, Tacoma, Washington, and East Helena, Montana, because these towns had already been studied. They found that PbB levels were significantly elevated in children living around two zinc smelters, but not near the two lead smelters. However, hair-lead levels were elevated in these areas.

Elevated lead levels have also been reported in the vicinity of secondary smelters and battery plants (U.S. EPA 1977). PbB levels >30 μg/100 ml appear to be common in these areas.

In summary, elevated lead levels (a term difficult to define, but generally meaning >30 μg/l) have been reported in numerous situations for children. Exposure to lead-containing paint, dirt, and dust are probably the major sources of exposure. Contaminated dirt and dust can result from paint, automobile emissions, and industrial emissions from such sources as primary and secondary smelters and battery plants.

5.2.7 Summary -- Exposure Scenarios

5.2.7.1 Introduction

This section will attempt to isolate certain subpopulations and determine their total exposure to lead. In addition, an attempt will be made to identify important routes and sources of exposure. Although these situations are somewhat hypothetical, they are based on the data available in this section and the preceding ones in order to represent typical examples of exposure.

This section concentrates on total intake; however, for comparison of routes, the absorbed dose must be considered. As discussed in the previous section, the absorption of lead depends on a number of factors, such as particle size, physiochemical form of lead, route, and extent of exposure, as well as host factors, such as age and nutritional status. For the estimates presented here, absorption of lead from the gastro-intestinal tract of adults will be assumed at 10%. Absorption of ingested lead by children is assumed to be 50%. Deposition of inhaled lead is assumed to be 30% and 100% of deposited lead is assumed to be absorbed. Little is known about absorption of inhaled lead by children; however, it is assumed to be the same as adults, although it may be greater.

5.2.7.2 Exposure Estimates

Exposure estimates for children and adults in different situations are summarized in Table 5-2. Figures 5-2 and 5-3 show the estimated exposure for adults and children living in rural, urban, and industrial (smelters, etc.) environments. The relative importance of the various routes are shown using the estimates derived earlier in this section. For adults, food is the greatest source of exposure in all the exposure scenarios shown in Figures 5-2 and 5-3. Intake of lead in food was considered the same (200 μg/day) in urban and rural areas; however, near smelters, it was assumed to be 1000 μg/day because of contamination of local crops. Drinking water intakes for all the scenarios shown in Figures 5-2 and 5-3 were assumed to be <20 μg/day, because most drinking waters contain <10 μg/l. However, concentrations of 50 μg/l in rural areas would increase the contribution of lead from this source from 9 to 31%. This effect would not be as dramatic in urban and industrial areas. Highly contaminated drinking water (1 mg/l) would overshadow all other sources of exposure (see Table 5-2).

TABLE 5-2. EXPOSURE ESTIMATES OF LEAD FOR ADULTS AND CHILDREN LIVING IN RURAL, URBAN, AND INDUSTRIAL ENVIRONMENTS

Population	Location	Route	Source	Assumption	Intake (μg/day)	Absorbed Dose[a] (μg/day)
Adults						
	Rural	Food	Total Diet		100-200	10-20
			Moonshine	1 mg/l, 1 l/day	1000	100
			Wine	0.2 mg/l, 1/day	200	20
				0.2 mg/k, 5 l/day	1000	100
		Drinking Water	Most Supplies	<10 μg/l, 2 l/day	<20	<2
			Contaminated	>50 μg/l, 2 l/day	>100	>10
			Highly Contaminated	>1000 μg/l, 2 l/day	>2000	>200
		Inhalation	Suburban	See Table 5-1	1.5-15	0.5-5
			Remote	See Table 5-1	0.4	0.1
			Cigarettes	-	-	1-5
	Urban	Food	Total Diet		100-200	10-20
			Moonshine	1 mg/l, 1 l/day	1000	100
			Wine	0.2 mg/l, 1 l/day	200	20
				0.2 mg/l 5 l/day	1000	100
		Drinking Water	Most Supplies	<10 μg/l, 2 l/day	<20	<2
			Contaminated	>50 μg/l, 2 l/day	>100	>10
			Highly Contaminated	>1000 μg/l, 2 l/day	>2000	>200
		Inhalation	Urban Air	See Table 5-1	15-62	5-21
			Cigarettes	-	-	1-5
	Smelting Areas	Food	Total Diet	-	100-2640	10-264
			Moonshine	1 mg/l, 1 l/day	1000	100
			Wine	0.2 mg/l, 1 l/day	200	20
				0.2 mg/l, 5 l/day	1000	100

TABLE 5-2. EXPOSURE ESTIMATES OF LEAD FOR ADULTS AND CHILDREN LIVING IN RURAL, URBAN, AND INDUSTRIAL ENVIRONMENTS (Continued)

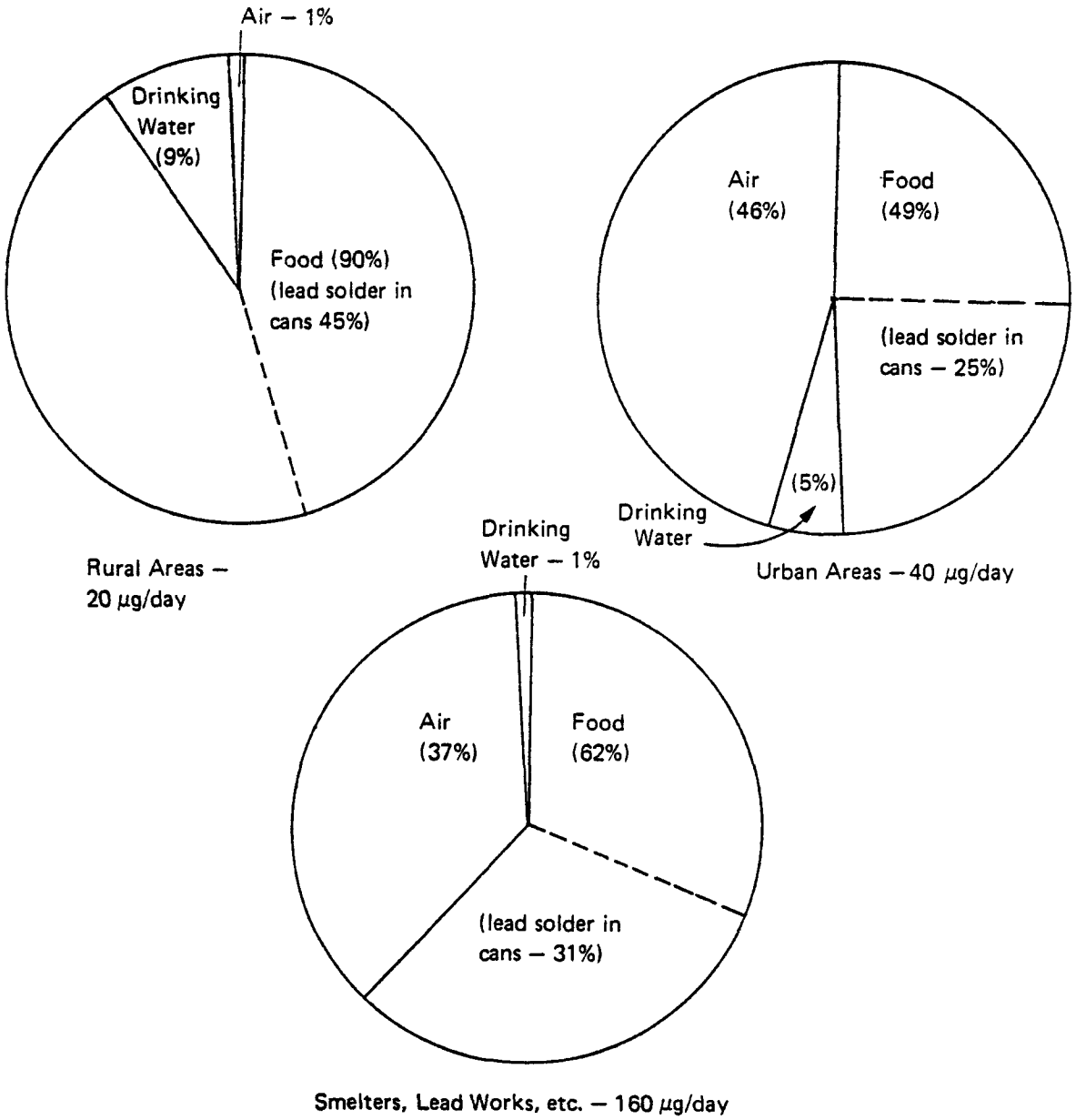
Population	Location	Route	Source	Assumption	Intake (μg/day)	Absorbed Dose[a] (μg/day)
		Drinking Water	Most Supplies	<10 μg/l, 2 l/day	<20	<2
			Contaminated	>50 μg/l, 2 l/day	>100	>10
			Highly Contaminated	>1000 μg/l, 2 l/day	>2000	>200
		Inhalation	Ambient Air	10 μg/m^3, 20 m^3/day	200	60
			Cigarettes	-	-	1-5
Children						
	Rural	Food	Total Diet	-	100	50
		Drinking Water	Most Supplies	<10 μg/l, 1 l/day	<10	<5
			Contaminated	<50 μg/l, 1 l/day	<50	<25
			Highly Contaminated	>1000 μg/l, 1 l/day	>1000	>500
		Inhalation	Ambient Air	See Table 5-1	0.34-3.4	0.1-1
		Pica	Lead Paint	1% lead, 1 mg chip	1000	500
			Paint or Otherwise Contaminated Dirt	1000 μg/g lead in dirt, 10 mg/mouthing, 10 mouthings/day	100	50
	Urban	Food	Total Diet	-	100	50
		Drinking Water	Most Supplies	<10 μg/l, 1 l/day	<10	<5
			Contaminated	<50 μg/l, 1 l/day	<50	<25
			Highly Contaminated	>1000 μg/l, 1 l/day	>1000	>500
		Inhalation	Ambient Air	See Table 5-1	3.1-12.6	0.9-4
		Pica	Lead Paint	1% lead, 1 mg chip	1000	500
			Dirt	1000 μg/g lead in dust 10 mg/mouthing, 10 mouthings/day	100	50
			Dust	10,000 μg/g lead in dust 10 mg dust/mouthing, 10 mouthings/day	1000	500

TABLE 5-2. EXPOSURE ESTIMATES OF LEAD FOR ADULTS AND CHILDREN LIVING IN RURAL, URBAN, AND INDUSTRIAL ENVIRONMENTS (Continued)

Population	Location	Route	Source	Assumption	Intake (μg/day)	Absorbed Dose[a] (μg/day)
	Smelting Areas	Food	Total Diet	Assumed to be half of adult	500	250
		Drinking Water	Most Supplies	< 10 μg/l, 1 l/day	< 10	< 5
			Contaminated	<50 μg/l, 1 l/day	<50	<25
			Highly Contaminated	> 1000 μg/l, 1 l/day	> 1000	> 500
		Inhalation	Ambient Air	See Table 5-1	40	12
		Pica	Lead Paint	1% lead, 1 mg chip	1000	500
			Dirt	1000 μg/g lead in dirt, 10 mg/mouthing, 10 mouthings/day	100	50
			Dust	10,000 μg/g lead in dust 10 mg dust/mouthing, 10 mouthings/day	1000	500

[a] A 10% absorption of ingested lead is assumed for adults and 50% for children. Deposition of inhaled lead is assumed to be 30% and 100% of deposited lead is assumed to be absorbed.

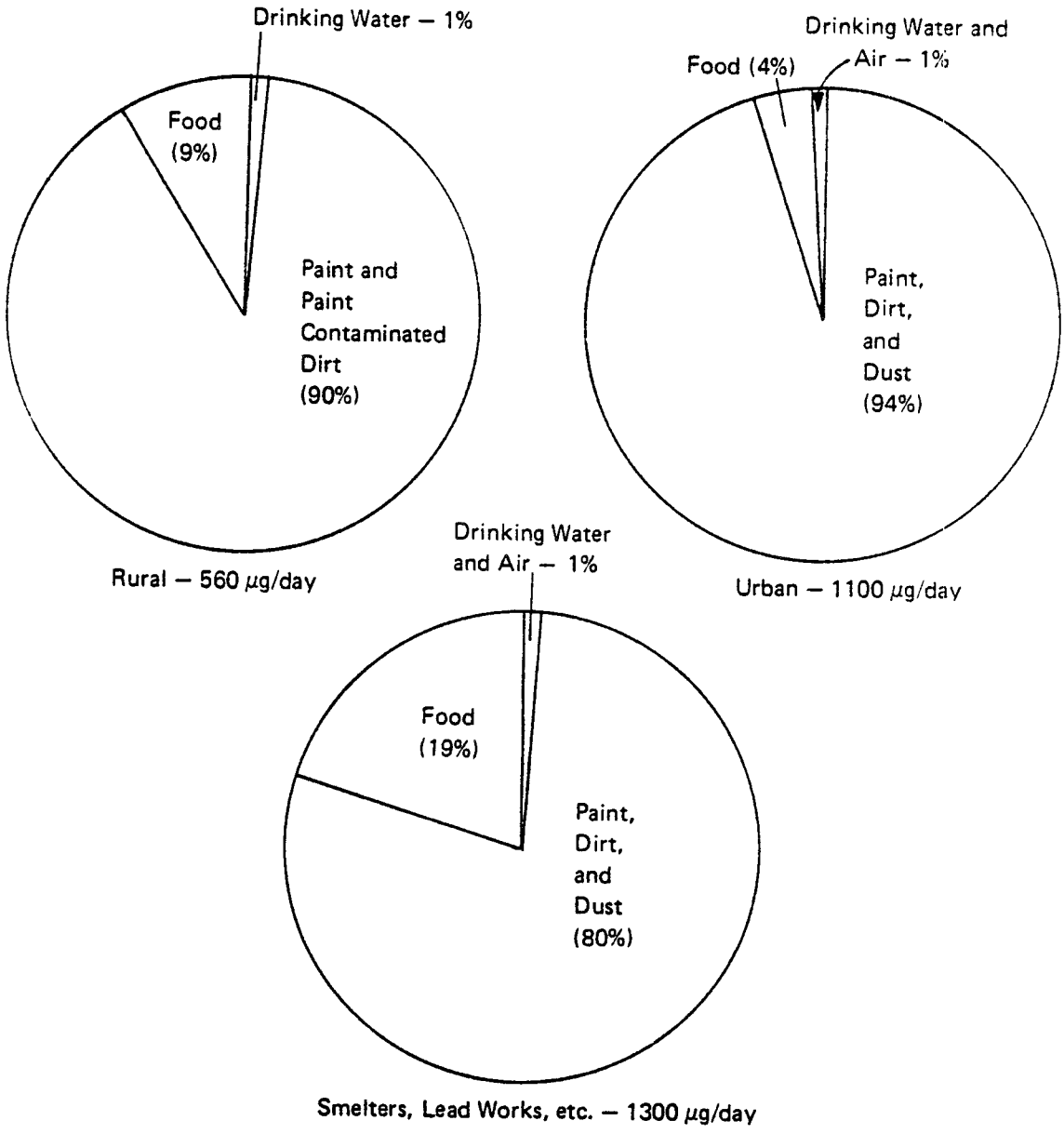
Source: See text.



Note: Concentrations < 10 μg/ℓ in drinking water were assumed for these estimates, and no consumption of wine or moonshine containing lead. In addition, these situations did not include exposure from smoking.

Source: Arthur D. Little, Inc., estimates.

FIGURE 5–2 EXPOSURE SCENARIOS – ADULTS (ABSORBED DOSE)



Source: Arthur D. Little, Inc., estimates.

FIGURE 5–3 EXPOSURE SCENARIOS – CHILDREN WITH PICA (ABSORBED DOSE)

If moonshine is consumed (1 liter containing 1 mg/l/day), this source of exposure would be far greater than any other source. Wines would have to be consumed in large amounts (5 l/day containing 200 μg/l lead) in order to be the dominant exposure route in lead industrial areas. However, consumption of 1 l/day would represent about 50% of the total absorbed dose in rural areas, and 30% in urban areas. The increase in total exposure from rural to urban areas is a result of the increased exposure through inhalation; primarily due to lead emissions from automobiles. Even in urban areas, however, food is at least as significant a source if not more significant than air. The increased exposure in the vicinity of smelters and perhaps other lead industries is related to increased inhalation and perhaps increased levels of lead in foods.

These results suggest that elimination of lead solder in cans would reduce exposure by, at most, ~6-50% in adults, depending on the subpopulation exposed. In addition, the elimination of lead in gasoline would reduce exposure in urban areas by about 35% (assuming no reduction in solder in cans). If lead were eliminated from cans as well as from gasoline, exposure could be reduced by about 60%. However, persons consuming large amounts of lead in drinking water, moonshine, or wine would not experience such reduction in exposure. These estimates are only an approximation and are intended to demonstrate the relative nature of exposure of lead to adults.

Similar exposure scenarios for children with pica are illustrated in Figure 5-3. As expected, consumption of paint, dirt, and dust containing lead represents the largest exposure route for these children. Exposure to children without pica would be much lower, for example, 560 as compared to ~60 μg/day in rural areas. In urban and industrial areas, children without pica may be exposed to somewhat higher levels than would be expected because dirt and dust may be contaminated, and mouthing of objects and hands can result in exposure.

In the scenarios shown in Figure 5-3, concentrations of lead in drinking water were assumed <10 μg/l. However, higher levels in drinking water would probably not contribute greatly to exposure for children with pica. It would be a significant source for infants and children without pica.

The increase in exposure from rural, urban, and industrial areas is primarily a result of the increase in lead levels of soil and dust. Gross estimates were made in order to evaluate the importance of this source in total exposure to children. However, even if these estimates were reduced by an order of magnitude, consumption of paint, dirt, and dust would still constitute the major source of exposure for children.

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6.0 BIOTIC EFFECTS AND EXPOSURE

6.1 EFFECTS ON BIOTA

6.1.1 Introduction

This section provides information on the exposure levels of lead that disrupt the normal behavior and metabolic processes in aquatic and terrestrial organisms. Data are based on the laboratory studies from the available literature. In addition to its widespread publicity with regard to human health effects, lead has been the focus of extensive research on plant and animal toxicosis. The U.S. EPA (1980) has reviewed this data, thus only brief discussions will be contained here.

In aquatic bioassays, a certain amount of variability is expected, owing to several factors. Differences in test type, for example, static, or continuous flow-through tests, may influence the results. In addition, water parameters, such as hardness and the presence of other chemicals, are known to influence the toxicity of lead to various aquatic organisms, as well as the form of lead (inorganic or organic) used. Thus, variations in these parameters between experiments could yield varying results. Other factors include the duration of exposure and species and developmental stage of the test organisms. Because some species and stages may be more sensitive to lead than others, it is not always appropriate to compare the results of different studies.

6.1.2 Freshwater Organisms

6.1.2.1 Chronic and Sublethal Effects

When chronically exposed to low levels, aquatic biota may become acclimated to a toxic substance, or they may exhibit adverse effects, such as decreased respiration, lack of responsiveness to stimulation, growth inhibition, and malformation. Even if aquatic fauna are not killed by long-term exposure to lead, the vigor and diversity of local populations may still be endangered.

Data on the sublethal effects of lead on freshwater fish are summarized in Table 6-1. The lowest concentration at which effects were reported was 7.6 µg/l (hardness 28 mg/l), which caused developmental irregularities in rainbow trout fingerlings in a chronic exposure test (Davies *et al*. 1976). Other sublethal effects have been noted at lead concentrations as high as 110 mg/l, and include fin erosion, tail blackening, loss of equilibrium, poor resorption of yolk in fry, and growth inhibition. The U.S. EPA (1980) reported chronic values ranging from 19 µg/l (hardness 28 mg/l) for the rainbow trout (*Salmo gairdneri*) to 174 µg/l (hardness 38 mg/l) for the whtie sucker (*Catastomus commersoni*). These values were primarily based on effects observed in early life stages.

TABLE 6-1. SUBLETHAL EFFECTS OF LEAD ON FRESHWATER FISH

Species	Concentration (mg/l)	Compound	Experimental Conditions	Effects	Reference
Rainbow trout, fingerling (*Salmo gairdneri*)	0.0076-0.064	Pb $(NO_3)_2$	Life-cycle, 28 & 353 mg/l hardness	Lordoscoliosis, paralysis, muscular atrophy, degeneration of caudal fin, blackening of tail	Davies *et al.* (1976)
Zebrafish, eggs (*Brachydanio rerio*)	0.036-0.072	Pb $(NO_3)_2$	Embryo-larval, 26° 1.6 mg/l alkalinity static	Poor resorption of yolk in fry, erosion of tail & fin, spinal curvature, apparent epitheliomas	Ozoh (1979a)
Goldfish (*Carassius auratus*)	0.070	Pb $(NO_3)_2$	pH 6.0-6.9, 23°	Significant impairment of conditioned response	Weir and Hine (1970)
	6.6	Pb $(NO_3)_2$	soft water	7-day LC_{50}	
	110		hard water	7-day LC_{50}	
Rainbow trout, yearling (*Salmo gairdneri*)	0.12	Pb $(NO_3)_2$	Flow-through 32 wk, 135 mg/l hardness, pH 7.7, 11°	Increase in number of erythrocytes, 30% incidence of black tails	Hodson *et al.* (1978)
	2.4			21-day LC_{50}	
Brook trout (Salvelinus fontinalis)	0.134	Pb $(NO_3)_2$	44 mg/l hardness	Reduced growth	Christensen (1975)
Chinook salmon (Oncorhynchus tshawytscha)	10	Pb $(NO_3)_2$	Static, 4-6 hour, 11° pH 7.2, 0-17 mg/l hard	Loss of equilibrium	MacPhee and Reulle (1969)

The range of sublethal and chronic effect concentrations for invertebrates seems considerably narrower than for fish (see Table 6-2), although this could be a function of the particular species tested. The isopod, Asellus meridianus, exhibited reduced growth rates in 0.1 mg/l lead over a period of 20 days (Brown 1976). The only other reported sublethal effect was reduced respiration in the sludgeworm, which was attributed to the mucous-metal complex that formed on the body wall and blocked the exchange of oxygen and carbon dioxide (Whitley 1968). The U.S. EPA (1980) reported chronic values ranging from 12 µg/l to 128 µg/l for Daphnia magna at water hardness of 52-151 mg/l $CaCO_3$. The chronic value for the snail (Lymnea palustris) was 25 µg/l at 139 mg/l hardness. These were based on life cycle or partial life cycle tests.

6.1.2.2 Acute Toxicity

Acute toxicity is defined as toxicant-induced mortality over a short period, generally within 96 hours. Although fish in natural waterways are more likely to be exposed to lower concentrations that might result in chronic or sublethal effects, industrial discharges and spills can temporarily result in lead levels high enough to cause fish kills.

Some of the acute toxicity data for freshwater fauna are summarized in Table 6-3. The lowest LC_{50} value reported in the literature for a freshwater fish was 0.3 mg/l for the three-spined stickleback (Jones 1938). With respect to the intra- and interspecies differences, the LC_{50} values given were derived under a variety of conditions. Such factors as exposure period (varying between 24 and 96 hours), age of test organisms, differences in certain water parameters, and bioassay type (static or flow-through) may account for some of the variation in reported LC_{50} concentrations. For example, values for the bluegill sunfish alone varied from 23.8 mg/l to 442 mg/l; apparently the variation was largely because of the differences in water hardness. Factors contributing to variability in lead toxicity and fish sensitivity are discussed in greater detail in Section 6.1.4.

The observed LC_{50}s for freshwater invertebrates range from 0.124 mg/l for the scud to 71.0 mg/l for the snail. The data are insufficient to determine the relationship between water hardness and lead toxicity for the species studied.

Other freshwater toxicity data observed mortality in adult leopard frogs (Rana pipiens) after a 30-day exposure to 100 µg/l lead nitrate (Kaplan et al. 1967).

6.1.2.3 Effects on Microflora

The toxic effects of lead have been observed in a variety of alga, diatom, and desmid species. Effects similar to those causing sublethal effects in fish have been observed in short-term bioassays at concentrations ranging from 50 µg/l to 28.0 mg/l (U.S. EPA 1980).

TABLE 6-2. CHRONIC AND SUBLETHAL EFFECTS OF LEAD ON FRESHWATER INVERTEBRATES

Species	Concentration (mg/l)	Compound	Experimental Conditions	Effects	Reference
Daphnia Magna	0.012	$Pb(NO_3)_2$	52 mg/l hardness, life cycle	Chronic value	U.S. EPA (1980)
Snail (*Lymnea palustris*)	0.025	$Pb(NO_3)_2$	139 mg/l hardness, life cycle	Chronic value	U.S. EPA (1980)
Isopod (*Asellus meridianus*)	0.1	?	25 mg/l hardness, static 20 days, pH 7.7	Reduced growth	Brown (1976)
Daphnia magna	0.119	$Pb(NO_3)_2$	102 mg/l hardness, life	Chronic value	U.S. EPA (1980)
Daphnia magna	0.128	$Pb(NO_3)_2$	151 mg/l hardness, life cycle	Chronic value	U.S. EPA (1980)
Daphnia magna	0.3	?	43 mg/l hardness 21 days exposure	LC_{50}	Biesinger and Christensen 91972)
Crayfish (*Orconectes virilis*)	0.5-2.0	PbAc	40 days exposure	O_2 consumption slightly reduced compared w/controls	Anderson (1978)
Sludgeworms (*Tubifex tubifex*) (*Limnodrilus hoffmeisteri*)	1.0->60	$Pb(NO_3)_2$	pH 6.5-9.5	Decrease in respiration rate	Whitley (1968), Whitley and Sikora (1970)
Mayfly (*Ephemerella grandis*)	3.5	?	50 mg/l hardness 14 days, pH 7.1	LC_{50}	Nehring (1976)
Mayfly (*Ephemerella grandis*)	16.0	$PbSO_4$	44 mg/l hardness 7 days, static	LC_{50}	Warnick and Bell (1969)
Stonefly (*Pteronarcys californica*)	>19.2	?	50 mg/l hardness 14 days exposure	LC_{50}	Nehring (1976)
Caddisfly (*Hydropsyche betteni*)	32.0	$PbSO_4$	44 mg/l hardness 7 days, static	LC_{50}	Warnick and Bell (1969)
Insect (*Acroneuria lycorias*)	64.0	$PbSO_4$	44 mg/l hardness 14 days, static	LC_{50}	Warnick and Bell (1969)

TABLE 6-3. ACUTE TOXICITY (LC_{50}) OF LEAD TO FRESHWATER FAUNA

Species	Concentration Range (mg/l)	Range of Hardness (mg/l as CaCO3)
Three-spined stickeback (*Gasterosteus aculeatus*)	0.3[a]	?
Rainbow trout (*Salmo gairdneri*)	1.17-542.0	28-353
Fathead minnow (*Pimephales promelas*)	2.4 -482.0	20-360
Brook trout (*Salvelinus fontinalis*)	4.1	44
Guppy (*Poecilia reticulata*)	20.6	20
Bluegill sunfish (*Lepomis macrochirus*)	23.8-442.0	20-360
Goldfish (*Carassius auratus*)	31.5	20
Mosquitofish (*Gambusia affinis*)	240.0	20
Red shiner (*Notropis lutrensis*)	630.0	?
Invertebrates		
Scud (*Gammarus pseudolimnaeus*)	0.124[b]	44-48
Isopod (*Asellus meridianus*)	0.28	25
Daphnia magna	0.45-1.91	45-152
Daphnia hyalina	0.60	66
Copepod (*Eudiaptomus padanus*)	4.0	66
Copepod (*Cyclops abyssorum*)	5.5	66
Snail (*Lymnaea emarginata*)	14.0	154
Sludgeworm (*Tubifex tubifex*)	27.5-49.0	?
Rotifer (*Philodina acuticornis*)	40.8	25
Snail (*Goniobasis livescens*)	71.0	154

[a]Jones (1938).

[b]Spehar *et al*. (1978).

Source: U.S. EPA (1980), except as otherwise noted.

6.1.3 Marine Organisms

Information on lead toxicosis in marine finfish is limited to two species, and only one acute toxicity study was available. Weis and Weis (1976) exposed mummichog (Fundulus heteroclitus) with partially amputated caudal fins to various lead nitrate solutions to determine the effect of lead on fin regeneration. A concentration of 0.1 mg/l was found to stimulate fin growth, while 1.0 mg/l lead significantly inhibited regeneration.

Dilling and co-workers (1926) observed growth inhibition in juvenile plaice (Pleuronectes platessa) exposed to 2 mg/l colloidal lead as $Pb(OH)_2$. Dorfman (1977) reported a 96-hour LC_{50} of 315 mg/l for the mummichog in static bioassays.

Data on lead toxicity in marine invertebrates are much more abundant. Sublethal effects include delayed larval development and reduced growth rate, which have been observed in concentrations as low as 50 µg/l in the mud crab (Rhithropanopeus harisii) (Benijts-Claus and Benijts 1975). Chronic LC_{50} values have been reported in the range of 1.0-30.0 mg/l, with the lower concentration determined for the polychaete (Ophryotrocha labronica) by Brown and Ahnsullah (1971).

Information on acute toxicity is summarized in Table 6-4. The lowest reported LC_{50} is 0.72 mg/l for the hardshell clam larvae.

6.1.4 Factors Affecting the Aquatic Toxicity of Lead

Numerous variables in a natural aquatic environment influence the availability and toxicity of lead to biota. The hardness of the water, dissolved solids, oxygen content, pH, and interactions between heavy metals modify the toxicity of lead to varying degrees.

Among these parameters, water hardness appears to be the most significant. The negative correlation between lead toxicity and hardness has been confirmed in numerous laboratory studies. The most illustrative example of this relationship is found in the results of a study by Pickering and Henderson (1966). In static bioassays on fathead minnows in water with either 20 mg/l or 360 mg/l hardness, the authors calculated 96-hour LC_{50}s of 5.58 mg/l and 482.0 mg/l lead, respectively. Similar results were obtained in experiments with the bluegill. Davies et al. (1976) found that the 96-hour LC_{50} for rainbow trout varied by a factor of >400 in a water hardness range of 28-353 mg/l; however, tests in soft water were flow-through, while those in hard water were static. Miller and Landesman (1978) reported that magnesium ions (component of water hardness) have suppressive effects on the toxicity of lead to frog (Xenopus sp.) embryos.

The presence of other dissolved solids may also alter the toxicity of lead to aquatic organisms. Wong et al. (1978) recommend the use of

TABLE 6-4. ACUTE TOXICITY (LC_{50}) OF LEAD TO MARINE INVERTEBRATES

Species	LC_{50} Concentration (mg/l)	Experimental Conditions
Hard-shell clam, larvae (*Mercenaria mercenaria*)	0.72-0.80	Static, nominal, 48 hours
Polychaete, trochophore (*Capitella capitata*)	1.2	Static, nominal, 96 hours
Eastern oyster, larvae (*Crassostrea virginica*)	2.2 - 3.6	Static, nominal, 48 hours
Isopod (*Jaera albifrons*)	10[a]	Static, 24 hours, 5°C, 1% seawater
Isopod (*Jaera nordmanni*)	10[a]	Static, 36 hours, 5°C, 1% seawater
Softshell clam (*Mya arenaria*)	27.0	Static, nominal, 96 hours, salinity 30°/oo, pH 7.95
Prawn (*Pandalus montagui*)	375[b]	Static, renewal, 48 hours, 15°C
Cockle (*Cardium edule*)	>500[b]	Static, renewal, 48 hours, 15°C

[a]Jones (1975).

[b]Portmann and Wilson (1971).

Source: U.S. EPA (1980), except as otherwise noted.

phosphate-free conditions in laboratory bioassays on algae, because phosphates also decrease lead toxicity. The assessment of Dorfman (1977) on the influence of varying salinity on lead toxicity to the mummichog proved inconclusive.

In natural waters, lead occurs in free ionic forms, in complexed forms with organic and inorganic molecules, and in association with particulate matter (Wong et al. 1978) (see Section 4.3). The consensus of the literature surveyed is that the free ion, with the exception of certain organolead compounds, is the form of lead most often associated with toxicity. Davies et al. (1976) found that when lead was added to hard water, precipitates of lead salts were formed, probably as carbonates and hydroxides. Such precipitates (in addition to chelates, organic complexes, and lead absorbed to particulates) are apparently less available to aquatic organisms for uptake and metabolism. Barber and Ryther (1969) found that the addition of the chelating agent ethylenediaminetetraacetic acid (EDTA) decreased or completely masked lead toxicity. On the other hand, small amounts of lead may be methylated to more available forms in sediments, the amounts produced are probably too small to contribute significantly to overall aquatic lead toxicity.

In the case of water hardness, Zitco (1976) has suggested another mechanism for lead toxicity suppression. Base metals, such as calcium or magnesium, might compete with heavy metals, such as lead, for active sites on the surface membranes of organisms. In hard water, the concentrations of these metals would be greater than lead, and any such sites would quickly be saturated, and hence unavailable to lead. Matthiesson and Brafield (1977) have suggested that the base metals may protect the biochemical processes with which zinc interferes; this hypothesis could also be applied to lead, assuming certain similar characteristics between the two heavy metals.

Dissolved oxygen level and pH are two parameters that are normally controlled in laboratory studies. In a static bioassay on rainbow trout, Lloyd (1961) found that the toxicity of lead increased with a decrease in oxygen concentration. Tubificid worms were more sensitive to lead at pHs of 6.5 and 8.5, even though they showed more resistance at pH 7.5 (Whitley 1968). In both cases, the direct effects of these parameters may have lowered the organisms' tolerance to lead. However, the toxicity of lead is enhanced in acidic waters, as the chemical equilibrium shifts in the direction of free ion formation.

Often, other heavy metals are found in waters polluted with lead from mining runoff or atmospheric emissions. Copper, zinc, mercury, and cadmium are toxic to varying degrees, and have some similar effects on aquatic life. In combination, heavy metals may either have a synergistic or antagonistic interaction with respect to toxicity. Gray and Ventilla (1973) combined zinc, mercury, and lead ions in solution to determine the effect on Cristigera protozoa. The growth rate was inhibited by 67.2%, compared with the theoretical reduction of 37.4% calculated from combining the individual effects of the metals (i.e., a synergistic effect was observed).

Other studies have also examined heavy metal interactions with lead. Pietilainen (1975) indicates that cadmium increased the toxicity of lead to phytoplankton communities if the cadmium concentration exceeded that of lead. This effect was markedly lessened when the lead concentration was higher. Benijts-Claus and Benijts (1975) found that both lead and zinc had a significant adverse impact on the development of the mud crab, Rhithropanopeus harrisi. In certain combinations, however, zinc apparently suppressed the more toxic lead, and larval growth actually accelerated. In toxicity tests with zebrafish, Ozoh (1979a) found that copper and lead at the highest concentrations used (72 μg/l) were mutually suppressive with respect to growth inhibition effects.

Long-term exposure may affect the resistance of an aquatic organism. Certain data show that fish can become acclimated to some chemicals so that their tolerance is increased, particularly if previous generations were exposed. Although no multi-generational studies were available for lead, Davies et al. (1976) reported that sensitivity actually decreased in rainbow trout when exposed as eggs; specimens exposed after hatching were more resistant.

6.1.5 Terrestrial Organisms

6.1.5.1 Animals

Most of the information on lead toxicity to wildlife is concerned with effects on waterfowl because of the concern for the extensive lead poisoning as a result of the consumption of spent lead shot (see Section 6.2). Other studies provide data on lead toxicosis in doves and gallinaceous birds. Most of the studies on lead in wildlife, other than waterfowl, deal primarily with tissue residues, because lead poisoning has seldom been observed in the field (see Section 4.2).

The symptoms generally associated with lead poisoning in waterfowl are lethargy, anorexia, weakness, flaccid paralysis, emaciation, anemia, greenish diarrhea, impaction of the proventriculus, and distention of the gallbladder (Clemens et al. 1975). Most toxicity tests with birds involve feeding the birds a certain number and type (size) of shot. Lead shot is particularly harmful to birds because the gizzard grinds the accumulated shot into a fine powder that is easily absorbed in the acidic conditions of the digestive tract. In mammals, on the other hand, the shot normally passes through the digestive system, with only superficial contact and absorption (Hartung 1973).

Of the numerous experiments that have been conducted to assess the toxicity of lead in birds, a representative sample is presented below. Further discussion is contained in Forbes and Sanderson (1978). Irby et al. (1967) fed game farm mallards (Anas platyrgynchos) with 8, No. 6 lead shot each; the birds survived an average of 17 days after the dosage, and lost an average of 37% of their initial body weight. Seven captive Canada geese (Branta canadensis) dosed with 10 or more lead pellets died within 23 days (mean of 10 days), while two other geese fed 5 pellets survived

for an average of 55.5 days (Cook and Trainer 1966). Damron and Wilson (1975) fed juvenile bobwhites (Colinus virgianus) an average of 5 lead shot; none of the birds showed any ill effects over 6 weeks, which indicates a much greater tolerance for lead than mallards. Morgan et al. (1975) reported that 500 mg/kg dietary lead (continuous exposure) produced growth inhibition and anemia in Japanese quail (Coturnix japonica), as well as a delay in the sexual development of males.

Evidence illustrates that diet plays a considerable role in the degree to which lead affects various waterfowl species. Lead pellets in mallards on a high fiber diet lost 46% of their initial weight within 20 days after dosing, while pellets in ducks fed a low fiber diet lost 62% of their initial weight during the same period (Clemens et al. 1974). Jordan and Bellrose (1951) found that the effects of dietary lead in mallards were most severe in birds fed a diet of whole corn. Effects were less pronounced when the mallards were fed smaller seeds such as wheat, rice, and millet; when a grain diet was supplemented with aquatic greens, toxicosis was further mitigated. Holmes (1975) reported that the addition of egg albumen, oyster shell, calcium carbonate, and phosphorous to mallard feed reduced the degree of lead toxicity by 50% over a straight corn diet.

Of the waterfowl species, the mallard appears to be the most susceptible to lead poisoning. Bellrose (1959) observed three species of ducks feeding in flooded chufa beds. Mallards browsed on bottom tubers, pintail (Anas acuta) ate both bottom tubers and floating seeds, while green-winged teal (A. cardinensis) limited their diet to floating seeds only. The mallards had a mortality rate (0.97%) ten times that of pintail, while no lead-poisoned teal were found. The author attributed the differential mortality rates to a higher incidence of lead in the gizzards of bottom-feeding ducks, which mistake lead pellets for aquatic seeds or grit. Diving species, such as redhead (Aythya americana), ring-necked duck (A. collaris), and canvasback (A. valisneria) are also thought to be more susceptible to lead poisoning, although mallard mortality is usually higher because of their preference for large seeds (Bellrose 1964).

6.1.5.2 Plants

Abundant data are available on the effects of lead on both wild and crop plants, partly because of the concern for the human consumption of lead. As with animals, lead has not been shown to be essential for plant growth. Even low levels may cause growth irregularities.

In a few instances, low concentrations of lead have been found to stimulate the growth of various crop plants, including barley, corn, wheat, buckwheat and sugar. However, toxicosis is more often observed. Cultivated grasses exhibit an extremely wide range of sensitivities; for example, in one experiment, maize (Zea mays) and rye (Secale cereale) showed no signs of toxicosis at lead acetate concentrations up to 100 mg/l. On the other hand, barley, oats, and particularly wheat were damaged at levels well below 100 mg/l (Holl and Hampp 1975).

Bean (Phaseolus vulgaris) plants were exposed to lead at concentrations of 33-33,000 mg/l. At the lower limit, growth was stimulated, while no effects were apparent at 330 mg/l. At 33,000 mg/l, the plants were visibly damaged within 30 minutes, and died within 24 hours (Holl and Hampp 1975). Differentiating young tissues tended to be more sensitive to lead than mature tissues.

Other toxicity data indicate that lead affects the photosynthetic process (Miles et al. 1972). Lead chloride concentrations as low as 14 mg/l reduced chlorophyll synthesis significantly in oat seedlings; moreover, lead levels up to 140 mg/l inhibited chlorophyll b bynthesis to a greater extent than the chlorophyll a synthesis (Hamp and Lendzian 1974).

Airborne lead is another potential exposure pathway that may have sublethal effects on plants. Bouganvillaea plants exposed to ~7 ppm tetraethyl lead vapor at 25°C for 3 days abscised an average of 14% of their leaves. Total recovery was observed 14 days after the exposure period (Siegal et al. 1973). Davis and Barnes (1973) report that 41-410 mg/l lead chloride in watering solutions reduced the stem and root dry weights and height of loblolly pine (Pinus taeda) and red maple (Acer rubrum) seedlings in two forest soils. At higher concentrations, root systems also became reduced and blackened.

Lead toxicity to plants depends on the chemical composition of the soil. As with aquatic organisms, the presence of calcium, phosphates, and other ions inhibit the effects of lead on plants. Acidic soils increase plant susceptibility to lead, as does increased soil temperature. The levels of organic nutrients and soluble silicon may also affect lead uptake in and availability to plants (NSF 1977).

6.1.6 Summary

Lead may cause acute toxicosis to aquatic organisms at concentrations as low as 0.3 mg/l. Sublethal effects have been observed in rainbow trout in lead concentrations of 7.6 μg/l. It appears as if freshwater invertebrates have a narrower range of lead sensitivities than fish, although this result could be a function of the test species chosen. Aquatic microflora have exhibited toxic effects in lead levels between 0.05 mg/l and 28.0 mg/l. The mummichog was the only marine finfish tested for acute toxicosis; the reported 96-hour LC_{50} was 315 mg/l. Marine invertebrates exhibited toxicosis (acute, chronic, and sublethal) in lead concentrations between 0.05 mg/l and >500 mg/l.

Several water parameters strongly influence the aquatic toxicity of lead. When the water hardness was raised from 28-353 mg/l, the 96-hour LC_{50} for rainbow trout increased by a factor of >400. Similar toxicity-suppressing effects of water hardness were observed for fathead minnow and bluegill. The presence of phosphates, chelating agents, and nutrients has also been observed to reduce lead toxicity. Water with a low pH shifts the equilibrium of aqueous lead toward free ion formation, which increases toxicity. Heavy metal interactions with lead may be either

synergistic or inhibitory, depending on the specific combination. Relatively little information exists on the comparative toxicity of various forms of lead.

With the consideration of all of these variables, a range of lead concentrations can be categorized for toxicity as shown below. These ranges are only a very general guide for the assessment of the potential ecological impact of a given lead concentration in an aquatic environment.

- 10 μg/l — Life-cycle toxicity test in soft water produced abnormal development in rainbow trout. All other species tested are unaffected.
- 10-100 μg/l — Sublethal effects, such as tail and fin degeneration, reported for several freshwater fish species in soft water, as well as growth inhibition in sensitive freshwater algae. Chronic toxicity reported in soft water and are based on the effects on early life stages in fish and life cycle test in Daphnia.
- 100-1000 μg/l — Sublethal toxicosis in a variety of freshwater fish and invertebrates in soft and moderately hard water. Acute effects observed in stickleback and some invertebrates. Numerous species of freshwater algae adversely affected, as well as more sensitive marine microflora.
- 1.0-10.0 mg/l — Acute toxicity level for several freshwater fish and invertebrate species in soft water and for sensitive marine invertebrates. Algicidal for some marine algae.
- 10.0-100 mg/l — Acute effects observed in a variety of freshwater fish and in most invertebrates tested. All algae species tested sensitive in this range.
- 100 mg/l — Only the most resistant species (e.g. red shiner, mosquitofish, cockle) are able to survive, even for short periods, in this range of concentrations. Tolerance to such levels would be increased in hard water.

To protect freshwater aquatic life, the U.S. EPA (1980) has established a function of the criterion for lead (as total recoverable lead) hardness, i.e., $e^{(2.35\,[\ln \text{hardness}]-9.48)}$ as a 24-hour average, and the concentration (in μg/l) should not exceed $e^{(1.22\,[\ln \text{hardness}]-0.47)}$. At hardness of 50, 100, and 200 mg/l as $CaCO_3$, the criteria are 0.75, 3.8, and 28 μg/l, respectively as a 24-hour average and the respective maxima would be 74, 170, and 140 μg/l.

Data on lead toxicity in waterfowl, doves, and gallinaceous birds are plentiful, and indicate that the ingestion of spent lead shot can be lethal. Mallards and diving ducks are particularly susceptible, largely as a result of feeding habits rather than physiological vulnerability.

Lead toxicosis has been observed in terrestrial plants from lead concentrations ranging from 0.005-33,000 mg/l in culture or watering solutions. Effects include growth stimulation (at very low levels), growth inhibition, leaf yellowing, abscission, inhibition of mitosis and chlorophyll synthesis, loss of turgor pressure, and death. Lead toxicity to plants is mitigated by factors similar to those in aquatic environments, i.e., high pH, high calcium and phosphate levels; in addition, the temperature, organic nutrient and soluble silicon content of the soil influence the toxicity of lead.

6.2 EXPOSURE TO BIOTA

6.2.1 Introduction

Lead is a pervasive element that appears at various concentrations in air, water, and soil systems, as well as in the tissues of organisms (see Section 4.2). In this section, lead exposure levels to aquatic and terrestrial biota are discussed, with special focus on factors that lead to acute and chronic exposure. For aquatic life, the following topics are reviewed:

- The various pathways of exposure in fish, invertebrates, and algae;
- Chemical parameters in the aquatic environment that may affect the degree of exposure to lead and uptake of the metal by biota;
- Areas of the country in which relatively high lead concentrations were reported in 1979; and
- Recent incidents of elevated lead exposure in various water bodies.

Data on lead exposure to terrestrial mammals are limited mainly to comparative residue studies on rodents in urban and rural locations. A considerable amount of information on lead in waterfowl is available, as a result of the widespread problem of exposure to spent lead shot. Habitat conditions and other factors, such as hunting pressure, are described for their effects on the availability of lead shot for ingestion by waterfowl.

Terrestrial plants are exposed to elevated lead concentrations primarily in the vicinity of anthropogenic sources. The chemical properties of the soil are crucial in determining the availability of lead.

6.2.2 Aquatic Organisms

6.2.2.1 Pathways of Exposure

The three general pathways of exposure in aquatic fauna consisting of diet, direct absorption from the water, and contact (dermal or dietary) with sediment. As with other heavy metals, relatively few studies attempt to evaluate the relevance of each of these pathways to different species. Moreover, the available data provide conflicting evidence concerning routes of uptake, particularly with fish.

Lead residues in many invertebrates reflect levels found in sediment; however, it remains unclear whether the lead is strongly concentrated from water or absorbed from sediment. Tubificid worms collected by Mathis and Cummings (1973) reported lead residues equivalent to sediment concentrations, while aqueous lead levels were four orders of magnitude lower.

McNurney and co-workers (1977) measured residues in a variety of freshwater stream organisms and found a positive correlation between lead burden and the degree of sediment contact. Burrowing oligochaetes and insect larvae had the highest lead residues, followed by fauna (pelecypods, crayfish, darters) subsisting at the sediment-water interface. Lower residues were detected in bottom-feeding omnivorous fish, while carnivores, which have very little contact with settled or suspended sediments, had the lowest body burdens. The authors hypothesized that food was the main source of lead in carnivores; however, they were uncertain of the dominant exposure pathway for the lower organisms.

Some circumstantial evidence demonstrates that suspended sediments and food particles are the primary source of lead to filter-feeders. Bryan (1973) attributed seasonal changes in lead tissue levels in two scallop species to variations in food supply in the form of suspended matters. Bryan and Uysal (1978) concluded that the ingestion of suspended sediments was the main pathway for lead uptake in the clam (*Scrobicularia plana*), because lead accumulated primarily in the digestive gland.

The relative importance of sediment and waterborne lead has also been examined for fish. In long-term tests with rainbow trout (*Salmo gairdneri*), Hodson *et al*. (1978) added lead in various concentrations to the diet. With doses up to 950 μg/day, the fish excreted virtually all ingested lead. At doses between 1935 μg/day and 3160 μg/day, approximately 50% was excreted; however, because no bioaccumulation was observed, the authors assumed some experimental error in the analysis of excreta at higher doses.

Such highly efficient depuration, at least at low concentrations, could explain why the predatory fish collected by NcNurney *et al*. (1977)

had low lead residues despite the high body burdens in lower-order fauna. On the other hand, Hardisty et al. (1974) discerned a distinct correlation between tissue concentrations and crustacean consumption in seven teleost species. Schell and Barnes (1974) found that lead residues in the northern squawfish (Ptychocheilus oregonensis) decreased with age. One explanation was that younger fish ate zooplankton, which are high in lead, while older fish preyed on various fish species with low lead burdens. Butterworth et al. (1972) reported a steady increase in lead residues with ascending trophic levels in an estuary. However, most of the evidence does not indicate significant biomagnification of lead in food chains (Wong et al. 1978).

In contrast to their work with exposure via ingestion, Hodson and co-workers (1978) measured tissue concentrations in rainbow trout after various periods of exposure to lead and found a linear relationship to aqueous lead concentrations. High aqueous lead levels resulted in elevated residues in the gills and kidneys, which, in turn, reflected the direct contact of gills with lead-contaminated water and the possible excretion of lead via urine. Thus, it is apparent that both ingestion of lead in food sources, as well as gill contact with water can result in exposure to lead.

6.2.2.2 Environmental Factors Affecting Lead Exposure and Uptake

The variables that alter the toxicity of lead to aquatic organisms may influence the availability of lead for uptake. Acidic water shifts the equilibrium of soluble lead toward the more toxic free ion form. Pumpkinseed sunfish (Lepomis gibbosus) exposed to ^{203}Pb at pH 6.0 accumulated three times as much lead as they did at pH 7.5 (Merlini and Pozzi 1977). These results suggest that free ionic lead is the form most available for uptake.

Water hardness levels also affect the solubility of lead, and hence its availability for absorption. Varanasi and Markey (1977) studied lead accumulation by rainbow trout in waters of varying hardness. Calcium (as $CaCO_3$) had a significant mitigative effect on lead uptake rates; however, the authors hypothesized that lead might, in turn, inhibit calcium absorption required for bone growth by the trout.

The presence of other metals may affect lead uptake. Luoma and Bryan (1978) indicated a possible antagonistic relationship between lead and iron. Lead residues in the clam (Scrobicularia plana) were inversely proportional to sediment concentrations of amorphic iron in 17 estuarine locations. The strong negative correlation between these two variables led the authors to speculate that the Pb:Fe ratio in sediments may be a good indicator of biologically available lead.

Brown (1977) studied lead uptake bioaccumulation experiments on three groups of isopods (Asellus meridianus) collected from different areas. The most tolerant group accumulated the most lead, and thus probably had a greater ability to store and detoxify the metal. The

tolerant group also had much higher levels of sulfur, which suggests that the detoxification mechanism was a possible sulfhydryl binding of lead. Thus, as with toxicity, various factors appear to affect lead uptake and hence exposure. Such factors include pH, water hardness, inhibition by other metals, and acclimation.

6.2.2.3 Monitoring Data

The most complete data base for measured levels of ambient aqueous lead in the U.S. river basins was found in STORET. Major basin data are analyzed in Section 4.2. Table 6-5 lists minor basins with mean total lead levels >50 µg/l, which represents a level at which sublethal effects have been observed, although some chronic values of <50 µg/l have been reported. Lead levels of ~100 µg/l may be lethal to some species, depending on the duration of exposure and various chemical factors. These values are means of positive values only; thus, they reflect a somewhat higher mean than if all the values had been included. Of the basins listed, those with water hardness usually <50 mg/l as $CaCO_3$ are also identified, because soft water effectively increases lead exposure levels.

Two minor basins had mean lead levels >100 µg/l. The Catawba-Wateref Basin in the Southeast had an average lead concentration of 124.5 µg/l, with 50.0% of the positive observations >100 µg/l. The Kanawha River in the Ohio River Valley had a mean lead level of 148.7 µg/l, with 7.5% of the measurements >100 µg/l. In this case, the mean was most likely skewed by a few extremely high measurements, including a maximum of 7000 µg/l. Other minor basins with a relatively high proportion of boservations (>20%) >100 µg/l were the James River in the North Atlantic, the Fox River-Wolf Creek drainage area near Lake Michigan and the Kootenai River in the Northwest.

Reports in the literature of elevated lead concentrations that result from a specific source are uncommon; thus, little opportunity is provided to assess the effects of acute exposure in the field. Two reports of fish kills related to lead concentrations were found. In one, in 1970, the total lead concentration was about 1000 µg/l at some type of industrial operation in Glover, Missouri. The other occurred at an old smelter site in Henrietta, Oklahoma in 1973. The reported lead concentration was 23,600 µg/l (U.S. EPA 1979).

6.2.3 Terrestrial Organisms

6.2.3.1 Mammals

Little information is available on lead exposure levels to mammals probably because of the lack of observations of lead poisoning in field situations. Most of the available studies are primarily concerned with correlations between proximity to anthropogenic sources of lead and residues in wild specimens.

TABLE 6-5. MEASUREMENTS OF TOTAL LEAD CONCENTRATIONS IN U.S. MINOR RIVER BASINS, 1979[a]

River Basin Major/Minor	River Basin Name	Mean Pb >50 µg/l	≥10% of Pb >100 µg/l	≥50% of Hardness Measurements <50 mg/l[b]
1/33	Lower Hudson -- NY Metropolitan Area	*		
1/34	New Jersey Coast	*		*
2/3	Delaware River -- Zone 1	*		
2/16	James River	*	*	*
3/7	Yadkin-Pee Dee-Lower Pee Dee River	*	*	*
3/8	Catawba-Wateref, Congaree, Santee-Cooper Res.	*	*	*
3/9	Edisto-Combahef River		*	*
3/13	Savannah River	*	*	*
3/24	Tampa Bay Area		*	
3/28	Lower Florida Area		*	
5/3	Beaber River	*	*	
5/7	Kanawha River	*		
5/9	Big Sandy River	*		
8/24	Green Bay Western Shore	*		
8/25	Fox River -- Wolf Creek	*	*	
9/8	Lower Platte River from North Platte	*	*	
9/12	Lower Missouri River from Niobrara River	*	*	
10/8	North Canadian River	*	*	
11/1	Lower Colorado River	*	*	
11/2	Middle Colorado River -- San Juan River	*	*	
11/4	Gila River		*	
11/5	Green River	*	*	
12/4	Brazos River		*	
13/1	Kootenai River	*	*	
13/3	Spokane River		*	*
14/7	Santa Ana River		*	
15/1	Northwestern Lahontan	*	*	
15/7	Great Salt Lake	*	*	

[a]Basins with ≥ 10 measurements in 1979.

[b]1978 data.

Note: There are 307 minor river basins in the continental U.S.

Source: U.S. EPA (1980).

Williamson and Evans (1972) examined the lead burdens in a variety of small mammals, and found no evidence of biomagnification in food chains. Though carnivorous shrews generally had higher residues than herbivorous mice, both groups contained less lead than their respective food sources. Shrews and mice collected close to roads had higher lead burdens than specimens collected at distances from roads.

In a study of lead residues in voles and field mice in England, Jeffries and French (1972) found similar results. Body burdens ranged from an average of 4.19 μg/g dry weight on woodland and arable sites to 5.98 μg/g near minor roads to a maximum of 7.00 μg/g near a busy highway. Mouw et al. (1975) found that lead residues in the bones and kidneys of urban rats were twenty times greater than those in rural rats, while concentrations in feces were four times higher. Rolfe and Haney (1975) analyzed eight species of small mammals in Illinois to determine the effects of proximity to roadways. Except for two species, animals trapped along medium-use roads had tissue levels intermediate to those captured along high- and low-use roads. These authors detected no seasonal variations in tissue concentrations of lead. Thus, evidence of elevated exposure to lead in terrestrial mammals has been observed in the vicinity of highways.

6.2.3.2 Birds

There are several possible pathways for lead exposure to birds. Bagley and Locke (1967) indicate that all birds are regularly exposed to sublethal levels of lead in their food. Atmospheric lead fallout (originating from industrial and auto emissions) is a potential exposure route; however, more data must be acquired on this potential exposure route. Still, Forbes and Sanderson (1978) have recorded several reports of waterfowl mortality caused by runoff from mine wastes and ingestion of mine tailings.

One source, however, appears to account for nearly all cases of lead toxicosis in birds. Each year, over 3000 tons of lead shot are deposited by waterfowl hunters in marshes, lakes, and fields of the United States, while as many as 122,000 lead pellets per acre have been found in some areas (U.S. FWS 1976). Numerous species ingest the spent shot, mistaking it for grit or plant seeds, or eating it by accident as they forage.

Many instances of lead-related waterfowl kills in large numbers have been reported; written accounts date from as early as 1894 (Grinnell 1894). Quortroup and Shillinger (1941) concluded that lead poisoning was the third most important (8.6% of the total) cause of death in 3000 specimens taken from various western lakes. Perhaps, because of the increased monitoring of waterfowl populations, such incidents have been reported more frequently over the last 30 years. In the most comprehensive survey, Bellrose (1959) reported 34 waterfowl die-offs in the United

States, between 1937 and 1957, with mortalities ranging from 100 to 16,000 in each event. Die-offs occurred in each of the four major flyways (Atlantic, Mississippi, Central, and Pacific); the greatest number of casualties occurred in the Mississippi flyway. However, Jordan and Bellrose (1951) believe that small, day-to-day losses constitute a larger portion of total lead-related mortalities than spectacular die-offs that receive more publicity.

Many factors may influence lead exposure levels to waterfowl. First, the hunting pressure determines how much lead shot will be scattered over a given area. As a measure of hunting pressure, the U.S. FWS (1976) estimated waterfowl hunter days in each of the major flyways for the period 1952-71. The Mississippi flyway had 40-45% of the total hunter days, the Central flyway had >20%, and the Atlantic and Pacific flyways each had <20% of the hunting activity.

The characteristics of lake and marsh bottoms also affect exposure levels; for example, in soft lake beds the lead shot sinks into sediment, which renders it relatively unavailable for consumption. Bellrose (1959) found that in such areas, little of the exposed shot is carried over from one year to the next; therefore, more lead shot would probably be available during and soon after the hunting season. On hard bottoms, however, spent shot may simply accumulate on a yearly basis. The ice cover and prevailing water levels also affect the degree of exposure, because they determine the size of the area in which waterfowl may come into contact with spent shot (Forbes and Sanderson 1978).

Dietary factors are important in lead exposure to waterfowl. Birds that consume mostly large quantities of corn are generally more susceptible to lead poisoning (Bellrose 1964). Various toxicity studies (see Section 6.1.5.1) have shown that diets high in calcium, phosphorous, and other nutrients led to decreased sensitivity in ducks. The addition of smaller grain seeds and aquatic plants to the diet mitigate the toxic effects of lead to some degree (Jordan and Bellrose 1951). Moreover, birds consuming large quantities of food are apparently less likely to experience lead poisoning (Bellrose 1964).

Because the various waterfowl have different feeding habits, some species naturally have greater levels of exposure. Diving ducks, on the whole, are more likely to ingest lead pellets because they feed on seeds, tubers, and rootstocks at lake bottoms. Lead pellets may be mistaken for certain aquatic seeds because of their similar size and shape; thus, they are consumed. Bellrose (1959) examined the gizzard contents of waterfowl bagged by hunters; he found the highest incidence of lead shot in diving ducks. Between 12 and 14% of all canvasback, redhead, lesser scaup, and ring-necked ducks had ingested lead pellets that had remained in the gizzard. In addition, birds that prefer corn, such as Canada geese, mallards, pintail and other species of dabbling ducks, ingest more lead pellets than most other types of waterfowl.

Lead poisoning has been reported in other types of birds; however, it is, in general, a rare phenomenon. Calvert (1876) and Holland (1882) reported pheasants ill from ingesting lead shot. Of 1977 gizzards of Gruiformes (cranes and rails), only 49 had lead shot, and nearly one-half were from sora rails. Jones (1939) doubted that cranes or limpkins ever ingested spent shot. Campbell (1950) reported the finding of a dead scaled quail with 13 lead pellets in its crop. In 1949 gizzards from doves taken by hunters, Lewis and Legler (1968) found a 1% incidence of lead pellets. A University of Minnesota study cited by the U.S. FWS (1980) found that one-fourth of the castings beneath bald eagle roosting trees contained lead shot. Several deaths from lead poisoning in bald eagles have been reported; mortality is more likely if the shot is retained in the digestive tract rather than cast (U.S. FWS 1980).

Lead accumulates in various bird tissues, most notably in bones. According to the U.S. FWS (1976), low-level chronic exposure to lead (as in diet) results in comparatively high levels in bones, lower levels in liver and kidney, and still lower levels in heart, lung, muscle, and brain. High levels in liver and kidney as well as bone are indicative of recent acute exposure. One experiment cited by the U.S. FWS (1976) demonstrated that the ingestion of a single lead pellet can produce high lead concentrations in the wing bones of ducks. Of the 28 species of birds examined by Bagley and Locke (1967), tissues from the osprey, an aquatic predator, had the highest lead burdens.

Several studies have been conducted to determine the effect of urban versus rural environments on lead residues in birds. Tansy and Roth (1970) found that pigeons in urban areas had higher lead residues in bone, feathers, and kidneys than their rural counterparts, although concentrations in blood were similar. Ohi et al. (1974) also compared rural with urban pigeons; they found the greatest differences in the femur (bone), ALA-D enzyme activity, and blood. Getz et al. (1977) measured lead residues in five passerine (songbird) species, and found that urban specimens had uniformly higher burdens in feathers, butt, liver, lung, kidney, bone, and muscle.

6.2.3.3 Terrestrial Plants

The potential for exposure of terrestrial flora to lead increases in areas of high lead particulate deposition. Sources contributing high levels to the atmosphere include lead smelters, highways, various stack-discharging industries, and urban areas in general. Mining areas have high associated levels of lead in soil. Section 4.2 (Tables 4-4 to 4-6) presents concentrations in air, soil, and vegetation associated with specific sources. Lead may be absorbed from deposition directly on leaves and stems or taken up from soil contaminated by particulate fall-out. Higher exposure levels are associated with direct deposition; however, cuticle barriers and washing off during rain or wind activities reduce the concentrations taken up by plants (see Section 4.3). Soil-originated exposure levels are usually lower but more persistent as a result of immobilization of lead in the upper soil profile. Once lead is taken up into vegetation,

it is likely to persist, and will recycle into the system with the carbon cycle.

Environmental factors influencing plant uptake of lead from soil and subsequent exposure include pH, presence of other nutrients and metals, and species acclimation. Low soil pH is favorable to uptake (MacLean et al. 1969); thus, standard agricultural practices (e.g. liming of soil) may decrease accumulation. The presence of calcium ions inhibits lead uptake (Wilkins 1957), and phosphate deficient plants are more susceptible to lead effects than normal plants (Koeppe and Miller 1970). In addition, Peterson (1978) has observed lead-tolerant ecotypes for several floral species that have had long-term exposure to the metal.

6.2.3.4 Summary

There are several possible pathways of lead exposure to aquatic fauna, including water, diet, and sediment. Most likely, contact with sediment is an important exposure route, particularly for benthic invertebrates and filter feeders. However, the assertion that sediment is the most important pathway for these organisms is based largely on circumstantial evidence; such conclusions must be verified by experiments that isolate the effects of water-borne and sediment-borne lead. Data on lead uptake by fish are scarce, although there is evidence for uptake from water and diet. However, sediment remains a potential direct or indirect source of large quantities of lead for invertebrates and vertebrates alike.

Lead exposure to aquatic organisms is enhanced by soft, acidic waters, and the presence of lead sources, such as industrial or automotive emissions, lead mines, and urban areas in general.

For minor river basins, STORET monitoring data indicate that mean total lead concentrations were generally <50 μg/l in 1979. Furthermore, because biologically available lead comprises only a fraction of total lead, exposure levels are normally well below 50 μg/l. Minor basins with relatively high concentrations are listed in Table 6-5. Only a few instances of fish kills caused by lead have been reported; however, elevated residues have been observed in areas with high aqueous lead levels.

Information on mammals is limited mainly to studies of rodents with varying degrees of exposure to urban and automotive lead. One study reported no evidence of lead biomagnification from food sources for shrews and mice.

Over the last century, there have been numerous reports of waterfowl mortality as a result of the ingestion of spent lead shot. Waterfowl kills are more prevalent in areas under heavy hunting pressure (such as the Mississippi flyway) and in lakes and marshes with hard bottoms, where lead shot can accumulate. Exposure is also greater for diving ducks, which have access to lead on lake bottoms, and for species that eat mainly large seeds, such as corn. Lead poisoning is relatively rare

in other types of birds. However, species dwelling in urban areas (e.g., pigeons) are reported to have higher lead residues than their rural counterparts. Lead accumulates most strongly in the bones of all species examined.

Lead exposure to terrestrial plants increases above background levels in areas of high lead particulate deposition. As for other organisms, sources include lead smelters, automobiles, various industries, and urban areas in general. Lead may be absorbed either through leaf or root tissues; direct deposition on leaves comprises a larger proportion of lead exposure than lead in soil; however, cuticle barriers and washing off will reduce absorption through leaves. Soils with low pH and calcium content are conducive to uptake through the roots. In natural conditions, lead poisoning is extremely rare; however, elevated residues in crop plants may increase human exposure levels.

The significance of high, but non-toxic, levels of lead in plants in this exposure assessment should be stressed. Human exposure through the ingestion of contaminated crops by humans can be an important pathway, which is described in Section 5.2. Non-human biota ingesting vegetation may be similarly exposed.

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7.0 RISK CONSIDERATIONS

7.1 HUMANS

7.1.1 Introduction

The preceding sections presented a discussion of the pathways of exposure and effects of lead on humans. This section identifies the subpopulations at risk. Section 5.2, which makes numerous assumptions about concentrations and consumption, describes exposure scenarios for certain subpopulations. Although this approach is useful for determining the relative importance of exposure routes for different subpopulations, it is not generally useful for evaluating potential risk. Because most of the observations of effects are correlated with levels of lead in the blood, measurements of such levels are currently the most appropriate way to evaluate potential risk.

Given a large enough sample population, levels of lead in the blood appear to show a log-normal distribution. In certain cases, such as in urban and smelter areas, this distribution is shifted, which results in a higher percentage of the population with a given level of lead in the blood. In addition, there is a wide range in exposure and thus blood levels at which persons are affected by lead depending on such things as age, sex, and nutritional status.

A summary of the blood lead levels at which adverse affects of lead have been observed in humans is shown in Table 7-1. The lowest reported effect level represents a value at which only a small proportion of the population is affected. However, the U.S. EPA (1977) developed dose-response curves that showed a sharp rise in the sigmoid dose-response curve for the percent of the population affected at only slightly higher blood lead levels. The no-effect levels are somewhat generalized and represent levels at which no effects were generally observed. However, recent studies suggest that subtle neuro-behavioral effects occur at blood lead levels for which no overt symptoms of lead toxicity are seen.

Available studies on the carcinogenic and mutagenic effects of lead are inappropriate for risk estimations. Although human data on the carcinogenicity of lead are scant, no evidence suggests that lead is carcinogenic to humans. A few feeding experiments with rodents have resulted in elevated incidences of renal tumors. However, if these dietary levels were extrapolated to humans, it would require consumption of 550 mg of elemental lead per day, which is far in excess of the maximum tolerated dose of lead in humans. Mutagenicity data are also inconclusive and contradictory.

Damage to the central and peripheral nervous systems and particularly to the brain, damage to the kidneys, adverse effects on reproductive capability, and the inhibition of the blood forming process are of considerable concern. Children, because of enhanced absorption and a variety of other factors, generally exhibit the adverse effects associated with lead exposure at lower blood lead levels (PbB) than adults.

TABLE 7-1. ADVERSE EFFECTS OF LEAD ON HUMANS[a]

Adverse Effect	Lowest Reported Effect Level (μg PbB/100 ml)	No-detected-effect-Level[b] (μg PbB/100 ml)
Carcinogenesis	--	> 40 occupational
Mutagenesis	--	40-120 occupational
Impaired Spermatogenesis	50	23-41
Fetotoxicity	30-40	--
Encephalopathy	80--children 100--adults	60--children ≥ 80--adults
Noticeable Brain Dysfunction	50-60--children	50--children
Peripheral Neuropathy	50-60	40
Nephropathy	40--children 50--adults	
Reversible		
Anemia	50-60--adults	40--children 50--adults
Elevated FEP (free erythrocyte proto-porphyrin)	15-20--children and women 25-30--men	20--children and women 25--men
Elevated ALA (δ-amino-levulinate acid) in urine	40	< 40
ALAD (δ-aminolevulinate dehydratase)	10	< 10

[a]Taken from data presented in Section 5.1.

[b]Note that recent studies suggest that subtle neuro-behavioral effects occur at blood-lead levels for which no overt symptoms of lead toxicity are seen.

The first indication of lead exposure is the inhibition of heme, the prosthetic group of hemoglobin responsible for oxygen transport. Inhibition of ALAD, the enzyme responsible for the synthesis of a precursor to heme, generally occurs at blood lead levels below 20 ug/100 ml. Several other steps in hemoglobin synthesis are also disrupted at PbB levels between 20 and 60 μg/100 ml. Mild anemia may occur in adults at or slightly above PbB values of 50 μg/100 ml and at a somewhat lower value (PbB=40 μg/100 ml) in children. Although these effects on heme synthesis are reversible, the implications of prolonged exposure to these effect levels are unknown.

Several studies indicate that sublethal lead exposures (PbB=30-40 μg/100 ml) may impair normal reproductive ability. Alterations in spermatogenesis are associated with lead exposure; available data, however, are limited to high occupational exposures (PbB=50-80 μg/100 ml); therefore, it is difficult to extrapolate to more typical exposure levels. Absorbed lead also crosses the placental barrier and enters the fetal bloodstream. Umbilical blood lead measurements indicate similar levels to those found in maternal blood. However, increased sensitivity of the fetus to PbB levels suggests that neurological damage can occur in the fetus with no overt symptoms of lead intoxication in the mother. No data suggest that lead is teratogenic in humans.

In addition, a major concern is the capability of lead to induce irreversible injury to the renal and nervous systems. Kidney disease associated with chronic lead exposure has not been adequately studied. Routine clinical tests are ineffective for early diagnosis and irreparable damage is sustained before chronic lead nephropathy is detected. Advanced lead nephropathy results in hypertension, interstitial fibrosis, and eventually reduced glomerular filtration, which progresses into renal failure. Reduced glomerular filtration occurs at relatively low blood lead levels (PbB=48-98 μg/100 ml).

Among the most devastating effects of increased lead absorption are the effects produced on the central and peripheral nervous systems. Manifested as encephalopathy, effects on the central nervous system are seen more frequently in children (PbB=80 μg/100 ml) than adults (PbB=100 μg/100 ml). The minimal level of lead exposure associated with lead encephalopathy, however, is not clearly known. Effects of lead exposure in the peripheral nervous system have also been documented. Peripheral neuropathy affects motor neurons and are characterized by loss of nerve fibers and segmental demyelination. The lowest reported PbB levels associated with these effects are 50-60 μg/100 ml.

Perhaps of greater societal concern are recent reports of subtle impairment of the cerebral function in children at PbB levels around 40 μg/100 ml. Many of these studies, as discussed in Section 5.1, are controversial; flaws in experimental design, complications of nutritional and socioeconomic status, insensitivity of behavioral tests, etc. complicate the issue. Despite the problems inherent in these studies, sufficient evidence exists to indicate that subtle neurobehavioral effects do occur in children exposed to sub-encephalopathic levels (PbB=40-80 μg/100 ml) of lead. The minimal level of lead exposure, duration, and age of greatest susceptibility cannot be defined with any degree of certainty at this time.

The exposures of various subpopulations as indicated by levels of lead in the blood are shown in Table 7-2. Because the data on blood levels are voluminous, this table presents only a few representative examples and is not all inclusive. The background information required for the interpretation of both Tables 7-1 and 7-2 is found in Sections 5.1 and 5.2. In addition, the actual exposure levels that may result in the levels of lead in the blood shown in Table 7-2 are discussed in Section 5.2. Because of the complexity of the toxicology and the exposure pathways for lead, this section, which is a discussion on the risk implications for various subpopulations as shown in Tables 7-1 and 7-2, should only be considered in conjunction with Chapter 5.0.

7.1.2 Adults

In general, food is thought to be the major exposure route of adults to lead (see Section 5.2). The total absorbed dose has been estimated to be 10-25 μg/day in rural areas. The mean blood levels of lead in these areas is around 10-16 μg/100 ml (see Table 7-2). In urban areas, when exposure via inhalation is more significant, total absorbed dose is estimated to be 15-40 μg/day. Mean levels of lead in the blood are about 15-24 μg/l. Persons living near smelters or other lead industries are exposed to higher levels of lead (about 70-300 μg/day as an absorbed dose), and a sizeable segment of the population show a blood level of greater than 40 μg/100 ml.

Although Table 7-2 does not identify the isolated subpopulations that can be exposed to high levels of lead, these have been discussed in Section 5.2. These subpopulations include those persons who consume drinking water that is highly contaminated resulting from the use of lead pipes, contaminated moonshine whiskey, and excessive amounts of contaminated wines. Of these population groups, the first two are likely to be larger than the group exposed to lead through contaminated wines.

In addition, certain segments of the population are particularly susceptible to lead exposure. For example, calcium and iron deficiencies, commonly observed in pregnant women, place them at a higher risk to lead toxicosis. In addition, Calabrese (1978) noted that individuals with latent porphyria may develop clinical symptoms when exposed to elevated levels of lead.

TABLE 7-2. HUMAN EXPOSURE TO LEAD AS EVIDENCED BY BLOOD LEVELS IN THE UNITED STATES

Location	Blood Level (μg/100 ml)	Reference
Adults		
Rural/Urban	9-24	Bell *et al*. (1979)
	Most ∿ 16	
Urban	18--mean (adjusted for age and smoking)	Tepper and Levin (1972)
	Less than 5% > 30	
Rural	16--mean (adjusted for age and smoking)	
	Less than 0.5% > 30	
Within 3.7 meters of Highway	23--mean	Daines *et al*. (1972)
Living Near a Smelter	16% >40	Landrigan *et al*. (1975)
Children		
Urban (primarily)	40,000 children detected annually > 30	
	∿ 20 yearly geometric mean	Billick *et al*. (1980)
Within 30 meters of Highway	50% > 40	Caprio *et al*. (1974)
Near Smelter--Kellogg, ID--1974 (immediate vicinity)	99% > 40	Walter *et al*. (1980)
	60% > 60	
1975	Somewhat reduced[a]	Anonymous (1979)
1979	Almost all < 60[a], and most < 40	
El Paso, TX	70% > 40	Landrigan *et al*. (1975)
	14% > 60	

[a]Reduction as a result of reduced atmospheric emissions as well as increased sanitary procedures for the workers who were apparently exposing their children to lead through their clothing.

Thus, a relatively large portion of the adult population is exposed to levels of lead that might result in some inhibition of heme synthesis. Effects on renal function and nervous systems are a potential risk to a much smaller subpopulation and are limited, for the most part, to occupational exposure. Impaired reproductive capacity has not been well studied; however, it does represent a potential risk to adults.

7.1.3 Children

Children are exposed to high levels of lead in numerous situations (see Table 7-2 and Section 5.2). Large numbers of children in urban areas, in rural areas where lead paint has been used, near highways, and near lead industries show levels of lead in blood greater than 30 μg/100 ml. Levels greater than 60 μg/100 ml are also not uncommon in children living in or near these areas.

The fetus, infant, and child have all been demonstrated to be especially susceptible to lead (see Section 5.1). At least to some portion of the subpopulations described in the previous paragraph, current exposure levels can result in anemia, irreparable damage to kidneys and motor neurons, noticeable brain dysfunction and encephalopathy.

Needleman (1980) discusses the problems of lead in children in the United States today. He points out that subtle effects on such things as learning capacity and number of synapses are being reported at increasingly lower exposures. The age of exposure appears to be especially important in effects observed later in life. Although studies in this area are controversial and somewhat contradictory, available evidence suggests cognitive impairment and behavioral effects in children with PbB concentrations consistently over 40 μg/100 ml. While some data indicate effects below this value, the evidence to date is inconclusive. In addition, he describes studies that suggest children who had apparently recovered from acute lead intoxication still showed behavioral disorders and sensory motor defects.

Thus, the preceding sections and most of the authorities on the subject (e.g., Needleman 1980, Mahaffey 1978) suggest that exposure to lead still represents a risk to children in the United States. The phasing out of lead in gasoline, the elimination of lead paint, the treatment of existing painted surfaces, controls on lead air emissions, especially from lead smelters, and the reduction of the use of lead solder in canned foods are expected eventually to greatly reduce the exposure of children to lead. However, the problem will continue for some time because of the existing contaminated soils and houses where lead paint was used.

7.2 RISKS TO BIOTA

7.2.1 Aquatic Organisms

Very few instances have been reported of fish kills attributed to lead; however, this is not a reliable measure of the risk that lead poses

to aquatic organisms. Chronic exposure to sublethal levels of lead may cause subtle yet significant harm to aquatic ecosystems, particularly in areas near anthropogenic sources of lead. To assess the risk of aquatic organisms with regard to lead, one can compare the concentrations that have resulted in effects in the laboratory with those concentrations that have been observed in the environment to cause effects.

The ranges of lead concentrations causing sublethal and lethal effects in various aquatic species as determined in laboratory studies are summarized below. The data supporting these conclusions are described in detail in Section 6.1.

- <10 μg/l — Life-cycle toxicity test in soft water produced abnormal development in rainbow trout. All other species tested are unaffected.
- 10-100 μg/l — Sublethal effects reported for several freshwater fish species in soft water, as well as growth inhibition in sensitive freshwater algae. Chronic toxicity reported in soft water are based on effects on early life stages in fish and life cycle tests in Daphnia.
- 100-1000 μg/l — Sublethal toxicosis in a variety of freshwater fish and invertebrates in soft and moderately hard water. Acute effects were observed on stickleback and some invertebrates. Numerous species of freshwater algae adversely affected, as well as more sensitive marine microflora.
- 1.0-10.0 mg/l — Acute toxicity level for several freshwater fish and invertebrate species in soft water and for sensitive marine invertebrates. Algicidal for some marine algae.
- 10.0-100 mg/l — Acute effects observed in a variety of freshwater fish and in most invertebrates tested. All algae species tested sensitive in this range.
- >100 mg/l — Only the most resistant species (e.g., red shiner, mosquitofish, cockle) are able to survive even for short periods in this range of concentrations. Hard water would be important for tolerance to such elevated levels.

This summary is intended only as a general guide in the assessment of the ecological impact of a given lead concentration in an aquatic environment.

Surface water concentrations of lead included in the STORET data base and from other miscellaneous sources are summarized in Table 7-3. The concentrations and their geographical distribution are discussed in more detail in Sections 4.2 and 6.2. For at least one year between 1975 and 1979, seven of the 18 major continental U.S. river basins designated by STORET have had mean total lead levels exceeding 50 ug/l -- a level indicating possible chronic toxicity in some species (see monitoring data in Chapter 4.0). Of these basins, only the Great Basin exceeded this level in 1979. Also, as indicated in Section 6.1, the levels of exposure increased in acidic or soft waters as a result of the increased availability of lead for uptake. In general, risk of exposure increases in basins with soft water, such as New England, the Pacific Northwest, and the Southeast.

From the limited data presented, it appears that the ambient concentrations occasionally reach levels of concern for most aquatic species. Source-specific locations, such as mining areas or in streams receiving urban runoff, may achieve lead concentrations potentially eliciting sublethal and lethal effects in both invertebrate and fish species. The U.S. EPA water quality criteria are exceeded in numerous locations.

Comparing monitoring data of lead concentrations in natural waters and effects levels to aquatic organisms in laboratory studies, however, can be a problem. Monitoring studies report total lead levels while suspended lead is not generally a consideration in laboratory studies. A large portion of the total lead in natural systems is bound to particulate matter (see Section 4.2). Only conjectures can be made about the percentage of bound lead and the role this fraction plays in lead toxicity. Some potential for re-release to the water column always exists, although Chapter 4.0 suggests that lead is tightly bound. Without information on the ambient levels and distribution of complexing agents, such as organic and inorganic ligands, suspended sediment levels, changes in pH (resulting from spring floods, acid rain) and the influences of industrial effluents on the dynamics of adsorption and complexing, no estimates of actual or potential lead availability to biota can be made. In addition, the role of ingestion of sediment lead and its transfer up the food chain is not well understood. The data have been presented to illustrate: (1) the prevalence of ambient concentrations exceeding levels that have adverse effects on certain aquatic species in the laboratory, and (2) specific situations where lead concentrations occur that may have adverse effects on most aquatic life.

TABLE 7-3. CONCENTRATIONS OF LEAD IN WATER

Source	Total Lead Concentration
STORET -- Ambient levels[a]	
7.7% of U.S. minor river basins have a mean concentration exceeding this level	50 μg/l
8.1% of U.S. minor river basins have 10% of maximum concentrations exceeding this level	100 μg/l
Miscellaneous Source-related Levels	
Mine wastewater, Yukon Territory Boyle (1965)	1000 μg/l
Settling pond, mine in MO Proctor *et al*. (1974)	1000 μg/l
Mine and creek waters in mining area, U.S.S.R. Edgington and Robbins (1976)	7000 - 9000 μg/l
Stream near lead smelter, MO (Gale and Wixson 1979)	300 μg/l
Storm runoff wastewater in Durham, NC (Chow 1978)	100 - 12,000 μg/l
Runoff from streets of high traffic density in Oklahoma City, OK (Chow (1978)	5500 μg/l
Sewage effluent in city with lead-emitting industries (Chow (1978)	100 - 500 μg/l
Sewage effluent in Los Angeles, CA Chow (1978)	250 μg/l

[a]Data described in greater detail in Table 6-5.

7.2.2 Terrestrial Organisms

7.2.2.1 Mammals

The risk of exposure to lead in toxic concentrations is apparently greatest for relatively nonmobile species that live near anthropogenic sources of lead. Most studies on lead poisoning in mammals have focused on rodents living various distances from a highway. Clark (1979) has observed mortality in laboratory tests on domestic mammals at daily doses of 1 to more than 10 mg/kg/day. The same author estimated that daily lead doses range from 1 to 128 mg/kg/day for wild rodents and insectivores (e.g., shrews) living near highways. Elevated tissue residues have been observed in specimens living near roadsides; however, there have been no reports of lead poisoning from this type of exposure.

Robert and co-workers (1978) found renal inclusions and edema in rodents living near abandoned mines in Wales, and attributed the effects to high lead burdens (8-45 mg/kg dry weight). Haschek and co-workers (1979) examined rodents living in an orchard that had been treated earlier with lead arsenate. Several specimens had renal damage, which the authors thought was associated with high residues of 14 mg/kg (dry weight) in the kidneys. Soil concentrations in the area averaged 1342 mg/kg (dry weight).

Grazing animals, such as deer, may be exposed to higher levels because they frequently forage near roads. Some evidence suggests that lead does not biomagnify in food chains (Williamson and Evans 1972), although this does not necessarily imply that carnivores are not at risk.

Because the relationship between tissue residues and toxicity is not clear, it is impossible to assess the risk to various species on the basis of lead concentrations in tissues. Nonetheless, body burdens do reflect elevated exposure in some areas and may be a measure of potential risk.

7.2.2.2 Birds

According to numerous reports of lead poisoning in birds, the vast majority of mortalities occurs in waterfowl as a result of ingested spent lead shot. Yearly, losses of waterfowl (from lead poisoning) were estimated at 2-3 million birds, or 2-3% of the U.S. waterfowl population (Bellrose 1959); more recent nationwide figures were not available.

The risk of lead poisoning from shot ingestion is most closely associated with the hunting pressure in a given area. Of the four major flyways in the United States, the Mississippi had 40-45% of the total waterfowl hunting activity. Approximately 25% of the hunter-days took place in the Central Flyway, while the Atlantic and Pacific each had less than 20% (Bellrose 1959).

Certain species of waterfowl are much more likely to ingest lead shot than others, largely because of feeding habits. Among several surveys of gizzard contents from bagged birds (U.S. FWS 1976), diving ducks had the highest incidence of ingested shot. In this group, canvasback, redhead, scaup, and ring-necked ducks appear to be at the highest risk because they have the greatest tendency to ingest lead shot. Other species of special concern are waterfowl that consume corn as a major component of the diet. These include the Canadian geese, mallard, pintail, and related species of dabbling ducks.

In 1976, the U.S. Fish and Wildlife Service began a gradual phasing-out of the use of lead shot for waterfowl hunting. By the fall of 1980, lead shot was completely prohibited for all types of shotguns in certain areas that account for about 20% of the waterfowl harvest of the United States (U.S. FWS 1980). In states that have aggressively promoted the use of nontoxic steel shot as a substitute, some promising results have been achieved. At the Sauvie's Island State Management Area in Oregon, 5000-6000 mallards perished each year from lead poisoning before lead shot was prohibited. For several years after the regulation requiring steel shot, mallard mortality as a result of lead ingestion decreased 25% per year. Officials at Turk's Pond in Colorado reported a "pronounced change" in goose mortalities after the use of steel shot became mandatory. In Michigan, lead shot has been selectively prohibited. Smith (1980) noted that 50% of the shot now found in waterfowl gizzards is steel. From this evidence, it seems likely that the continued phase-out of lead shot will further reduce the incidence of lead poisoning in waterfowl.

Other species of birds may also ingest lead shot accidentally during feeding. Of these, the species with perhaps the highest rate of lead shot ingestion are doves. In an examination of doves bagged by hunters, Lewis and Legter (1968) found lead shot in 1% of the gizzards. Aquatic predators, such as ospreys and bald eagles, are also at risk to the extent that they feed on animals containing lead shot (U.S. FWS 1980, Bagley and Locke 1967).

Like other organisms, birds in urban areas tend to have higher lead residues in their tissues than populations in rural areas. Possible sources of lead exposure to urban birds include dirt and air pollution. The ingestion of tailings from lead mine wastes has led to death in waterfowl on several occasions; therefore, the risk of lead poisoning is probably somewhat higher in mining areas than other areas.

7.2.2.3 Plants

It is extremely difficult to relate effects data to exposure levels in order to estimate environmental risk for plants. In laboratory experiments, lead is usually presented as a salt in aqueous solution or in vapor form (Section 6.1). However, with these exposure techniques, the observed effects levels are not equivalent to concentrations available for uptake from soil. For example, lead concentrations of 30 mg/l reduced corn seedling growth in a nutrient culture, but

had no effect when present at 270 mg/l in soil (Tornabene et al. 1977). In field toxicity studies, the factors influencing biological availability are unknown; thus, generalization from the results to other situations and conditions is impossible.

The literature does not contain reports of lead poisoning to plants in field situations. However, indirect effects on productivity (i.e., delay nutrient recycling because of microbial inhibition) have been observed within one-half mile of a lead smelter.

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APPENDIX A. NOTES ON THE DERIVATION OF TABLE 3-1

1. Information on lead supplies were derived from the Mineral Commodity Profiles for 1977 (U.S. Bureau of Mines 1977a).

2. Consumption data are from Lead Industries Association, Inc. (1978), and from statistics compiled by the American Bureau of Metal Statistics (Kirk-Othmer 1967).

3. According to Nriagu (1978), the following emission factors apply to the mining and milling of lead ores on a global basis for 1974 and 1975:

mining	0.91 kg/kkg
milling	1.32 kg/kgg
Total	2.23 kg/kkg

   Assuming an airborne emissions capture rate of 50% for U.S. mining and milling operations, the effective total emission factor is 1.12 kg/kkg of lead mined and milled domestically (517,897 kkg); this corresponds to an airborne emission for domestic mining and milling of 580 kkg. To this must be added the airborne emission from the smelting and refining of domestic ore concentrates, a process for which Nriagu (1978) gives a worldwide emission factor of 6.36 kg/kkg; however, assuming an emissions capture rate of 75% in domestic smelting and refining operations in 1976, the effective emission factor for smelting and refining is 1.59 kg/kkg for which the airborne emission would be 823 kkg. Thus, the total airborne emission from the mining, milling, smelting, and refining of domestic ores is 1403 kkg. The solid wastes that result from emissions capture are reflected in the solid waste emission level for the category of primary domestic lead production.

4. Imported ores must be smelted and refined in the United States. Nriagu (1978) assigns an emission factor of 6.36 to the worldwide smelting and refining of lead. In the United States, some of the airborne emissions are captured; and if the emissions capture rate is taken to be 75%, then the effective emission factor is 1.59 kg/kkg. The amount of lead refined from imported ores was 78,909 kkg in 1976, which corresponds to an airborne emission of 125 kkg.

   The emissions that are captured, 75%, are assumed to show up in the solid waste stream for the category of imported ores that are domestically smelted and refined, or 375 kkg.

5. The airborne emission volume of 184 kkg associated with secondary lead production is derived on the basis of an emission factor of 0.35 kg/kkg of secondary lead produced (U.S. EPA 1974). The amount of secondary lead produced was 526,060 kkg in 1976; therefore, the air emission was 184 kkg.

6. No data are available concerning airborne emissions from battery production. However, in the production of batteries, grids and posts are cast, which may give rise to losses via volatilization (U.S. EPA 1975, 1976). Material handling may also contribute to airborne emissions. Using the emission factor of 0.5% (see note 10) of the lead used in smelting and casting, the estimated airborne emissions from battery production is about 1700 kkg. With regard to lead oxide used in batteries, airborne emissions might be associated with handling procedures, however, amounts are unknown; and, if the lead oxide is produced from lead metal, then emissions could be significant, depending on the amount of emissions capture machinery used to minimize emissions.

7. Airborne emissions of lead used in gasoline antiknock additives consist of two components: emissions due to manfuacturing processes, which according to data compiled by the Noyes Data Corporation (Sittig 1975) was 1614 kkg in 1976; and emissions from the combustion of leaded gasolines, which is taken as 80% of the 217,461 kkg of lead used in antiknock additives (Hepple 1971). The sum of these two airborne emissions associated with antiknock additives is 175,584 kkg.

8. Ammunition is assumed to consist primarily of lead shot and small-caliber bullets that are steel- and copper-jacketed. The airborne emission of 666 kkg is based on an assumed air emission factor of 1%, which is evolved during the firing of the round and during the impact, especially in firing ranges owned by the military, the police forces, and by private gun clubs. Air emissions during the manufacture of rounds is possibly even greater than the loss during actual firing; however, no data are available on which to base an estimate. The solid waste (land-destined) lead burden associated with ammunition is assumed to be about 75% of the total amount of lead used in ammunition; this is attributed to rounds spent in military and sport gun use where the rounds are unrecoverable, and to practice ranges where the spent rounds are not recovered. Losses to POTWs are considered to be negligible; and lead losses to water, if they occur at all, probably take place during manufacturing operations.

9. Air emissions of lead during solder use are estimated to be on the order of at least 2% of the amount of lead used in solder. The loss to the air is attributable to volatilization during the solder use, especially during manual use operations as in plumbing and electronics repair. Airborne emissions of lead during solder manufacture no doubt take place but the amount is unknown and no estimate has been made; also losses to the air during automated soldering operations are probably minimal because temperatures are automatically controlled. Aquatic and POTW emissions of lead due to solder use are probably small, though the amount of lead going to POTWs because of solder use is probably greater than the amount going directly to waters since solders are used extensively in plumbing systems

carrying water that ultimately goes to POTWs (though, of course, many plumbing systems carry water to nonPOTW sinks). The amount of lead going to land as a result of solder use is taken as 10% of the total amount of lead used in solder and is assumed to be primarily due to the disposal of electronic apparatus and losses of solder during manual soldering use in plumbing and electronics repair.

10. Airborne emissions of lead during the production of weights and ballasts is attributed to volatilization losses during melting and casting. The emission factor is estimated to be on the order of 0.5% of the amount of lead used (20,286 kkg), or about 101 kkg. Losses to water and POTWs during production and use are unknown, but are probably an extremely small percentage of the total amount of lead used in weights and ballasts. Solid losses to the environment are attributed to the loss and disposal of lead weights in the form of wheel balance weights and fishing-gear sinkers and are estimated to be on the order of at least half of the lead used in the weights and ballast category; i.e., 10,143 kkg to solid waste.

11. Emissions associated with lead pigments are based on emissions factors taken from the cadmium production and use report for losses due to cadmium pigment manufacture. Emissions during use processes are not known, but solid waste associated with packaging disposal, plus water losses associated with the use of paints and pigments might be at least equal to the amounts shown below and in Table 3-1. The emission factors derived from the cadmium production and use report (Versar 1980) are as follows (the emission is based on 15,087 kkg of lead used in pigments):

	Emission Factor (%)	Emission (kkg)
Air	1.4	212
Water	0.1	15
POTW	0.3	45
Solid	2.4	363

12. Emissions resulting from the use of 14,449 kkg of lead in 1976 in lead cable covering is estimated to be 0.1% due to rolling and handling processes during production, or 14 kkg. Losses to water are unknown, probably small; and to POTWs the losses are estimated to be small because of the low solubility of lead in water. Solid wastes are estimated at about 25%, due to the replacement and disposal of lead-covered cable in commercial and residential applications; i.e., about 3612 kkg to land.

13. Air emissions of lead during the manufacture of leaded brasses and bronzes are estimated at about 5% of the lead used. (The high volatilization figure is used because of the higher melting tempera-

ture of brasses and bronzes relative to that of lead.) At 5% the 14,203 kkg of lead associated with brasses and bronzes result in an airborne emission of 710 kkg. Because lead is added to brasses and bronzes to improve machineability, significant losses would occur during machining operations, especially in small shops where recovery of metal scraps is not consistent. Also, the rate at which machined brass and bronze components of consumer goods are disposed of and replaced by new units, plus the solid losses due to machining processes, together amount to an estimated 20% of the lead used in brasses and bronzes, or about 2840 kkg of lead to solid waste. Losses of lead to water and to POTWs are probably negligible, especially during industrial and consumer use of leaded brasses and bronzes. However, the manufacture of leaded brasses and bronzes might result in some amount of loss of lead to the waters.

14. According to the U.S. Bureau of Mines (1977a), lead sheet is used in radiation shielding, vibration dampening, and sound attenuation, the latter category being widely used in modern architecture. However, lead use in roofing and flashing has decreased, which means that old buildings that are torn down might be a source of solid lead wastes. Because sheet lead must be rolled during production, an air emission estimate of 0.1% is reasonable, so that the 22.165 kkg of lead used in sheeting accounts for an air emission of 22 kkg; airborne emissions during use of lead sheeting is negligible. Because lead sheeting is used in industry to protect process vessels, especially in the handling of highly corrosive materials, some losses of lead are likely to result in a way that will reach aqueous media; however, data are unavailable on the amounts of lead sheeting actually used by industry to protect its process vessels. Generally, lead losses to the waters and to POTWs due to the manufacture and use of lead sheet are estimated to be negligible. Solid losses of lead due to sheeting are estimated to be on the order of at least 10% of the amount of lead used in sheeting, i.e., 2216 kkg.

15. Lead-alloy bearings are of the "plane" type that operate usually in a forced lubrication environment of the kind found in automobile engines and railway car journal boxes. Bearing wear results in losses of lead to the lubricant, which in turn can be discarded to land, and a small amount might be disposed of by incineration, thereby causing an airborne emission of unknown magnitude. The primary airborne emission assoicated with lead-alloy bearings would result during the manufacturing process, when it is estimated that about 0.5% of the lead would be volatilized to the air. Thus, the air emission from the use of 11,848 kkg of lead in bearings is 59 kkg. Some emission of lead might result to water becasue of the manufacturing of lead-alloy bearing, however, the amount is unknown and the amount goint to POTWs during manufacure is estimated to be negligible. The solid wastes of lead attributable to bearing use is estimated to be on the order of at least 80% of the amount of lead used, the rationale being that it is uneconomical to recycle and recover the lead alloy used in the large amount of steel-backed

replaceable bearing units; the other 20% of lead use is taken to account for an increase in the number of lead-alloy bearing in service and in replacement stocks.

16. The amount of lead used in calking metals was 11,315 kkg in 1976. Lead calking is still required, because of its corrosion resistance, in the building codes of some locales (U.S. Bureau of Mines 1977a). The manufacture of sheet lead and strips of lead for calking is estimated to result in an airborne emission of 0.1% due to rolling and handling processes, or a total emission of 11 kkg. Water discharges due to manufacture are unknown but probably small, as are POTW discharges. Discharge to waters and POTWs during use can be taken as zero. With regard to solid wastes, lead calking has been used in buildings for a long time, the discharging of lead from old buildings that are destroyed must be on the order of at least an estimated 25% of the amount used in manufacturing in 1976, or a total solid emission of 2716 kkg.

17. Lead pipes, especially traps and "ells" (bends) are still required by the building codes of some localities (U.S. Bureau of Mines 1977a) was much more common because of the ease of fabrication of lead pipes and fittings. If a conservative estimate of 0.1% is taken as the airborne emission factor for the manufacture (rolling, casting and extrusion) of lead pipe fittings, then the manufacturing air emission of lead would be about 12 kkg, based on a use level of 12,507 kkg of lead in 1976. Losses of lead to water and POTWs are unknown and could be a significant portion of the lead used in pipes and pipe fittings. If the disposal rate of old lead pipes in buildings that are demolished is taken at 25% of the amount of lead used in pipes in 1976, then the solid waste volume from lead-pipe uses is about 3127 kkg.

18. Collapsible tubes are used for the packaging of toothpastes, artists colors and corrosive chemicals (U.S. Bureau of Mines 1977a). Because they are manufactured by extrusion, a small amount of airborne emission can be assumed due to high working temperature and abrasion with the extrusion dies. An estimated emission factor of 0.1% means that the manufacturing air emission would be on the order of 2 kkg (out of the total amount of lead used in collapsible tubes of 2112 kkg). Emissions to waters and POTWs can be taken as effectively zero, as far as use is concerned because the tubes are not exposed to water very much during use, though a small aqueous discharge might be associated with tube production. All of the lead uses in collapsible tubes can be assumed discarded to land, except for a small amount that might find its way to disposal by incineration.

19. The bulk of lead used in printing type is used by the newspaper industry and related industries where large volumes of materials must be printed in short periods; situations where photographically

prepared plates of the kind used in off-set printing would not have adequate durability for printing upwards of half a million impressions. Because "hot-type"linotype machines used melted type metal, some losses due to volatilization can be expected, and because the temperature is precisely controlled, it is likely that, conservatively, the volatilization losses would be no more than that encountered in the controlled temperature melting of lead in other automated melting processes, i.e., on the order of 0.5%/yr. Thus, the airborne losses of lead as a result of printing operations would be about 68 kkg/yr, based on a total lead use of 13,611 kkg in 1976. Losses of lead as solid waste from the use of lead in type would be by means of spills, drips, and adherence of small amounts of lead to the disposable cardboard plates into which the lead is cast; an estimate of the losses of lead to solid waste streams as a result of floor sweepings and other loss processes is at least 1%, and probably much higher. At 1% the amount is 136 kkg. With regard to water losses, it is considered improbable that much lead finds its way into the waters either directly or indirectly, however, because most users of hot-type printing processes are urban located, any lead losses that do take place probably go to POTWs, but in amounts that cannot be estimated.

20. Terne metal is a coating of four parts lead to one part tin and is used to coat sheet iron and steel. Because only 1447 kkg of lead were used in terne metal applications in 1976, the amounts lost to the media were probably also small. Because industrial melting processes are well controlled, the volatilization losses during coating processes would be small but probably not less than 0.5%, or about 7 kkg. And assuming a disposal rate of 5% the solid waste burden from the use of terneplate would be about 72 kkg. Losses to water and POTWs are considered negligible because of the low water solubility of lead and because of the relatively small amount of lead used in terne metal.

21. Baths of molten lead can be used in the annealing of certain alloys. The presence of baths of lead in a sustained molten state would be expected to account for relatively large volatilization losses, especially because the effective surface area for volatilization would be large as a result of the dipping and withdrawal of hot metal parts. Therefore, a conservative estimate of volatilization losses from annealing processes is on the order of 10%/yr, or 262 kkg of the 2624 kkg of lead used in annealing processes in 1976. Solid losses due to the adhering of lead to annealed parts, and the subsequent losses of lead in ways that it would be discarded along with other solid waste (e.g., as floor drippings, and as flakings from annealed parts), plus the disposal of lead oxide scums that form on the surface of baths of lead, must account for at least 20%/yr of the lead used, or 524 kkg minimum. Losses to waters and POTWs is estimated to be minimal.

22. White lead is a term applied primarily to lead carbonate; it is also used for lead sulfate and lead silicate, and the primary use is as a pigment in paints. The amount of lead in white lead in 1976 was 2715 kkg. An estimate that is considered conservative for losses as solid waste due to the production and use of white lead is 2% due to dust losses that settle out and to losses associated with packaging disposal. Because it is likely that some packaging used by white lead manufacturers to ship the product to paint formulators is incincerated, a volatilization loss probably occurs. Estimating the airborne loss at 0.5% and the solid loss rate at 1.5%, the amount of lead lost to air is 14 kkg and the amount lost to landfills is 41 kkg; to the latter amount must be added the amount of painted products that are discarded, which, assuming a discard loss rate of 10%/yr minimum, the total solid lead loss rate to land is at least 312 kkg. Losses to waters and POTWs are unknown, however, it is possible that upwards of 5% might be lost to aqueous sinks, albeit in a nonsolubilized form, because lead compounds are not readily soluble in water.

23. Only 350 kkg of lead were used in plating processes in 1976. Losses to waters and POTWs would probably be small also, however, because of the lack of data, an estimate cannot be assumed. Airborne losses are probably negligible, except for losses of lead compounds due to dusting, which would probably rapidly settle out. Solid losses due to plating would most likely be in the form of discarded lead-plated products, however, because of the lack of data, the disposal rate of plated products cannot be estimated.

24. The amount of lead used in cast products was 6084 kkg in 1976. Because lead that is cast must be heated to a temperature that is somewhat higher than its melting point, higher levels of volatilization losses can be expected than would result from other manufacturing processes involving the use of molten lead, as in the manufacture of ammunition, solder, weights and ballasts; therefore, the volatilization loss rate is estimated to be at least 1% or 61 kkg. Solid losses due to spilling, and the removal of casting flash, plus any losses that take place with disposable molds is at least several percent, which, along with the solid disposal of used cast products, could be conservatively estimated at 25%, or 1521 kkg. Losses to waters and POTWs are unknown, but probably only on the order of several kkg.

25. As with annealing, galvanizing processes involve hot dipping in molten baths of lead; thus, the volatilization loss rate of 10%/yr seems a reasonable estimate and corresponds to 114 kkg of the 1136 kkg of lead used in galvanizing in 1976. Disposal of lead-galvanized products is taken at 25% (which includes the lead losses due to the galvanizing processes, e.g., through dripping and spillage). Losses to waters and POTWs are considered negligible because of the relatively small amount of lead used in annealing and because of its low solubility in water.

26. Losses of lead due to the manufacture and use of lead foil are primarily to land, as a result of the disposal of lead foil wrapped hazardous materials as well as foil trimmings. An estimated airborne loss rate of 0.1% due to the rolling of lead into foil results in an emission of 5 kkg from the 4649 kkg of lead used in foil. The solid waste stream is taken to consist of about half of the foil produced, or 2324 kkg, because the foil is assumed to be used primarily for the packaging and disposal of hazardous waste. Lead foil is also used to package radioactive materials in transit (U.S. Bureau of Mines 1977a), and then it is discarded. A good portion of the foil is used to wrap low-level radioactive waste that is then sent for storage/disposal. Lead loadings to water and POTWs are estimated to be zero because of both the small amount of lead used in foil and its low solubility in water.

27. The worldwide airborne emission factor for the combustion of fuel oils is, according to Nriagu (1978), 0.0091 kg/kkg. The amount of fuel oil burned in 1976 was about 289 million kkg (U.S. Bureau of Mines 1977a). Therefore, the airborne emission was 2630 kkg. Emissions to all other media as a result of oil combustion processes are assumed to be zero.

28. The combustion of coal, the production of iron and steel, and the smelting of copper and zinc result in substantial airborne emissions of lead. Assuming a 75% emissions capture rate, then 75% of the captured lead can be assumed to be a solid waste, and the other 25% remains an airborne emission. According to Nriagu (1978), the worldwide emissions of lead from these processes are:

Coal combustion	14,900 kkg
Iron and steel production	49,700 kkg
Copper and zinc smelting	42,300 kkg

Coal used in the United States is about 15% of the worldwide value (U.S. Bureau of Mines 1979); and only 85% of coal consumed in the United States is consumed as fuel (U.S. Bureau of Mines 1977b). Therefore, the U.S. emission of lead from coal combustion is:

$$(0.25)\ (0.85)\ (0.15)\ (14{,}900) = 475 \text{ kkg airborne emission}$$

And the corresponding solid waste emission resulting from airborne emissions capture is:

$$(0.75)\ (0.85)\ (0.15)\ (14{,}900) = 1425 \text{ kkg to land}$$

For iron and steel production the United States uses 10% of the worldwide production or iron (U.S. Bureau of Mines 1978a). Assuming, again, a 75% emissions capture efficiency overall, the airborne and solid emissions of lead due to iron and steel production are:

Airborne: $(0.10)\ (0.25)\ (49{,}700) = 1243$ kkg
Solid: $(0.10)\ (0.75)\ (49{,}700) = 3729$ kkg

Copper and zinc production are actually handled separately by Nriagu (1978). Worldwide emissions of lead from copper production and zinc production are 26,600 kkg and 15,700 kkg, respectively. The corresponding portions of the worldwide use of copper and zinc with the U.S. use in 1976 were 22.4% (U.S. Bureau of Mines 1975) and 7.5% (U.S. Bureau of Mines 1978b), respectively. Therefore, the corresponding U.S. emission would have been in 1976:

Copper production:	6000 kkg
Zinc production:	1200 kkg
Total	7200 kkg

Of the total airborne emission of 7200 kkg, the portion that is assumed to be captured in emissions-capture devices is 75%, which is assumed to be a solid waste. Therefore, the total lead emission to land and air from the production of copper and zinc is:

Air: (0.25) (7200) = 1800 kkg
Land: (0.75) (7200) = 5400 kkg

29. These data are derived from the U.S. EPA MDSD's TABS DATA SYSTEM and only includes data points that are currently in the database. Consequently, these data are documentable, however, they represent a minimum estimate of source discharges.

30. No data are available on the airborne lead emissions from the refining of petroleum; however, it is possibly significant because the amount of petroleum refined is very large.

31. Airborne and solid waste lead emissions from the mining of coal are unknown, but probably small.

32. According to the SRI (1979) report on the agricultural sources of lead, 95 kkg of lead are contained as an impurity in phosphate fertilizers. The entire burden is assumed to be applied to land, but no doubt a portion of runoff exists.

33. The estimate of lead discharges can be made based on preliminary effluent data compiled by Burns and Roe (U.S. EPA 1980) and (U.S. EPA 1977). The usefulness of such an estimate is only speculative at best because data are available on 9 POTWs and effluent concentrations of lead are variable (0-53 μg/l). Removal efficiencies for lead in POTWs also vary, although they seem to depend, to some degree, on the influent concentration. According to the U.S. EPA (1977), the national POTW flow rate is 22674.694 MGD. Based on 9 plants for which data are available, the average effluent concentration is 19 μg/l (U.S. EPA 1980). This is a mathematical average, not a flow-weighted average.

$$\text{Annual discharge} = (22674.694 \times 10^6 \frac{gal}{day})\ (3.785 \frac{l}{gal})$$
$$(19 \times 10^{-9} \frac{gms}{l}\ (365 \text{ days})$$
$$= 5.48 \times 10^6 \text{ gms}$$
$$\approx 5.5 \text{ kkg}$$

Accordint to SRI (1979), sewage sludge contains between 15 and 1900 mg/kg of lead. The amount of sewage sludge generated in 1976 was 3.6 million kkg. The distribution of sewage sludge is as follows: 25% is distributed to land as fertilizer; 25% is landfilled; 15% is ocean dumped; and 35% is incincerated. If the lead contained in the sewage sludge that is incinerated is assumed to be half volatilized and the other half stays with the ash and is landfilled, then the distribution of lead follows this pattern:

Air:	64 - 955 kkg
Land:	264 - 3683 kkg

Loses to water as a result of runoff are unknown.

34. These data are based on information on one plant; the information was supplied by the U.S. EPA (1979). The identity of the plant is unknown. It is assumed that this data point is representative of all the plants and that all plants discharge directly.

35. According to the U.S. EPA (1975, 1976), approximately 200 domestic plants manufacture lead acid storage batteries and battery oxides. Recycling is highly prevalent among these plants. Virtually all off-spec product is processed as well as most of the process wastes and expended consumer materials. Approximately 32-114 kg of lead containing waste solids are process derived per kkg of product (U.S. EPA 1976). Approximately 0.015-0.61 kg of lead/kkg of product occur as waterborne waste (U.S. EPA 1976). Based on 1972 production of 889,000 kkg the wastewater discharge of lead from this industry is between 13 to 542 kkg. However, over 80% of the lead subcategory plants discharge to POTWs (U.S. EPA 1976). Assuming this ratio is reflected in the wastewater flow, 3-108 kkg lead are discharged directly, and 10-434 kkg are discharged to POTWs. Only about 30 kkg of wastewater treatment sludges are reported to be disposed of annually. Presumably the remainder is recycled at the plant and at a lead smelter (Versar 1980). Thus, it is expected that land-disposed lead resulting from battery manufacture is insignificant.

36. No data were available to estimate the losses to water, POTW or solid waste from the production and use of lead as a gasoline antiknock additive. However, these releases are expected to be small relative to the airborne releases from this source.

37. More than 90% of the lead content of ores is retained during beneficiation (U.S. Bureau of Mines 1977a). Assuming that the amount retained is 90%, then the total quantity of lead contained in the ore from which the domestic supply of 517,897 kkg is recovered is:

$$\frac{517,897}{0.9} = 575,441 \text{ kkg}$$

Thus, the solid waste generated is the amount of lead that remains in the ore tailings, which is the above amount, 575,441 kkg, less the amount of lead recovered, 517,897 kkg, less the airborne emissions, 1403 kkg, less the aquatic emission, which is 175 kkg for both domestic and imported ore smelting and refining; this latter amount, 175 kkg, can be proportioned between the imported and the domestic amounts of lead recovered such that the aquatic emission due to the production of lead from domestic ore is on the order of 152 kkg. Thus, the total solid emission from the production of domestic lead is:

$$575,441 - 517,897 - 1,403 - 152 = 55,989 \text{ kkg}$$

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