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REVIEW OF PROPOSED EPA AMBIENT LEAD CRITERIA STANDARD DOCUMENT

Final Report

March 4, 1984

Work Performed Under Contract No. AC01-82EP12086

Radian Corporation
Austin, Texas

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Final Report

Task Assignment No. 10

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EXECUTIVE SUMMARY

This report reviews the proposed October 1983 EPA ambient lead criteria document, Air Quality Criteria for Lead, from the perspective of DOE's policies and programs and addresses potential impacts on energy production and energy-intensive industries.

Following an introduction, the study is organized in five subsequent sections. Section 2.0 addresses environmental and health effects of exposure to lead. Section 3.0 reviews sources of lead emissions. Section 4.0 presents information on lead concentrations in ambient air. Section 5.0 examines dose-effect relationships among lead emissions, ambient air concentrations and blood lead levels. Section 6.0 presents Radian's evaluation of the regulatory implications of the criteria document and the information it provides. A summary of the major conclusions from each of these sections if provided below.

Environmental and Health Effects

Environmental effects of lead include reduced growth and lifespan of plants, and effects on the central nervous system of animals. The level at which symptoms occur in animals is above 40 micrograms per deciliter (µg/dl) of whole blood. Perhaps the most significant environmental impact is that high lead concentrations eliminate bacteria and fungi on leaf surfaces and in the soil and thus inhibit the nutrient-providing plant decomposition process.

In humans, health effects begin to occur at blood levels of 15 µg/dl. A large number of symptoms have been documented in the range from 40 to 60 µg/dl, and blood lead levels above 100 µg/dl produce permanent brain damage or fatality.

Subpopulations at risk include preschool children and pregnant women. Preschool children are particularly susceptible because of mouthing habits and their active metabolisms. Pregnant women are at risk because of potential impact on the unborn child.

Sources of Lead Emissions

Gasoline combustion is the source of 86% of current U.S. lead emissions. An additional 5.7 percent of total emissions are due to other combustion sources, and the remaining emissions are due to primary and secondary lead smelting (3.8 percent), iron and steel production (1.3 percent) and other stationary sources.

Lead emissions have decreased since 1977 because of increased use of unleaded gasoline and the regulations which have constrained the lead content allowed in gasoline. The percentage contribution of stationary and mobile sources has not changed during this period because stationary source emissions have also been reduced.

Ambient Air Lead Concentrations

As a result of the reduction in lead emissions from gasoline use, ambient air lead concentrations in both urban and rural areas decreased in the late 1970's. In 1970, only about 80% of reporting stations had met the standard of 1.5 $\mu g/m^3$. By 1980, more than 99 percent of reporting stations met the standard. Eighty percent of the stations with concentrations over 2 $\mu g/m^3$ are near stationary sources, reflecting the local nature of the remaining noncompliance problem. New monitoring requirements in 1981, placing monitors nearer roadways, may result in slightly higher reported concentrations in the future.

Dose-Effect Relationships

Based on a summary of lowest observed effect levels for lead-induced health effects in adults, "the rationale for continuing to view 30 $\mu g/dl$ as a 'maximum safe' blood-lead level is called into question, and substantial impetus is provided for revising the criteria downward, i.e., to some blood-lead level below 30 $\mu g/dl$". To establish a dose-response

relationship, the document concludes than an inhalation slope of 1.5 μg/dl per $\mu g/m^3$ is appropriate for adults, and 2.0 μg/dl per $\mu g/m^3$ is appropriate for children. Since dietary contributions to total lead burden are high (particularly for adults), the role of lead in food processing will become relatively more important as the lead content of gasoline continues to decrease.

Regulatory Implications

The newly revised criteria document has all of the information available to justify a lead standard of 0.5 $\mu g/m^3$, one third of the present standard, as well as changes in averaging times which would further increase the stringency of the standard.

Since the trend in ambient concentrations is downward, 99 percent of stations are in compliance with the present standard and the mean reported concentration is now 0.84 $\mu g/m^3$, compliance with a more stringent standard may not be difficult provided use of leaded gasoline continues to be reduced.

The major impact on energy-consuming industries from a more stringent standard will be on the petroleum refining sector because of reductions in leaded gasoline allowed. According to API, five to ten percent more energy is required to produce unleaded than leaded gasoline. An additional impact will be more stringent particulate controls on stationary lead sources.

1.0 INTRODUCTION

The purpose of this study is to provide a review of the proposed EPA ambient lead criteria document[1] from the perspective of DOE policies and programs, addressing potential impacts on energy production and energy-intensive industries.

This study is based exclusively upon the information provided in the criteria document. No effort was made to review the literature and analyses upon which the document was based. A comparison was made between the current document and the earlier criteria document published in 1977.[2]

This study is organized in five sections. Section 2.0 addresses environmental and health effects of exposure to lead. It identifies affected sub-populations and defines the geographic distribution of populations at risk.

Section 3.0 reviews sources of lead emissions identified in the criteria document. It focuses especially on energy production and energy-intensive industry sources.

Section 4.0 reviews the information provided on lead concentrations in ambient air. It also reviews the discussions of monitoring for air lead.

Section 5.0 examines dose-effect relationships among lead emissions, ambient air concentrations and blood lead levels, and reviews the health effects as a function of blood lead concentrations.

Section 6.0 discusses the regulatory implications of the criteria document and the information it provides. The possibility of a more stringent air standard is reviewed.

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[1] EPA, Air Quality Criteria for Lead, Review Draft, EPA-600/8-83-028A, 4 volumes, Environmental Criteria and Assessment Office, EPA, October 1983.

[2] EPA, Air Quality Criteria Document for Lead, EPA-600/8-77-017, December, 1977.

2.0 ENVIRONMENTAL AND HEALTH EFFECTS OF EXPOSURE

The purpose of this section is to describe the environmental, health, and safety insults, or adverse impacts, which are cited in EPA's Draft Air Quality Criteria Document for Lead. This description includes an identification of subpopulations and geographic areas which appear to be particularly impacted by ambient lead concentrations.

Section 2.1 describes environmental impacts; Section 2.2 describes human health effects in general; and Section 2.3 addresses subpopulations and geographic areas particularly impacted by ambient lead.

2.1 Environmental Impacts

2.1.1 Exposure to Lead

Central to any discussion of the environmental and health effects of lead is an overview of routes of exposure. Figure 2-1 presents a simplified flow diagram of these exposure routes. The circles at the top of the figure are the three distinct sources of lead released to the environment.

As discussed in Section 3, lead from gasoline-powered motor vehicle emissions accounts for about 85 percent of the man-made lead emitted to the environment. Industrial processes (such as smelting) and combustion of fossil fuels account for the remainder denoted by the second circle. Less than 1 percent of total lead emissions result from natural sources such as crustal weathering (third circle) or volcanic explosions.

The main routes of exposure to plants is direct deposition on leaf surfaces and uptake of lead-contaminated soil moisture through plant roots. Humans and animals ingest lead primarily through consumption of plants rather than direct inhalation. Lead is ingested by humans through the eating of plants and animals as food (the processing of food inadvertently adds lead--e.g., soldered seams in cans). Also, some lead is ingested from drinking

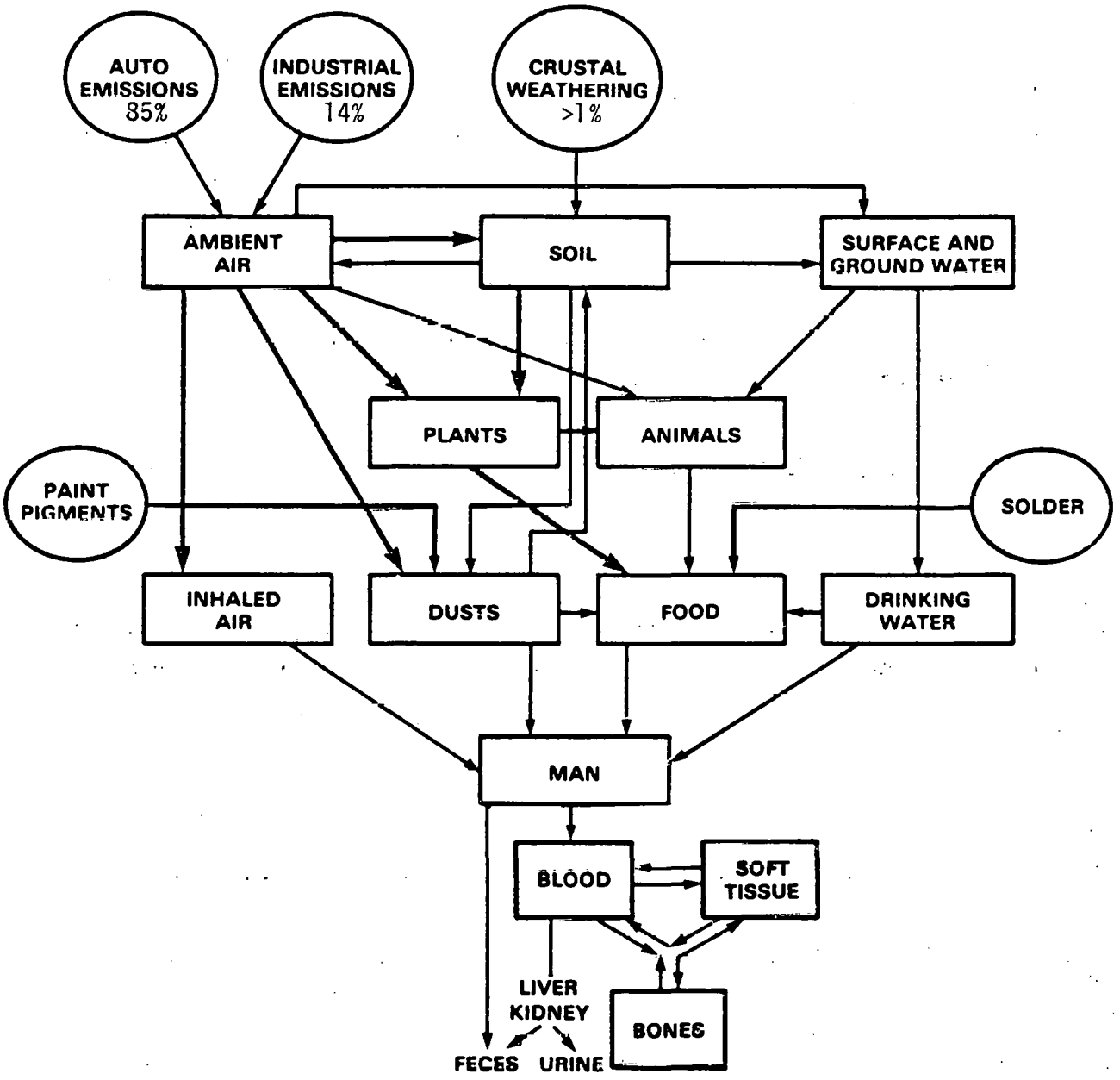

Figure 2-1. Pathways of Lead From the Environment to Man

Source: Air Quality Criteria for Lead, Vol. I, p. 1-2.

water supplies via metal pipes. Medically significant quantities of lead are sometimes ingested through certain occupational exposures and contact with urban dusts which include particles from vehicles and lead paint chips. The latter is a particular concern for children.

Finally, the bottom portion of Figure 2-1 shows that body lead is exchanged among the tissues, blood, and bones. Excretion occurs through feces and urine.

2.1.2 Effects of Lead on Ecosystems

Lead enters the ecosystem primarily through atmospheric emissions but also through other man-made sources such as paint chips, spent ammunition, application of agricultural chemicals, careless disposal of batteries and other industrial lead sources. Lead is not degradable but it may undergo transformations which affect its solubility in water and soils, its bioavailability, or its toxicity. The particular impacts generally associated with lead in plants, animals, and microorganisms are discussed below.

Plants

The most commonly reported effects of lead on plants are the inhibition of the photosynthesis process of respiration and cell elongation, both of which reduce growth. Lead may also reduce the lifespan of plants. The concentrations at which these effects have been detected in laboratories are generally lower than normally found in most rural and urban environments.

The mechanism by which lead accumulates in plants is almost entirely confined to uptake through the roots. Although lead may be deposited on leafy surfaces and although the concentration of lead on these surfaces may be several times that of the lead inside the plant, very little lead enters the plant through the exterior surfaces above the soil. Thus, it is lead in the soil that poses the main threat to plants. Much of this lead may have originally been deposited from the air. Geochemical conditions in the soil create

variations in the lead-binding capacity of the soil. The greater the tendency to bind to soils, the less the likelihood of releasing lead to plants. However, even under the most favorable lead-binding circumstances, most plants will be adversely affected when soil concentrations reach 10 microgram (μg) of lead per gram (g) of dry soil. Concentration approaching this level are often found in the top couple inches of soil near lead smelters and busy traffic intersections.

Acid rain reduces the lead-binding capability of soil. Thus, one of the adverse consequences of acid deposition is the release or mobilization of lead and other trace metals from the soils.

Animals

The major ecological effect of lead at ambient concentrations appears to be on the animals which graze plants and on humans who eat crops, and on microorganisms which decompose plant biomass, rather than on the plants themselves.

For wild and domestic animals, direct inhalation of ambient lead is a minor route of exposure (rarely more than 10 to 15 percent of total). Food is the primary route of exposure. Animals which eat bark and leaves are exposed to higher concentrations of lead than are animals which eat berries, nuts, and fruits (because collection times are less for the latter). Plant tissues and roots typically have an order of magnitude lower concentrations of lead than is found on exterior surfaces. Similarly, carnivores will ingest more lead from consumption of skins and furs than from animal flesh.

The most commonly reported effects of lead on animals are hematological and neurological. Hematological effects include the destruction of red blood cells and the inhibition of the enzyme required to produce red blood

cells. Symptoms of neurological responses are difficult to detect at low exposures but at high exposures include neuromuscular distortion, muscle tremors, and spinal curvature,

While it is impossible to set a safe limit of daily lead consumption, it is reasonable to generalize that a regular diet of 2 to 8 milligram (mg) of lead per day per kilogram (Kg) of body weight over an extended period of time will cause death in most animals. Grazing animals generally ingest the highest concentrations. Near roadsides and near smelters these animals typically consume more than 1 mg of lead per day per Kg of body weight. No documented toxic effects exist for grazing animals ingesting these levels of lead.

Microorganisms

Terrestrial ecosystems, especially forests, accumulate a great deal of cellulose as woody tissues. Few animals can digest cellulose and therefore are dependent upon specialized bacteria to decompose the biomass. An estimated 80 percent of this cellulose must pass through this decomposition food chain. This process is vital for the provision of nutrients to both plants and animals. Lead inhibits and, in some cases, prevents the decomposition process. The concentrations which cause this to occur are not known. Sandy soils, lacking organic compounds, have been shown to suffer 30 percent reduction of decomposition from as little as 750 µg of lead per gram of dry soil, whereas little or no effect was observed at the same level in rich organic soils such as peat soils.

2.1.3 Summary of Environmental Impacts

In summary, the basic effect of lead on plants is to stunt growth. These effects have been documented at levels that are found only near smelters and near major highways. Lead affects the central nervous system of animals and their ability to synthesize red blood cells. The level at which symptoms

are known to have occurred are blood concentrations above 40 µg of lead per deciliter (dl) of whole blood (µg/dl). Perhaps, the most significant non-human environmental impacts of high lead concentrations involve the elimination of populations of bacteria and fungi on leaf surfaces and in the soil thus affecting the nutrient-providing plant decomposition process.

2.2 Effects of Lead on Humans

The biological basis of lead toxicity is its ability to bind with biomolecular substances crucial for various physiological functions. The lead competes with native essential metals for binding sites, thus inhibiting enzyme activity or altering ion transport. These effects occur primarily at the subcellular level in mitochondrion--the body within cells which provides energy for cellular activities. At "relatively moderate" levels of lead exposure, mitochondrion damage in brain and neural tissue has been demonstrated. Young children appear to be particularly sensitive to this effect.

There have been a large number of studies documenting the effect of lead on production of hemoglobin or red blood cells. Hemoglobin is the primary carrier of oxygen in the blood stream. Lead exposure reduces the formation of hemoglobin and makes the molecules which are produced far more fragile and shorter lived than usual. One result is anemia, a condition leading to reduced oxygenation of all parts of the body.

High blood lead levels have been associated with irreversible brain damage accompanied by acute and chronic symptoms of encephalitis. For most adults, this does not occur until blood levels exceed 120 µg of lead per deciliter (dl) of blood. However, acute encephalopathy and death have occurred at lead levels near 100 µg/dl. In children, the threshold is around 80 µg/dl. Symptoms of neurotoxicity are often detectable for adults at levels as low as 40 to 60 µg/dl--well below the previously held "safe" level of 60 to 80 µg/dl.

Little is known about the reversibility of the effect of lead on the central nervous system. Results of occupational studies conflict on this issue; various animal studies indicate that alterations of neurobehavioral functions are long-lived and continue after lead blood levels have returned to normal.

For more than a century it has been known that kidney disease can result from lead poisoning (sometimes at levels of as low as 40 µg/dl). Although the linkage between lead exposure and kidney disease is known, the mechanism for injury is not well established.

Both human and animal studies indicate that lead may affect the survival and development of unborn children (fetuses). Although fetal hemoglobin dysfunction occurs at levels as low as 15 µg/dl of blood, no threshold levels for fetal development have been developed.

Similarly, there are no reliable data indicating whether and to what extent impacts on human reproductivity and future offspring may occur due to lead exposure. Occupational studies indicate that chronic blood levels of 40 to 50 µg/dl appear to result in altered testicular function

Although lead is generally not regarded as a potent carcinogen, statistically significant high cancer rates (respiratory and digestive) have been noted in workers exposed to lead. Also, lead acetate has been clearly demonstrated as a producer of renal tumors and is regarded by the International Agency for Research on Cancer as a carcinogen.

Lead is suspected to have an adverse effect on the body's immune system, thus rendering animals and humans highly susceptible to endotoxins and infectious agents. This may occur at relatively low levels (i.e., 20 to 40 µg/dl blood lead) based on animal studies.

Finally, the cardiovascular, endocrine, and gastrointestinal systems are affected at relatively high levels of lead exposure. However, some studies document gastrointestinal dysfunction at levels as low as 40 to 60 μg/dl.

2.3 Subpopulations and Geographic Areas at Risk

Perhaps more than any other criteria pollutant, the adverse health effects of lead fall most heavily on a particular age group. Compared to adults, young (i.e., preschool) children ingest more lead on a unit body weight basis. Furthermore, they absorb a greater portion of the lead they intake, and retain a greater portion of the lead that is absorbed in their bodies.

Pregnant women are also at greater risk to lead exposure than is the general population. More precisely, it is the unborn child that is at risk but delivery complications resulting from the effects of lead exposure can affect the mother as well.

With respect to geographic areas, the degree of ambient lead exposure is primarily a function of exposure to motor vehicle exhausts and, in a few instances, exposure to plumes from lead-emitting stationary sources. Therefore, one's exposure to lead is primarily related to whether one lives in a rural or urban area. Ambient lead levels may vary by a factor of seven between rural and urban areas due to the difference in exposure to motor vehicle traffic.

2.3.1 Children as a Population-at-Risk

Pre-school-aged children are particularly susceptible to ambient lead exposure because: (1) their behavioral patterns usually result in higher lead intake; and (2) their basic physiologic mechanisms and relatively active metabolisms differ from adults.

Because of young children's "mouthing" habits (e.g., hand-to-mouth and toy-to-mouth) they are much more likely to come into contact with lead-laden dusts. The majority of the lead in dust is deposited from the air; the rest (an average of about 10 percent) is natural. Also, young children's dietary habits include more canned foods and processed baby foods and therefore more contact with lead soldered seams. Although this latter source of lead is not related to ambient air exposures, it raises the baseline lead intake to which children are exposed.

Table 2-1 shows how much more an average two-year-old is exposed to lead in general and atmospheric lead in particular than are adults. The first column shows that the two-year-old takes in 61.4 µg per day. This is somewhat more than the average adult female but somewhat less than the average male. (Note that the child ingests about five times as much dust as the adults.) The second column shows the total lead consumed per kilogram of body weight. Here the increased exposure of children is obvious as the two-year-old consumes 6.15 µg of lead per day per kilogram of body weight compared to about 1.1 µg for adults. Of particular relevance to the setting of an ambient air standard, the third column shows that a larger fraction of the child's lead consumption is attributable to ambient air lead as opposed to lead from food processing, soldered cans, etc. The reason for this is the child's higher consumption of dust.

The lead consumption figures shown in Table 2-1 are baseline data. Much greater consumption totals are plausible under some circumstances. In Table 2-2, the baseline data plus additional exposure possibilities are shown. Note that a two-year-old living near a smelter will consume 2250 additional µg of lead per day from dust alone. This is more than 30 times the baseline total.

Although Table 2-2 indicates a potential exposure of 800 µg/day for a two-year-old due to a family garden, the criteria document text does not discuss the implications of this large potential exposure. Data provided

TABLE 2-1. RELATIVE BASELINE HUMAN LEAD EXPOSURES EXPRESSED PER KILOGRAM BODY WEIGHT*

	Total Lead Consumed	Total Lead Consumed Per Kg Body Wt µg/Kg•Day	Atmospheric Lead Per Kg Body Wt µg/Kg•Day
Child (2 yr old)	(µg/day)		
Inhaled air	0.5	0.05	0.05
Food	28.7	2.9	1.1
Water and beverages	11.2	1.1	0.12
Dust	21.0	2.1	1.9
Total	61.4	6.15	3.17
Adult Female			
Inhaled air	1.0	0.02	0.02
Food	33.2	0.66	0.25
Water and beverages	17.9	0.34	0.04
Dust	4.5	0.09	0.06
Total	56.6	1.13	0.37
Adult Male			
Inhaled air	1.0	0.014	0.014
Food	45.7	0.65	0.25
Water and beverages	25.1	0.36	0.04
Dust	4.5	0.064	0.04
Total	76.3	1.088	0.344

*Body weights: 2 year old child = 10/kg; adult female = 50 kg; adult male = 70 kg.

Source: Air Quality Criteria for Lead, Vol. I, p. 1-127.

TABLE 2-2. SUMMARY OF POTENTIAL ADDITIVE EXPOSURES TO LEAD

	Total Lead Consumed (μg/day)	Atmospheric Lead Consumed (μg/day)	Other Lead Sources (μg/day)
Baseline exposure:			
Child (2 yr old)			
Inhaled air	0.5	0.5	-
Food, water & beverages	39.9	12.1	27.8
Dust	21.0	19.0	2.0
Total baseline	61.4	31.6	29.8
Additional exposure due to:			
urban atmospheres:[1]			
air inhalation	7	7	0
dust	72	71	1
family gardens[2]	800	200	600
interior lead paint[3]	85	-	85
residence near smelter:[4]			
air inhalation	60	60	-
dust	2250	2250	-
secondary occupational[5]	150	-	-
Baseline exposure:			
Adult Male			
Inhaled air	1.0	1.0	-
Food, water & beverages	70.8	20.2	50.6
Dust	4.5	2.9	1.6
Total baseline	76.3	24.1	52.2
Additional exposure due to:			
urban atmospheres:[1]			
air inhalation	14	14	-
dust	7	7	-
family gardens[2]	2000	500	1500
interior lead paint[3]	17	-	17
residence near smelter:[4]			
air inhalation	120	120	-
dust	250	250	-
occupational[6]	1100	1100	-
secondary occupational[5]	21	-	-
smoking	30	27	3
wine consumption	100	?	?

[1]includes lead from household and street dust (1000 μg/g) and inhaled air (.75 $\mu g/m^3$)

[2]assumes soil lead concentration of 2000 μg/g; all fresh leafy and root vegetables, sweet corn of Table 7-15 replaced by produce from garden. Also assumes 25% of soil lead is of atmospheric origin.

[3]assumes household dust rises from 300 to 2000 μg/g. Dust consumption remains the same as baseline. Does not include consumption of paint chips.

[4]assumes household and street dust increases to 25,000 μg/g, inhaled air increases to 6 $\mu g/m^3$.

[5]assumes household dust increases to 2400 μg/g.

[6]assumes 8 hr shift at 10 μg Pb/m^3 or 90% efficiency of respirators at 100 μg/ Pb/m^3. and occupational dusts at 100,000 $\mu g/m^3$.

Source: Air Quality Criteria for Lead, Vol. I, p. 1-128.

indicates soil lead concentrations which vary between 150-500 μg/g near roadways to 10,000 μg/g near houses with exterior lead based paint. The table assumes a soil lead concentration of 2000 μg/g although earlier in the chapter it is noted that soil concentrations above 10 μg/g will adversely affect most plants. The consensus result of 61.4 μg/day total lead consumed for children appears to completely discount this family garden exposure.

Finally, some children are prone to pica--the tendency to eat non-food substances. When these substances include leaded paint chips or dust, lead ingestion levels increase to several thousands or tens of thousands of micrograms per day.

Not only do children ingest much more lead than do adults on a unit weight basis, but lead absorption rates for children are three times that for adults. Added to this is the fact that children are metabolically more active than adults which make them (children) more susceptible to the toxic effects of lead.

In summary, children are exposed to greater amounts of lead than adults, absorb more of the lead they are exposed to, and are more susceptible to adverse health effects from lead than are adults. Also, some children with nutritional deficiencies are afflected with pica and consume much larger amounts of lead.

2.3.2 Other Demographic Variables

Other than age, demographic variables which indicate variations in blood lead level include sex, race, income, and, as noted above, degree of urbanization.

Above the age of 7, males generally have slightly higher blood lead levels than females. Blacks generally have higher blood lead levels than whites or Hispanics. Urban black children under the age of 5 have much

higher blood lead concentrations than any other demographic segment. The second National Health and Nutrition Examination Study (NHANES II) shows that the median blood lead level for black children is 20 µg/dl compared to 13 µg/dl for the U.S. population as a whole (see Table 2-3).

The NHANES study also showed a clear relationship between blood lead level and family income. For both blacks and whites, higher incomes are associated with lower blood lead levels. The racial differences in blood lead levels are decreased at higher income levels.

The primary geographic variable in blood lead concentrations is the degree of urbanization. For the general population, the persons living in urban areas of greater than 1 million population have a blood lead level of 14.0 µg/dl compared to 12.8 for urban areas of less than 1 million and 11.9 for rural areas. Persons living in very remote areas have blood levels of well below 10. The urban/rural differences appear to be almost entirely attributable to leaded gasoline emissions from motor vehicles.

2.4 Regulatory Implications of Environmental and Health Effects of Exposure

The blood lead concentrations at which health effects in both humans and animals are observed are at about 40 µg/dl, and "safe" levels need to be well below this level. This is discussed in more detail in Section 4, but the implication is that from both human health and environmental considerations, the ambient standard should be lowered.

Geographic data indicate that the most serious exposures are in the vicinity of lead smelters and other local point sources. This may be a stimulus to establish more stringent emission standards for these facilities.

TABLE 2-3. WEIGHTED GEOMETRIC MEAN BLOOD LEAD LEVELS FROM NHANES II SURVEY BY DEGREE OF URBANIZATION OF PLACE OF RESIDENCE IN THE U.S. BY AGE AND RACE, UNITED STATES 1976-1980

Race and age	Degree of Urbanization		
	Urban, ≥1 million	Urban, <1 million	Rural
	Geometric mean (µg/dl)		
All races			
All ages	14.0	12.8	11.9
6 months-5 years	16.8	15.3	13.1
6-17 years	13.1	11.7	10.7
18-74 years	14.1	12.9	12.2
Whites			
All ages	14.0	12.5	11.7
6 months-5 years	15.6	14.4	12.7
6-17 years	12.7	11.4	10.5
18-74 years	14.3	12.7	12.1
Blacks			
All ages	14.4	14.7	14.4
6 months-5 years	20.9	19.3	16.4
6-17 years	14.6	13.6	12.9
18-74 years	13.9	14.7	14.9

Source: Annest et al., 1982 as reported in Air Quality Criteria for Lead, Vol. I. p. 1-89.

3.0 SOURCES OF LEAD EMISSIONS

This section summarizes data on the uses and sources of emissions of lead, with an emphasis on lead emissions from energy production and consumption activities.*

3.1 Background

Lead enters the biosphere as a result of natural and anthropogenic processes. However, the contribution of natural processes, 25 x 10^3 metric tons per year (t/y), is small when compared to the contribution of anthropogenic sources, 450 x 10^3 t/y. It is estimated that the ambient natural particulate lead level is less than .0005 $\mu g/m^3$. In contrast, average lead concentrations in urban suspended particulate matter range as high as 6 $\mu g/m^3$. The current national ambient standard is 1.5 $\mu g/m^3$.

Lead is used in the production of a variety of goods. Table 3-1 lists product categories and tons of lead used in production for 1971-1981. In 1981, two product categories, storage batteries and gasoline antiknock additives, accounted for 75 percent of all the lead used in production in the United States. Lead is added to gasoline in the form of two tetraalkyl compounds, tetraethyl and tetramethyl lead. Since 1971, lead usage overall has decreased by 11 percent. Although use in the production of storage batteries has increased, lead used in gasoline dropped by 54 percent from 1971 to 1981.

3.2 Emissions

Lead enters the environment during mining, smelting, processing, use, recycling, and disposal. The atmosphere is the major initial recipient of these emissions. Table 3-2 lists the sources of atmospheric lead in the

*The summary is drawn exclusively from Volume II, Chapter 5, of Air Quality Criteria for Lead.

TABLE 3-1. U.S. UTILIZATION OF LEAD BY PRODUCT CATEGORY (1971-1981), METRIC TONS/YEAR
(U.S. Bureau of Mines, 1981, 1982)

Product category	1971	1972	1973	1974	1975	1976	1977	1978	1979	1980	1981
Storage batteries	616,581	661,740	697,388	772,656	634,368	746,085	858,099	879,274	814,332	645,357	770,152
Gasoline antiknock additives[a]	239,666	252,545	248,390	227,847	189,369	217,508	211,295	178,473	186,945	127,903	111,367
Pigments and ceramics	73,701	80,917	98,551	105,405	71,718	95,792	90,704	91,642	90,790	78,430	80,165
Ammunition	79,423	76,822	73,091	78,991	68,098	66,659	62,043	55,776	53,236	48,662	49,514
Solder	63,502	64,659	65,095	60,116	52,011	57,448	58,320	68,390	54,278	41,366	29,705
Cable coverings	47,998	41,659	39,006	39,387	20,044	14,452	13,705	13,851	16,393	13,408	12,072
Caulking lead	27,204	20,392	18,192	17,903	12,966	11,317	8,725	9,909	8,017	5,684	5,522
Pipe and sheet lead	41,523	37,592	40,529	34,238	35,456	34,680	30,361	23,105	27,618	28,353	28,184
Type metal	18,876	18,089	19,383	18,608	14,703	13,614	11,395	10,795	10,019	8,957	7,838
Brass and bronze	18,180	17,963	20,521	20,172	12,157	14,207	15,143	16,502	18,748	13,981	13,306
Bearing metals	14,771	14,435	14,201	13,250	11,051	11,851	10,873	9,510	9,630	7,808	6,922
Other	56,958	63,124	61,019	62,106	54,524	68,181	64,328	75,517	68,329	50,314	52,354
TOTAL	1,298,383	1,349,846	1,397,376	1,450,679	1,176,465	1,351,794	1,435,497	1,432,744	1,358,335	1,070,303	1,167,101

[a]Includes additives for both domestic and export markets

SOURCE: Air Quality Criteria for Lead, Vol. II, p.5-6.

TABLE 3-2. ESTIMATED ATMOSPHERIC LEAD EMISSIONS FOR THE UNITED STATES, 1981, AND THE WORLD

Source Category	Annual U.S. Emissions (t/yr)	Percentage of Total U.S. Emissions	Annual Global Emissions (t/yr)
Gasoline combustion	35,000	85.9%	273,000
Waste oil combustion	830	2.0	8,900
Solid waste disposal	319	0.8	
Coal combustion	950	2.3	14,000
Oil combustion	226	0.6	6,000
Wood combustion	--	--	4,500
Gray iron production	295	0.7	50,000
Iron and steel production	533	1.3	
Secondary lead smelting	631	1.5	770
Primary copper smelting	30	0.1	27,000
Ore crushing and grinding	326	0.8	8,200
Primary lead smelting	921	2.3	31,000
Other metallurgical	54	0.1	
Zn smelting			16,000
Ni smelting			2,500
Lead alkyl manufacture	245	0.6	
Type metal	85	0.2	
Portland cement production	71	0.2	7,400
Miscellaneous	233	0.5	5,900
TOTAL	40,739[a]	100%	449,170

[a]Inventory does not include emissions from exhausting workroom air, burning of lead-painted surfaces, welding of lead-painted steel structures, or weathering of painted surfaces.

Source: For U.S. emissions, Battye (1983), for global emissions, Nriagu (1979) as reported in Air Quality Criteria for Lead, Vol. II, p. 5-8.

U.S. Most sources, with the exception of primary smelters, are located in areas of high population density. Primary smelters are associated with ore extraction, whereas secondary smelters are involved in recycling. Figure 3-1 shows the location of primary and secondary smelters, mines, refineries, and lead alkyl plants.

As Table 3-2 shows, gasoline combustion accounts for 85.9 percent of the lead emitted annually in the U.S. When coal and gas combustion are included, fossil fuel combustion processes emit approximately 90 percent of the lead discharged to the atmosphere. Figure 3-2 also shows contributions to atmospheric lead of fossil fuel burning activities and other activities, by stationary and mobile source categories.

As Table 3-2 shows, the largest use of lead, which is the production of storage batteries, does not result in correspondingly large discharges to the environment. The data supports the conclusion that fossil fuel combustion, especially in the transportation sector, is the primary contributor to atmospheric lead.

3.3 Mobile Sources

Several reports indicate that motor vehicles account for over 80 percent of the total atmospheric lead loading. Lead is added to gasoline as an antiknock additive to enhance engine performance. It is emitted from vehicles primarily in the form of inorganic particles, although less than 10 percent may be released as volatile organic compounds.

The size of the particle emitted, as well as the total amount emitted, and the chemical composition of the particle as a function of size, are important characteristics of particulate emissions. The mass median equivalent diameter (MMED) is the most commonly used unit of measurement of particle size. It is defined as the point in the size distribution of particles such that half the mass lies on either side of the MMED value. The

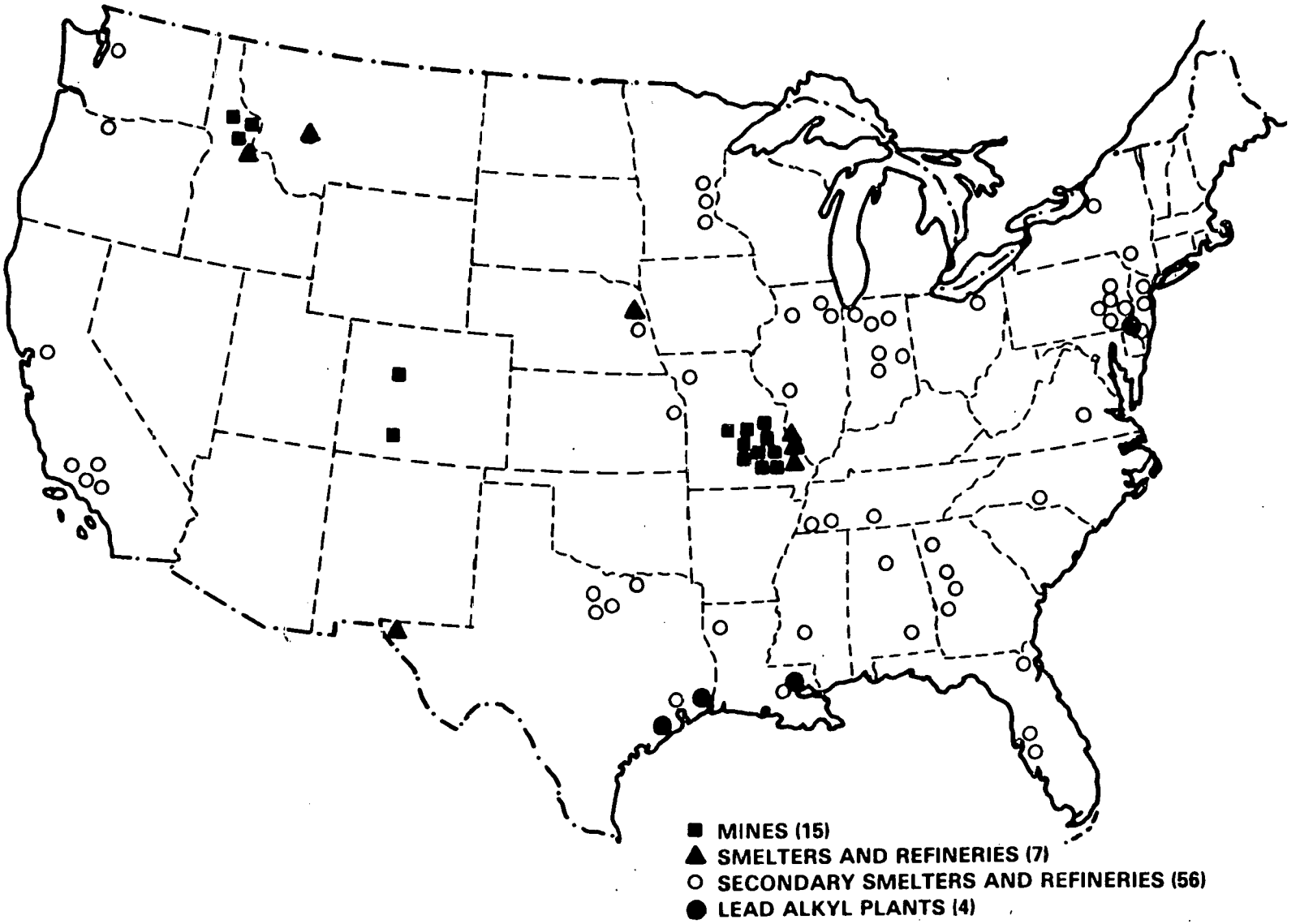

Figure 3-1. Locations of Major Lead Operations in the United States

Source: International Lead Zinc Research Organization (1982) as reported in Air Quality Criteria for Lead, Vol. II, p. 3-5.

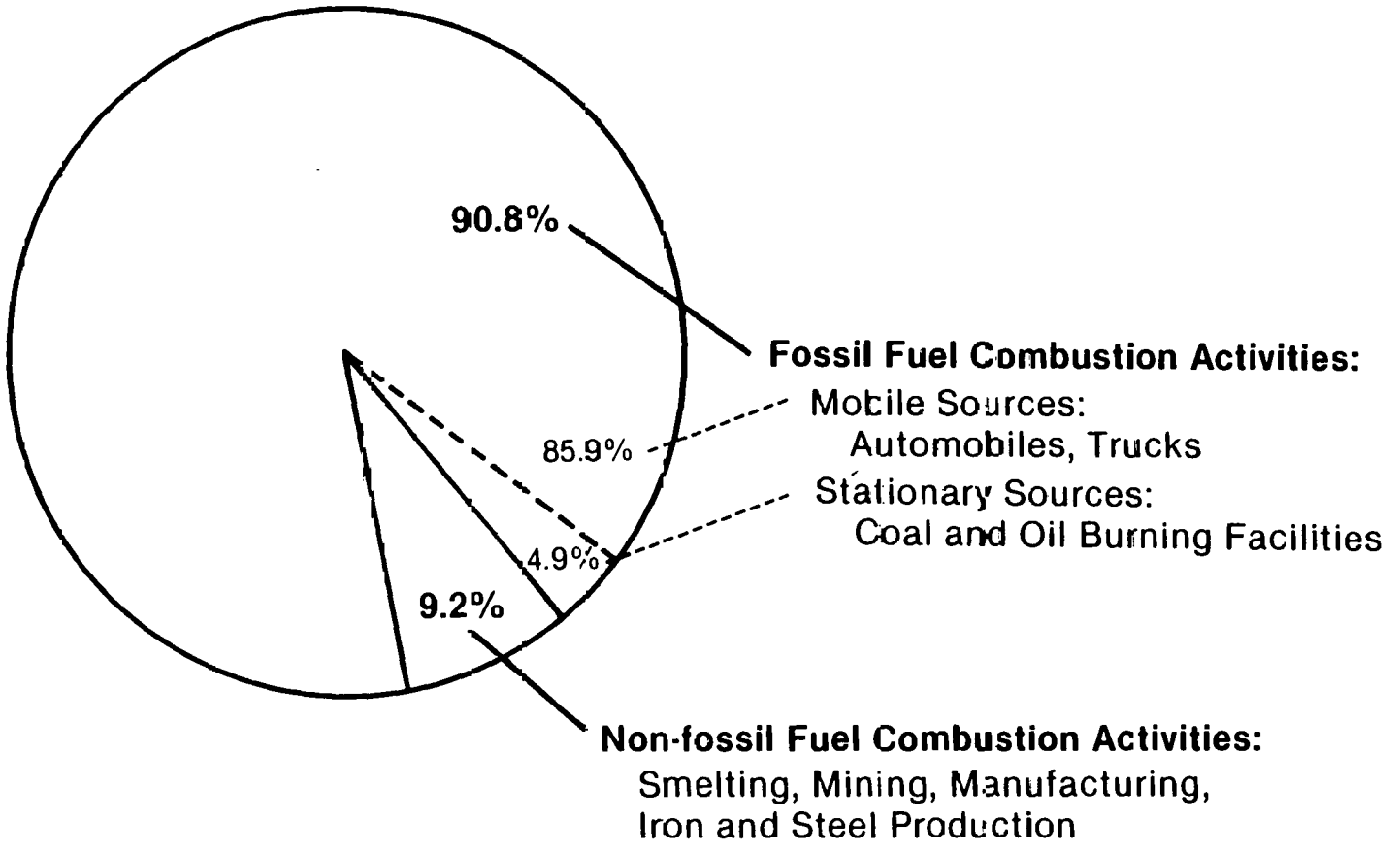

Figure 3-2. Sources of Lead Emissions by Combustion and Non-Fossil Fuel Combustion Activities

size of the lead particle determines its atmospheric fate. Small particles (well under .1 μm in diameter) grow by coagulation. They can remain airborne for 7 to 30 days and travel thousands of miles from their source. Larger particles have limited atmospheric lifetimes.

The estimated MMED of leaded particles from light-duty vehicles is <.25 μm, suggesting a long atmospheric residence time and the potential for long-distance transport. During a vehicle's lifetime, 35 percent of the lead in the gasoline burned by the vehicle will be emitted as small particles (<.25 m MMED), and approximately 40 percent will be emitted as larger particles (>10 μm MMED). Fifteen percent of the lead is deposited in the tailpipe and 10 percent is disposed of in waste oil. Figure 3-3 illustrates the fate of lead in leaded gasoline.

The largest source of lead in the form of volatile organic compounds arises from the manufacture, transport and handling of leaded gasoline. The vapors are photoreactive and have a half-life of less than half a day under typical summertime conditions. Organo lead vapors are most likely to occur in occupational settings.

The use of lead as a gasoline additive is decreasing in importance, due to two regulations (Federal Register, 1973 December 6). The first rule required the availability of unleaded fuel for use in automobiles equipped with catalytic converters which are lead-sensitive emission control devices. The second rule set a standard for the lead content in leaded gasoline (F.R., 1982 October 29). Table 3-3 shows the recent and projected consumption of gasoline lead. Since 1975, leaded gasoline sales have declined and this trend is expected to continue to 1990. The weighted pool total of lead, the total lead in gasoline divided by the total of all gasoline sold (leaded and unleaded) has also declined. After 1982, the weighted pool total of lead is expected to level off at .5 g/gallon of gasoline sold. Under a new rule issued October 1982, the gasoline lead standard will not be based on the weighted pool total; instead, the lead content of leaded gasoline will be limited to 1.1 g/gal. The 1.1 g/gal standard will apply to small and large refineries after July 1983.

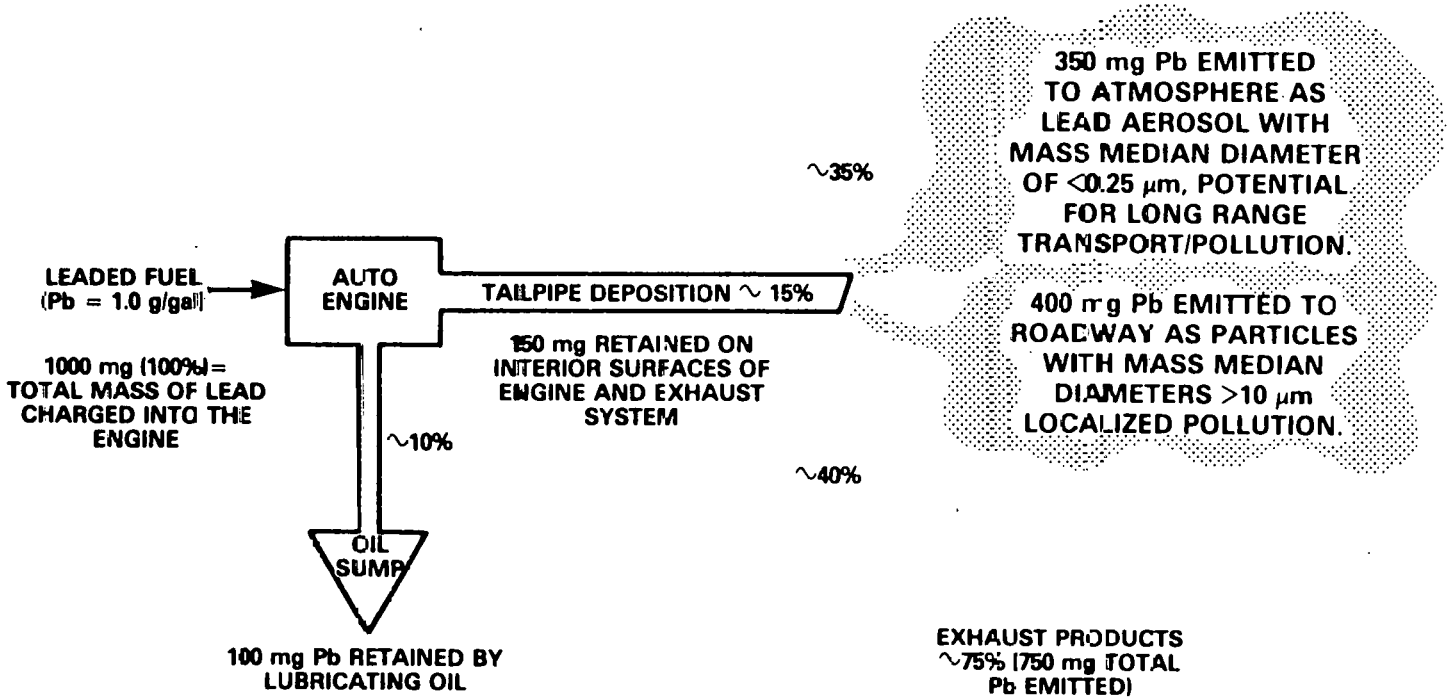

Figure 3-3. Estimated Lead-Only Emissions Distribution Per Gallon of Combusted Fuel
Source: Air Quality Criteria for Lead, Vol. II, p.5-14.

TABLE 3-3. RECENT AND PROJECTED CONSUMPTION OF GASOLINE LEAD

Calendar Year	Gasoline Volume (billions of gallons) Total	Leaded	Average Lead Content (g/gal) Weighted Total Pool	Leaded	Total Lead (10^3 t) 0.5 gpg Pooled Std	1.1 gpg Leaded Std	Air-Lead ($\mu g/m^3$)[d]
1975[a]	102.3	92.5	1.62	1.81	165.6	---	1.23
1976	107.0	87.0	1.60	1.97	171.0	---	1.22
1977	113.2	79.7	1.49	2.14	168.7	---	1.20
1978	115.8	75.0	1.32	2.04	153.3	---	1.13
1979	111.2	68.1	1.16	1.90	129.5	---	0.93
1980	110.8	57.5	0.71	1.37	78.5	---	0.60
1981	102.6	51.0	0.59	1.19	61.0	---	0.47[c]
1982	100.0	40.6	0.64	1.44	62.0	---	0.45[c]
1983[b]	96.1	41.7		1.10	48.1	47.0	
1984	92.3	35.4		1.10	46.1	39.0	
1985	89.2	29.7		1.10	44.6	32.7	
1986	86.1	25.3		1.10	43.0	27.8	
1987	83.8	22.1		1.10	41.9	24.3	
1988	81.5	19.5		1.10	40.7	21.4	
1989	79.2	17.0		1.10	39.6	18.7	
1990	77.7	14.7		1.10	38.8	16.2	

[a]Data for the years 1975-1982 are taken from U. S. Environmental Protection Agency (1983b), in which data for 1975-1981 are actual consumption of lead and for 1982, estimates of consumption.

[b]Data for 1983-1990 are estimates taken from F. R. (1982 October 29, p. 49329).

[c]Estimates (this work).

[d]Data from Hunt and Neligan (1982), discussed in Chapter 7, are the maximum quarterly average lead levels from a composite of sampling sites.

Source: Air Quality Criteria for Lead, Vol. II, p.5-12.

As Table 3-3 also shows, the atmospheric lead concentration has declined since 1975. This is discussed further in Section 4.

3.4 Stationary Sources

The leading stationary sources of lead emissions are facilities which burn coal and oil, primary and secondary lead smelters and iron and steel plants. Together, these sources contribute less than 10 percent to the total atmospheric lead loading. Loading from all stationary sources is estimated at 14 percent.

Contamination of soil and water also occurs directly due to leaching of mine and smelter wastes. Quantitative estimates of the extent of contamination are not available. Spillage along transportation routes is known to result in significant contamination. On a southeastern Missouri roadside, lead levels in leaf litter were measured at from 2000 to 5000 µg/g. Ten mines and three lead smelters are located in southeastern Missouri.

3.5 Regulatory Implications of Sources of Lead Emissions

Despite regulations controlling the lead content of gas and the use of unleaded fuels, the contribution of mobile sources to atmospheric lead loading has remained at 85 percent. From 1971 to 1981, reductions in stationary source loadings kept pace with reductions in emissions from mobile sources, leaving little change in their relative contributions. The atmospheric loading from gasoline lead is expected to decline further, as the use of unleaded gasoline grows. The ratio of mobile source to stationary source emissions remaining constant suggests that reductions in the ambient standard will be implemented with additional emission controls for both mobile and stationary sources.

4.0 AMBIENT AIR LEAD CONCENTRATIONS

This section examines the data reported on lead concentrations in air. The effects of regulations which limit lead in gasoline and require the availability of unleaded gasoline are also discussed. In addition, this section addresses the issue of the placement of monitors for measuring ambient air lead concentrations.

As discussed earler, air provides one pathway for human exposure. The other major pathways for human exposure are soil, ground and surface water, and food. Figure 4-1 shows the pathways of human exposure to lead. Soil and water are contaminated as a result of settling of airborne lead.

4.1 Lead Concentrations in Air

Data support the conclusion that lead concentrations in rural and urban areas in the U.S. decreased in the late 1970s. Table 4-1 shows the changes in ambient lead concentrations for urban areas for which valid annual averages were available from 1970-80. In 1980, 99 percent of the urban stations were in compliance with the 1.5 $\mu g/m^3$ ambient air lead standard. Nearly all of the stations had annual averages below 1.0 $\mu g/m^3$. The maximum quarterly average for urban stations fell below 1.5 $\mu g/m^3$ for the first time in 1980.

As Figure 4-2 shows, the number of stations reporting concentration levels above .5 $\mu g/m^3$ fell dramatically after 1977. This drop could be explained in part by the reduction in the number of monitoring stations included in the network. From 1970 to 1980, the number of monitoring stations was reduced from 797 to 220. The most common reason for removing stations from the monitoring network was compliance with the air quality standard of 1.5 $\mu g/m$. Most of the stations removed were in areas with

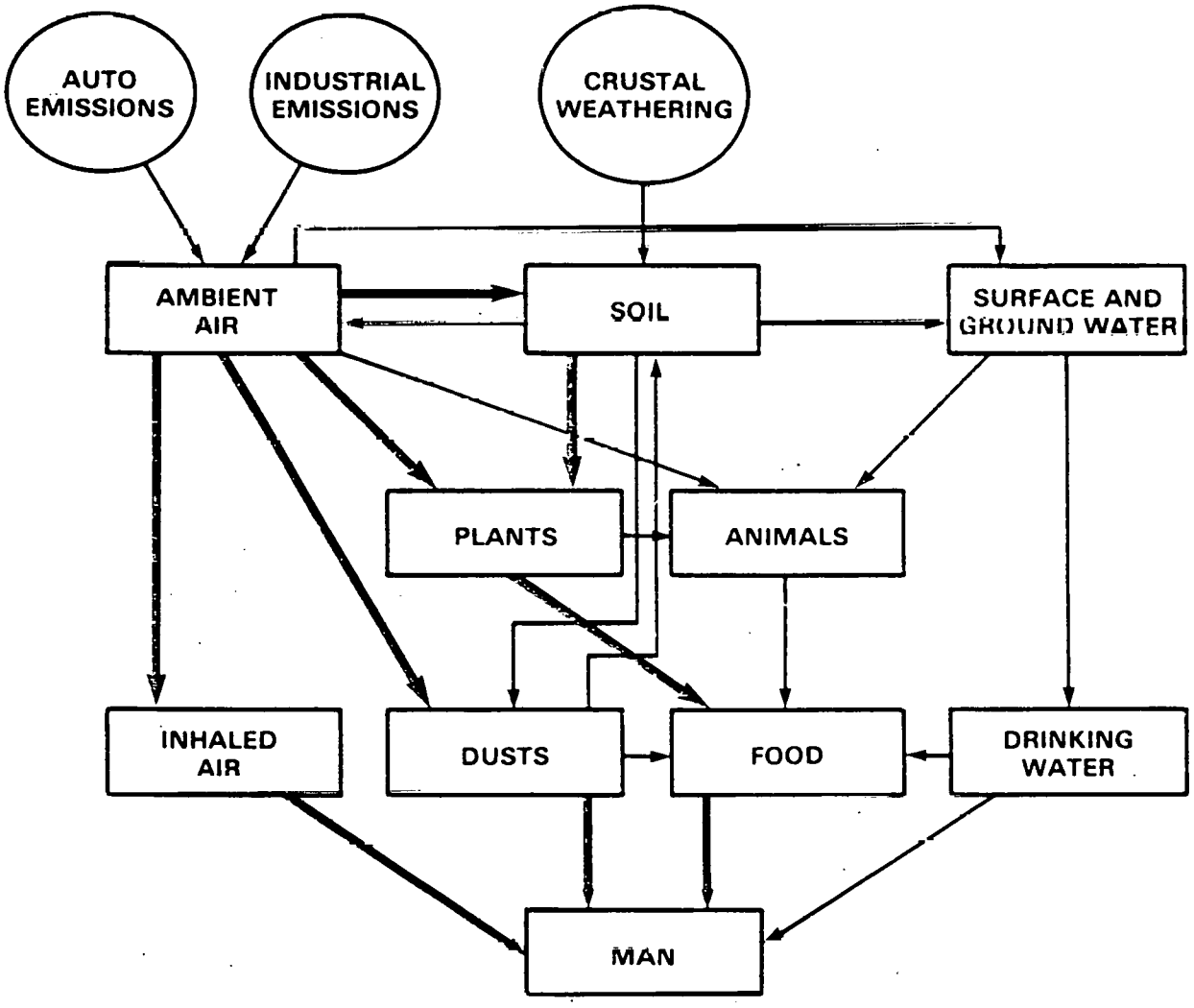

Figure 4-1. Pathways of Lead from the Environment to Human Consumption. Heavy Arrows Are Those Pathways Discussed in Greatest Detail in this Chapter.

Source: Air Quality Criteria for Lead, Vol. II, p. 7-2.

TABLE 4-1. CUMULATIVE FREQUENCY DISTRIBUTIONS OF URBAN AIR LEAD CONCENTRATIONS*

Year	No. of Station Reports	Percentile 10	30	50	70	90	95	99	Max. Qtrly. Avg.	Arithmetic Mean	Arithmetic Std. Dev.	Geometric Mean	Geometric Std. Dev.
1970	797	0.47	0.75	1.05	1.37	2.01	2.59	4.14	5.83	1.19	0.80	0.99	1.80
1971	717	0.42	0.71	1.01	1.42	2.21	2.86	4.38	6.31	1.23	0.87	1.00	1.89
1972	708	0.46	0.72	0.97	1.25	1.93	2.57	3.69	6.88	1.13	0.78	0.93	1.87
1973	559	0.35	0.58	0.77	1.05	1.62	2.08	3.03	5.83	0.92	0.64	0.76	1.87
1974	594	0.36	0.57	0.75	1.00	1.61	1.97	3.16	4.09	0.89	0.57	0.75	1.80
1975	695	0.37	0.58	0.78	0.96	1.54	2.02	3.15	4.94	0.89	0.59	0.74	1.82
1976	670	0.37	0.58	0.74	0.96	1.41	1.72	3.07	4.54	0.85	0.55	0.72	1.80
1977	533	0.37	0.57	0.75	0.95	1.67	2.13	3.29	3.96	0.91	0.80	0.68	1.79
1978	282	0.27	0.43	0.57	0.74	1.19	1.49	2.40	3.85	0.68	0.64	0.50	1.87
1979	167	0.22	0.33	0.43	0.63	1.09	1.33	2.44	3.59	0.56	0.58	0.39	1.89
1980	220	0.14	0.21	0.30	0.38	0.55	0.66	0.84	1.06	0.32	0.27	0.24	1.88

*The data reported here are all valid quarterly averages reported from urban stations from 1970 to 1980, in $\mu g/m^3$. The vertical line marks compliance with the 1978 1.5 $\mu g/m^3$ EPA National Ambient Air Quality Standard. In 1980, the quarterly average for all but the highest 1 percent of the stations was 0.84. The sources of the data are Akland, 1976; U.S. EPA, 1979; Quarterly averages of lead from NFAN, 1982, as reported in Air Quality Criteria for Lead, Vol. II, p. 7-7.

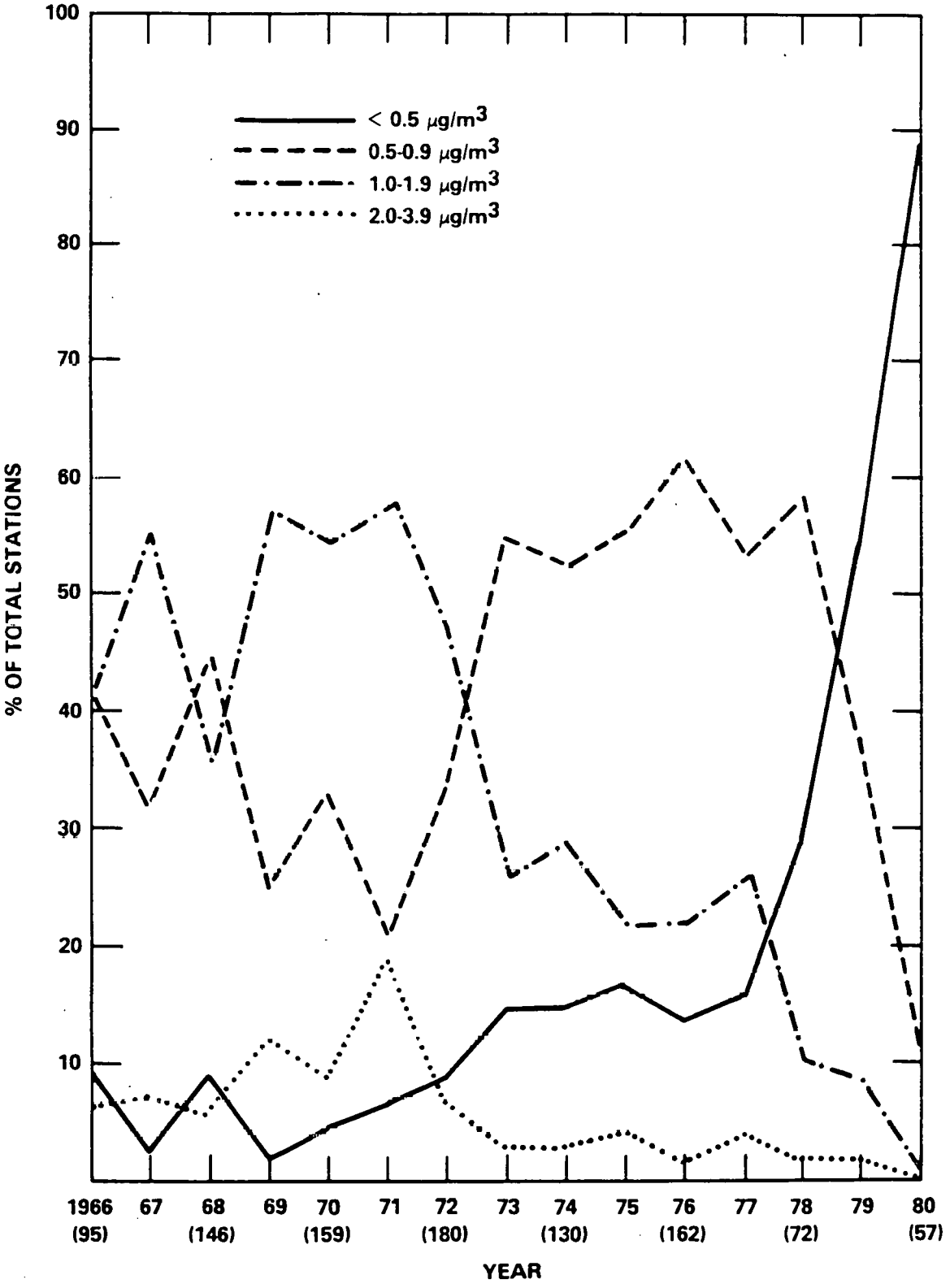

Figure 4-2. Percent of Urban Stations Reporting Indicated Concentration Interval

Source: Air Quality Criteria for Lead, Vol. II, p. 7-6.

concentrations below 1.0 $\mu g/m^3$. Also, data at individual stations confirm the apparent national trend of lower lead concentrations.

Concentrations for stations in five urban areas are depicted in Figure 4-3. Since 1976, the five areas have shown a decrease in lead levels. The peaks in the plots are the result of seasonal variations in dispersion capacity.

In non-urban areas, concentrations are approximately one-seventh of urban area concentrations. However, the concentrations for 1979 and 1980 showed percent reductions comparable to urban area decreases. The maximum quarterly average lead level decreased from 1.471 $\mu g/m^3$ in 1970 to .13 $\mu g/m^3$ in 1980.

4.2 Effects of Clean Air Regulations

The national trend toward lower air lead concentrations is attributed to the decreasing lead content of leaded gasoline and the increasing use of unleaded fuel. The linear correlation between lead consumed in gasoline and reported average quarterly ambient lead concentrations is .99, showing an almost perfect correspondence. Figure 4-4 illustrates ambient lead concentrations and lead in gasoline for 1975-1982. Over the period, lead consumed in gasoline decreased 52 percent while the ambient lead concentration decreased 51 percent.

The effect of the 1978 National Ambient Air Quality Standard for lead has been a reduction in lead concentration levels in major urban areas. The data reflect the impact of both stationary and mobile sources. Table 4-2 shows the maximum quarter lead concentrations for stations reporting four valid quarters in 1979-1981, by type of source. Fifty-seven percent of all sites reported lead concentrations under .5 $\mu g/m^3$. Only five percent of the

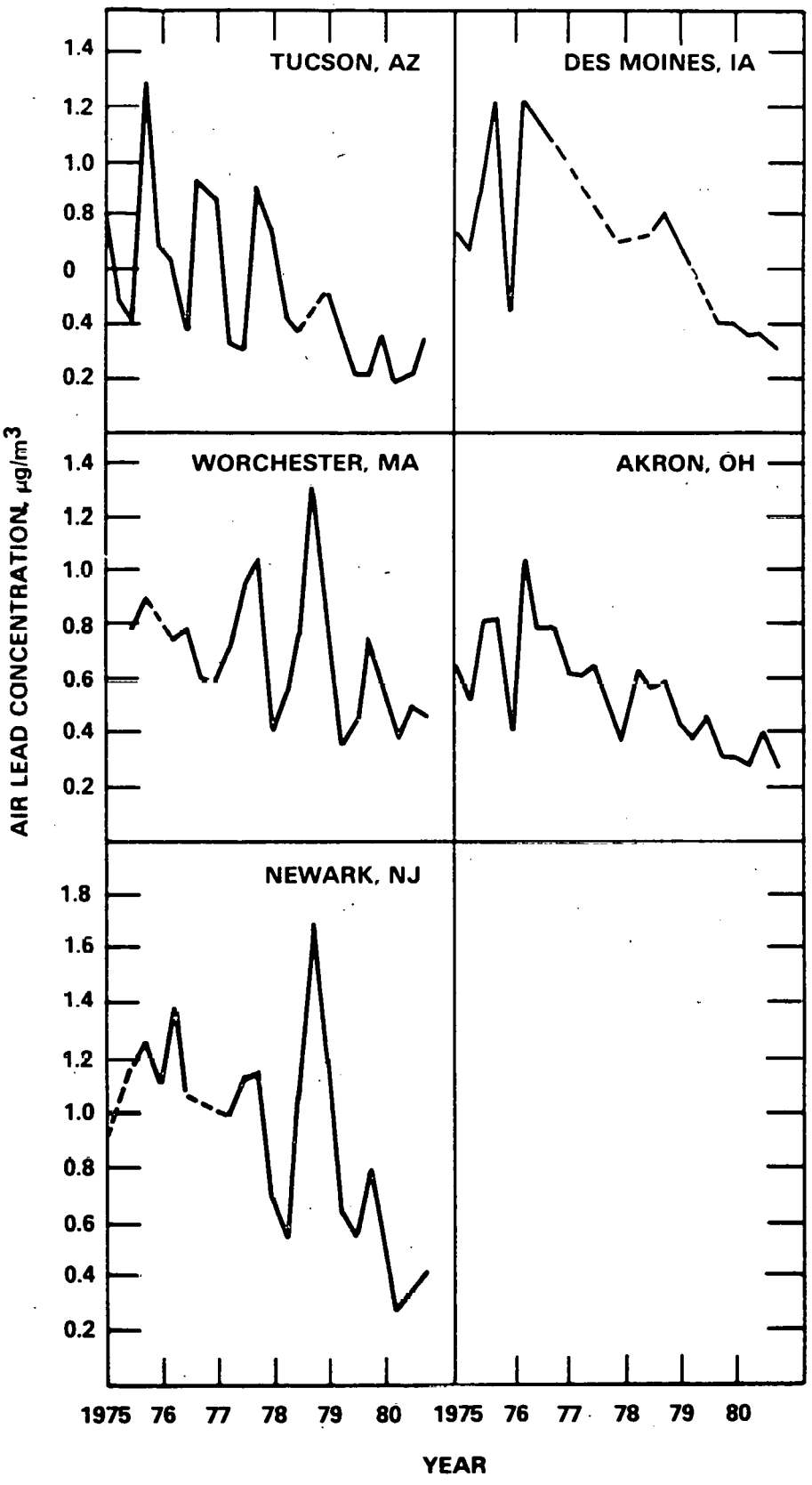

Figure 4-3. Time Trends in Ambient Air Lead at Selected Urban Sites
Source: Air Quality Criteria for Lead, Vol. II, p. 7-12.

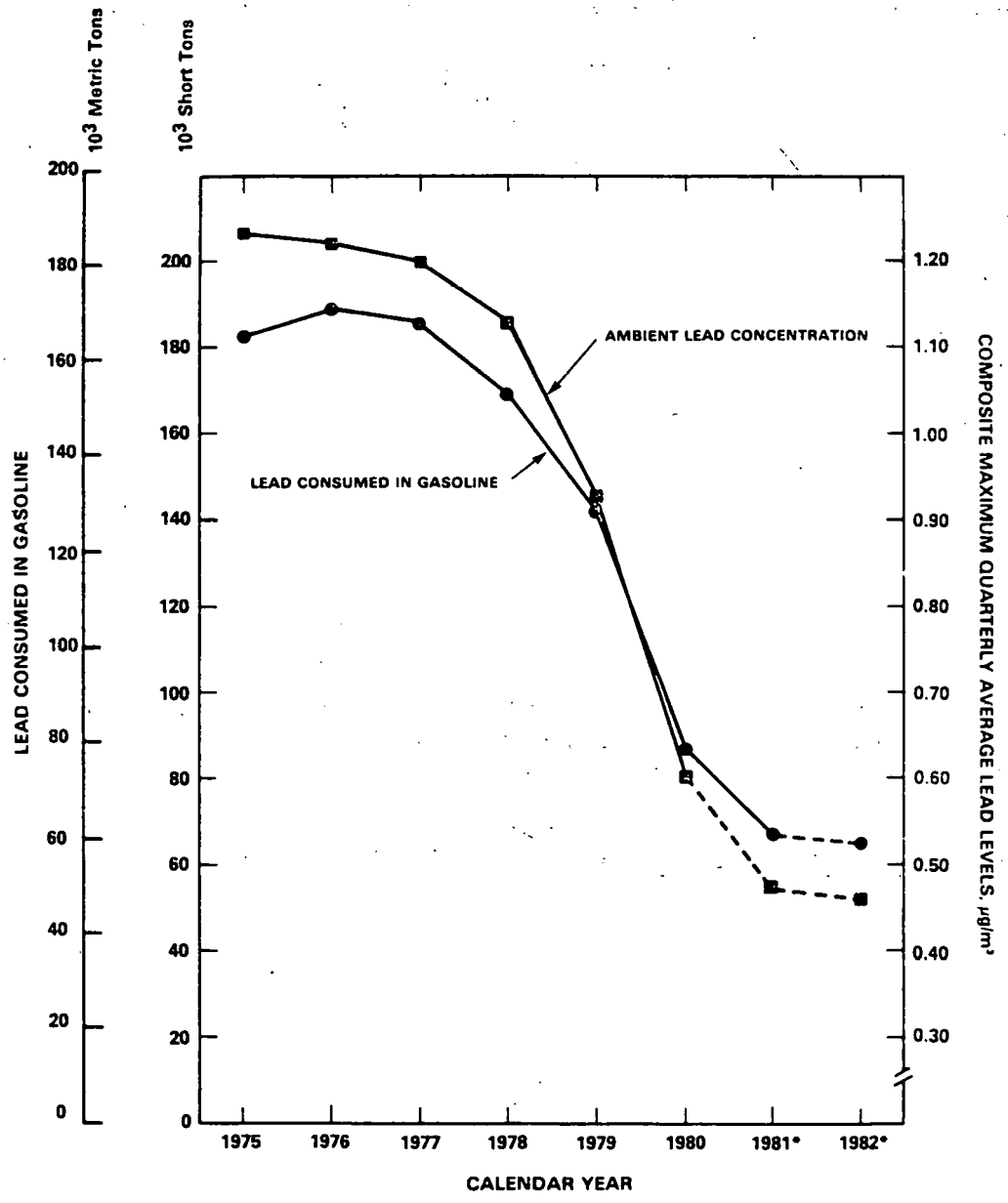

Figure 4-4. Lead Consumed in Gasoline (DuPont, 1982) and Ambient Lead Concentrations, 1975-1982. (Hunt and Neligan, 1982). (Dashed lines are estimates.)

Source: Air Quality Criteria for Lead, Vol. II, p. 5-18.

TABLE 4-2. DISTRIBUTION OF AIR LEAD CONCENTRATIONS BY TYPE OF SITE

Site-Type	Concentration Ranges ($\mu g/m^3$) ≤.5	>.5 ≤1.0	>1.0 ≤1.5	>1.5 ≤2.0	>2.0	Total No. of Site Years
Population	300	173	46	7	5	531
Stationary Source	50	12	10	2	21	95
Background	21	0	0	0	0	21
Total (site-years)	371	185	56	9	26	647
Percent of sites in concentration range	57%	29%	9%	1%	4%	100%

Data are the number of site years during 1979-81 falling within the designated quarterly average concentration range. To be included, a site year must have four valid quarters of data.

Source: Air Quality Criteria for Lead, Vol. II, p. 7-19.

sites reported concentrations above 1.5 $\mu g/m^3$. Monitoring sites near stationary sources accounted for 80 percent of the sites with lead concentrations over 2 $\mu g/m^3$ and 66 percent of the sites with concentrations above 1.5 $\mu g/m^3$. It is anticipated that the distribution reflected in Table 4-2 will change as new guidelines for urban monitoring stations are implemented. Under guidelines issued in 1981, monitors will be located closer to roadways.

4.3 Monitoring

Under new guidelines issued July 1981, air lead monitors are to be placed between 5 and 15 meters from the thoroughfares and 2 to 7 meters above the ground. The new sites, called "microscale" sites, will be located closer to roadways than has been the practice in the past. Correspondingly, these sites might be expected to show higher lead concentrations than more distant middlescale monitors, due to vertical gradients in concentrations nearer a source.

One study shows, in a limited way, the relationship between microscale measured concentrations and lead concentrations at more distant monitors. A microscale monitor at 5 meters from the road and 2 meters above ground would be expected to show concentrations 20 percent higher than concentrations at monitors 21.4 meters from the roadway. However, the difference in concentrations decreases as the distance from the source increases. For example, lead concentrations 2.8 meters from the roadway are a function of height, whereas average concentrations at different heights converge rapidly with greater distance form the source. It appears that distance from source, either horizontally or vertically, can be the primary factor determining ambient concentrations. Overall, the addition of microscale monitors to the national monitoring network can be expected to shift measured concentrations toward higher values for monitors near roadways.

4.4 Exposure

Other factors enter into the calculation of human exposure to airborne lead. Ambient air lead concentrations may not be an accurate measure of actual exposure as people spend much of their time indoors. The average ratio of indoor lead concentrations to outdoor concentrations is .6 to .8 for houses without air conditioning and .3 to .5 for air conditioned houses. Fixed monitors, whether inside or outside, do not fully measure human exposure. One exposure study showed higher lead levels measured by personal exposure monitors. The personal monitors averaged .16 $\mu g/m^3$ while fixed monitors indicated lead concentrations of .092 and .12 $\mu g/m^3$.

The effects of inhaled lead on human health are a function of the size of the lead-containing particles. Size also affects the length of time a particle will remain airborne, as discussed in Section 3.0.

Most of the airborne lead mass is associated with small particles. Figure 4-5 summarizes airborne lead particle size data from the literature. Many of the distributions also show a distinct peak in their upper ends. The peaks in the distribution are the result of two types of sources. The small particles are from nucleation of vapor phase lead emissions, mostly from automobiles. The larger particles are emitted from combustion or mechanical processes. The distribution figures shown in Figure 4-5 have been used to estimate the fraction of inhaled airborne lead deposited in the human respiratory system. Although gathered under varying circumstances with different instruments, the information displayed in Figure 4-5 is the most extensive size distribution data available.

4.5 Regulatory Implications of Ambient Air Lead Concentrations

Responding to changes in the amount of lead used in gasoline, lead concentrations in rural and urban areas in the U.S. decreased in the late 1970s. In 1980, 99 percent of the urban stations were in compliance with

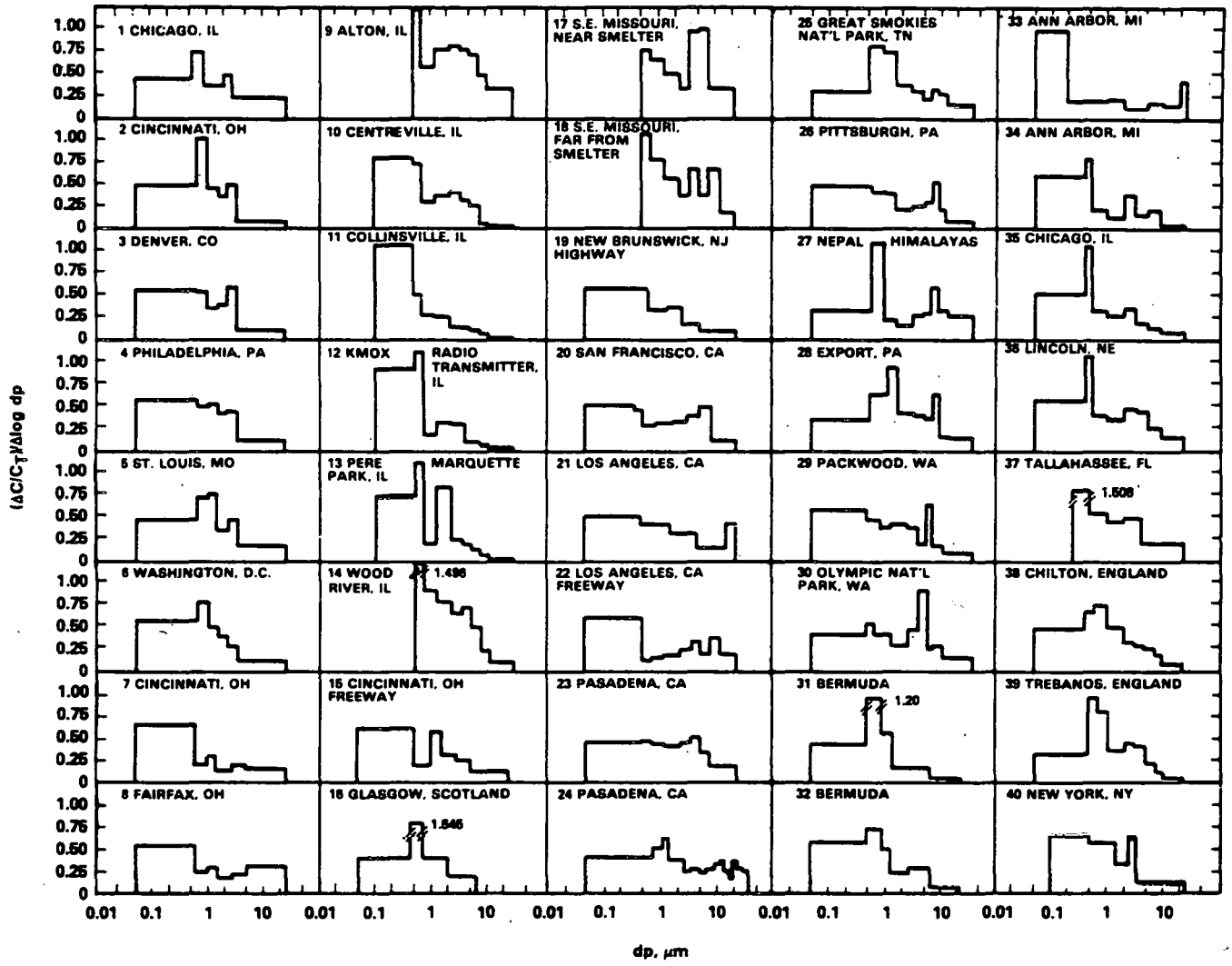

Figure 4-5. Airborne Mass Size Distributions for Lead Taken from the Literature. ΔC represents the Airborne Lead Concentration in each Size Range, C_T is the Total Airborne Lead Concentration in All Size Ranges, and d_p is the Aerodynamic Particle Diameter.

Source: Air Quality Criteria for Lead, Vol. II, p. 7-21

the 1.5 $\mu g/m^3$ ambient air standard. The reduced lead concentrations cannot be attributed to a reduction in the number of monitoring stations. Data at individual stations confirm the apparent national trend of lower lead concentrations. The national trend is attributed to the decreasing lead content of gasoline and the increasing use of unleaded fuels. Eighty percent of sites with concentrations over 2 $\mu g/m^3$ are near stationary sources. A more stringent standard will make compliance that much more difficult for these sources, and could result in closure of these facilities. This continuing trend in ambient concentrations suggests that implementing a more stringent standard will only be possible with further reductions in leaded gasoline use.

Guidelines for locating air monitors are expected to result in the measurement of increased levels of lead. The new monitoring stations, to be located closer to roadways than previous stations, might be expected to show higher lead concentrations. Changes in lead concentrations are estimated to be 20 percent or less. There appear to be no significant regulatory implications to this relocation.

5.0 DOSE-EFFECT RELATIONSHIPS

5.1 Background

In the setting of an ambient standard, EPA attempts to identify a threshold below which no significant adverse health effects are observed. Ideally, the standard is set below this threshold to leave a margin of safety. The purpose of dose-effect studies is to provide a data base upon which a threshold can be determined. This section summarizes the dose-effect findings of the lead criteria document.

The most readily measured index of lead content in the body is blood lead concentration. Blood lead concentrations are frequently measured in micrograms of lead per deciliter of whole blood (µg/dl).

Table 5-1 summarizes the lowest blood lead levels at which various health effects have been observed for adults. These health effects include:

- Hematological or blood-related effects (first column),
- Neurological effects (second column),
- Renal (or kidney) effects,
- Reproductive effects, and
- Gastrointestinal effects.

From the table it is obvious that a clear threshold for three of the five health effects occurs at 40 µg/dl with slowed nerve conduction observed 25 to 30 µg/dl. The lowest threshold exists for hematological effects with some effects occurring at less than 10 µg/dl.

TABLE 5-1. SUMMARY OF LOWEST OBSERVED EFFECT LEVELS FOR KEY LEAD-INDUCED HEALTH EFFECTS IN ADULTS

Lowest Observed Effect Level (PbB)	Heme Synthesis and Hematological Effects	Neurological Effects	Renal System Effects	Reproductive Function Effects	Gastrointestinal Effects
100-120 µg/dl		Encephalopathic signs and symptoms	Chronic renal nephropathy		Overt gastrointestinal symptoms (colic, etc.)
80 µg/dl	Frank anemia				
60 µg/dl		?			
50 µg/dl	Reduced hemoglobin production	Overt subencephalopathic neurological symptoms		Altered testicular function	
40 µg/dl	Increased urinary ALA and elevated coproporphyrins	?			
30 µg/dl		Peripheral nerve dysfunction (slowed nerve conduction)			
25-30 µg/dl	Erythrocyte protoporphyrin (EP) elevation in males	?			
15-20 µg/dl	Erythrocyte protoporphyrin (EP) elevation in females				
<10 µg/dl	ALA-D inhibition				

Abbreviations: PbB = blood lead concentrations.

Source: Air Quality Criteria for Lead, Vol. I, p. 1-19.

As discussed earlier, children are more susceptible to the toxic effects of lead than are adults. Table 5-2 shows the observed thresholds of five categories of health effects for children. These categories are similar to those for adults except that there is no category for reproductive effects in children. Instead, the fifth column in Table 5-2 addresses "other biochemical effects." Three of the five categories of effects show either very low or no thresholds.

As with adults, the most serious of the health effects is severe, irreversible central nervous system damage. In children the blood lead levels associated with severe symptoms of encephalitis (including death) start at about 80 to 100 µg/dl compared to 100 to 120 µg/dl for adults.

The criteria document states that "all of these effects are reflective of widespread impact of lead on normal physiological functioning of many different organ systems in children at blood lead levels at least as low as 40 µg/dl." In addition, the document concedes that although methodological problems prevent conclusive acceptance of various neurobehavioral studies, these studies are indicative of association between neuropsychologic deficits and blood lead levels at least as low as 30 µg/dl.

The body's production of hemaglobin seems to be affected by much lower lead levels than was previously throught for both adults and children. This, taken together with new evidence that neurological damage for children especially occurs at very low levels, led the authors of the criteria document to the conclusion that "the rationale for continuing to view 30 µg/dl as a 'maximum safe' blood-lead level is called into question and substantial impetus is provided for revising the criteria downward, i.e., to some blood-lead level below 30 µg/dl."

Thus, it appears that the criteria document is setting the stage for a more stringent standard.

TABLE 5-2. SUMMARY OF LOWEST OBSERVED EFFECT LEVELS FOR KEY LEAD-INDUCED HEALTH EFFECTS IN CHILDREN

Lowest Observed Effect Level (PbB)	Heme Synthesis and Hematological Effects	Neurological Effects	Renal System Effects	Gastrointestinal Effects	Other Biochemical Effects
80-100 µg/dl		Encephalopathic signs and symptoms	Renal dysfunction (aminoaciduria)	Colic, other overt gastrointestinal symptoms	
70 µg/dl	Frank anemia				
60 µg/dl		?			
50 µg/dl					
40 µg/dl	Reduced hemoglobin	Cognitive (CNS) deficts			
	Elevated coproporphyrin	Peripheral nerve dysfunction (slowed NCV's)			
	Increased urinary ALA				
30 µg/dl					Vitamin D metabolism interference
15-20 µg/dl	Erythrocyte protoporphyin elevation	CNS electrophysiological deficits			
10	ALA-D inhibition	?			Py-5-N activity inhibition
	?				

Abbreviations: PbB = blood lead concentrations; Py-5-N = pyrimidine-5'-nucleotidase.

Source: Air Quality Criteria for Lead, Vol. I, p. 1-141.

5.2 Linking Blood Lead Levels to Ambient Levels

Even if a consensus threshold blood lead level were to emerge from the medical studies of health effects of lead, regulators would be left with the difficult problem of linking ambient lead levels with the maximum blood lead level considered safe for the most susceptible populations at risk (i.e., pre-school children).

The key question is: What is the relationship between ambient lead levels and blood lead levels? Analyses of this relationship are complicated by the fact that lead does not remain suspended in the atmosphere but instead falls to the ground where it is incorporated into the soil, dust, and water and enters the food chain over time. Since humans are exposed to lead from all of these media, studies which relate air lead levels to blood lead levels may underestimate the overall impact of air lead on blood lead levels.

Although there is a great deal of variability in the results of studies attempting to establish such a relationship, the authors of the criteria document conclude that:

- At air lead exposures of 3.2 $\mu g/m^3$ or less, the relationship between air and blood level appears to be linear.

- EPA analyses of population studies suggest that for children: for every 1 $\mu g/m^3$ increase in air lead levels, blood lead levels increase by approximately 2 $\mu g/dl$.

- This relationship, or inhalation slope, is somewhat less for adults, averaging around 1.5 $\mu g/dl$ per $\mu g/m^3$.

These data do not include the lead ingested by means other than direct inhalation (i.e., lead deposited on crops from the air and later ingested as food). The relationship between total air lead ingested and blood lead levels is not clearly indicated in the document.

5.3 Importance of Specific Source Categories

As discussed in Section 3.0, the combustion of leaded gasoline is the primary contributor to ambient lead levels in the U.S. It is not surprising to find that as the use of leaded gasoline has been phased down, the levels of blood lead have also decreased. In Table 5-3, the blood lead levels for 1- to 4-year-old urban black children (the most highly exposed population group) are shown for the years 1970 through 1977. This period corresponds with efforts to begin reducing the lead content in gasoline. The table indicates that the high summer blood lead levels (>34 µg/dl) decreased from an average of 42 percent in 1970 to 7.2 percent in 1977. Similarly, Figure 5-1 shows how average blood lead levels for the U.S. population as a whole have decreased from between 15 and 20 µg/dl in 1976 and 1977 to less than 10 µg/dl in 1980.

It appears likely that as sales of leaded gasoline continue to decline in response to the replacement of noncatalytic converter equipped cars with catalyst equipped cars, stationary sources such as primary and secondary smelters, will become the only major sources of ambient airborne lead. However, much of the previously emitted lead will continue to be a source of lead contamination of food crops, dust, and ambient air (through reentrainment) for many years.

With respect to energy-using versus energy-producing sources, virtually all of the lead emissions result from energy consumption. However, lead emissions from coal mining, coal-fired power plants, and other "energy producers" are negligible compared to motor vehicles and stationary energy-using sources.

5.4 Regulatory Implications of Dose Response Data

The implications of the more recent dose response studies is that the current ambient standard is likely to be made more stringent. This implication is not stated explicitly in the criteria document. Rather, it

TABLE 5-3. DISTRIBUTION OF BLOOD LEAD LEVELS FCR 13 TO 48 MONTH OLD BLACKS BY SEASON AND YEAR* FOR NEW YORK SCREENING DATA

Year	January - March Percent			July - September Percent		
	<15µg/dl	15 to 34µg/dl	>34µg/dl	<15µg/dl	15 to 34µg/dl	>34µg/dl
1970	(insufficient sample size)			3.4	54.7	42.0
1971	3.8	69.5	26.7	1.3	56.0	42.7
1972	4.4	76.1	19.5	4.3	72.2	23.4
1973	7.3	80.3	12.4	2.7	62.4	34.9
1974	9.2	73.8	17.0	8.2	65.4	26.4
1975	11.1	77.5	11.4	7.3	81.3	11.4
1976	21.1	74.1	4.8	11.9	75.8	12.3
1977	28.4	66.8	4.8	19.9	72.9	7.2

Source: Air Quality Criteria for Lead, Vol. III, p. 11-21.

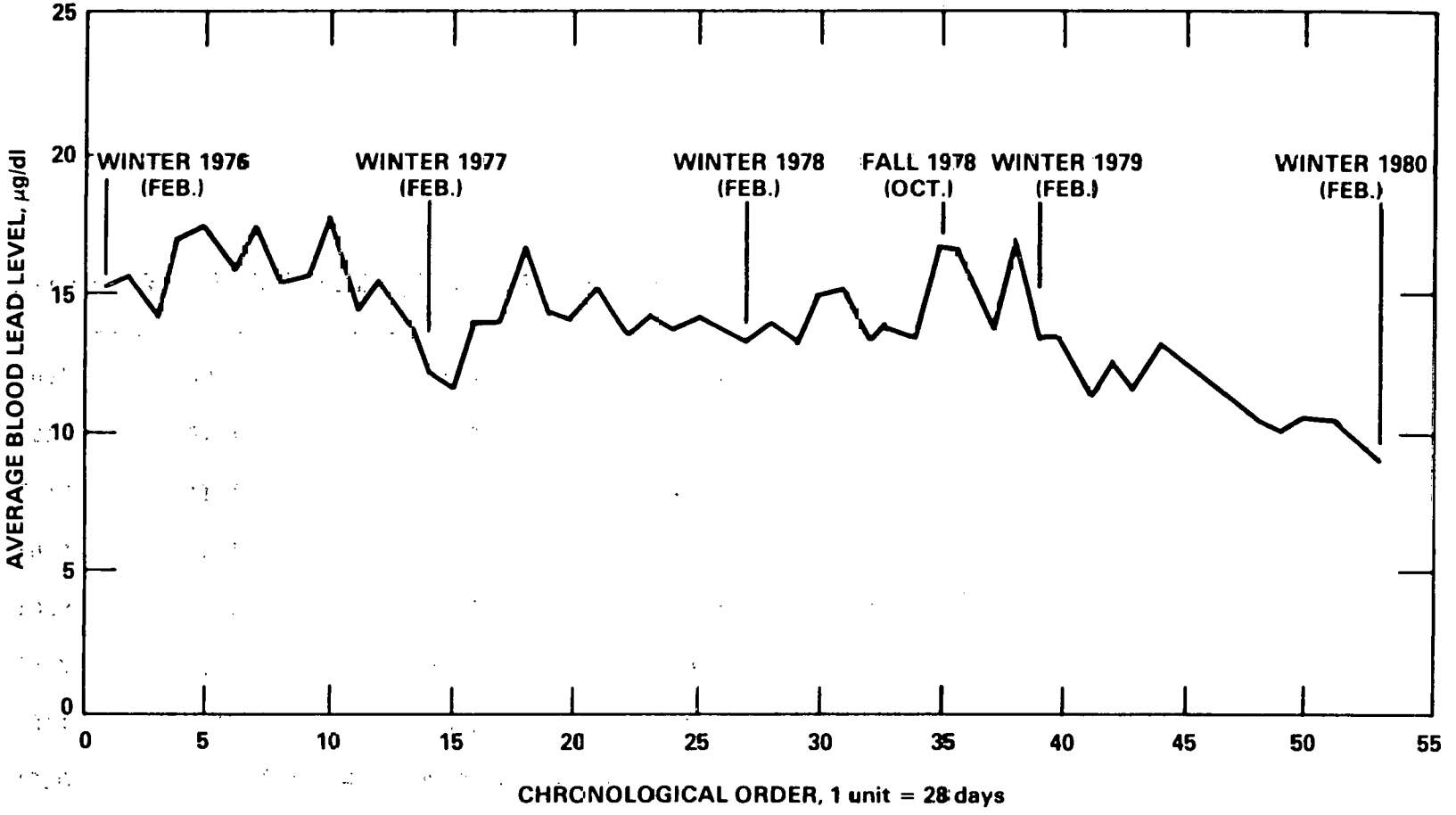

Figure 5-1. Average Blood Lead Levels of U.S. Population 6 Months - 74 Years, United States, February 1976 - February 1980, Based on Dates of Examination of NHANES II Examinees with Blood Lead Determinations

Source: Annest et al. (1983), from Air Quality Criteria for Lead, Vol. III. p. 11-26.

is inferred from the conclusions. The continued reduction of leaded gasoline sales is likely to increase the ability of most areas of the country to meet a more stringent future lead standard. The exceptions would be "hot spots" in the vicinity of certain stationary sources.

Since dietary contributions to total lead burden are high (particularly for adults), the role of lead in food processing, in bottle caps and soldered cans, for example, will become relatively more important as the lead content of gasoline continues to decrease.

Specific energy issues which merit possible concern are:

1. What are the energy implications of new and retrofit controls for smelters and other stationary sources of lead emissions?

2. What are the energy implications of regulating dietary lead intake? (This is largely within the purview of the FDA). For example, is the shift to non-solder cans much more energy intensive?

6.0 REGULATORY IMPLICATIONS

In the previous four sections, the draft Air Quality Criteria for Lead has been reviewed with respect to: (1) environmental and health effects of exposure, (2) sources of lead emissions, (3) ambient air lead concentrations, and (4) dose-effect relationships. In this section, observations are offered about the regulatory implications of the criteria document. These implications are based upon the previous sections and, unlike the previous sections, on a comparison with the 1977 criteria document.*

6.1 Overall Comments

The present revision of the criteria document is much stronger than the original published in 1977. It is better written; identifies more health effects more clearly; identifies more serious effects, such as neurological impairment and damage; identified effects at far lower levels of exposure; makes a more direct correlation between ambient air lead concentrations and blood lead concentrations; presents better arguments regarding use of animal toxicology data; and states dose-response and absence of threshold conclusions more clearly.

The multimedia characteristics of lead are handled better than in 1977. A distinction has been made between pathways and ultimate sources. The use of isotope ratio data not available earlier has allowed distinction between body lead accumulated from air sources and from other sources.

Data on the accumulative properties of lead both in the environment and biologically are more detailed than in the previous criteria document. The effect on metabolism regulation in cells is well documented, and the identification of small children as a population-at-risk is better substantiated.

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*The observations presented in this section are Radian's and were made by an individual involved in the drafting of the current ambient standard.

6.2 Standard Revisions

The current primary and secondary air quality standards for lead are 1.5 $\mu g/m^3$ maximum arithmetic mean averaged over a calendar quarter. The newly revised criteria document has all the information available to justify a new standard of 0.5 $\mu g/m^3$. The document directly describes the criteria for adverse health effects and demonstrates that lead meets those criteria in ambient concentrations at and below the present standard.

In addition, information in the document that shows peak levels for a few weeks could be damaging to health can be used to justify a shorter averaging time--a monthly or even a daily standard. Reducing the averaging time will also increase the stringency of the standard.

As noted in Section 5.0, substantial impetus is provided in the criteria document for revising the blood lead criteria downward from the present 30 $\mu g/d\ell$ as a "maximum safe" level. With the better documented linearity between air lead and blood lead, such a reduction would directly yield a mandate to reduce the ambient air standard.

Although this scientific basis appears to be provided in the criteria document, changing the ambient air standard requires a regulatory process which takes considerable time, involves several decision makers, and includes participation by several interested parties who may introduce contradictory data.

The document examined in this study is a Review Draft Air Quality Criteria for Lead. It has not been formally released by EPA but "is being circulated for comment on its technical accuracy and policy implications." At the time the original criteria document was released (December, 1977), an air standard for lead was proposed. Assuming a similar approach will be used again, a new standard could be proposed in late spring or summer, 1984, after comments on the review draft have been received and incorporated.

The timing on the standard proposal could be affected by political considerations. Proposing a lower standard would be applauded by environmentalists and opposed by gasoline- and lead-related industries. Because the President has little support from environmentalists, there would be political benefit to proposing a more stringent standard before the election.

After the standard is proposed, public comments are received. A final standard was promulgated in October, 1978, ten months after it had been proposed last time. Assuming a similar timetable, promulgation could be expected in the summer of 1985. Legal challenges to the standard would occur after promulgation, and could be expected from environmental interest groups if stringency is not increased substantially.

Following promulgation of the standard, new control strategies and emission reductions would be developed and incorporated into State Implementation Plans. This process has not yet been completed for promulgation of the original standard five years ago, and it is not likely that the process would proceed more rapidly in response to a new standard.

Since the trend in ambient concentrations is downward (as discussed in Section 4.0), and over 99 percent of stations were in compliance with the present standard, compliance with a more stringent standard may not be difficult provided use of leaded gasoline continues to be reduced.

6.3 Implications for Energy-Producing and Energy-Intensive Industries

The strategy to be used for complying with a new ambient lead standard is likely to be similar to that used for the present standard: Controlling lead in gasoline and total suspended particulates (TSP) controls on lead smelters, tetraethyl lead plants and other major sources. These controls are likely to become more stringent if a more stringent standard is adopted.

The major impact on energy-consuming industries will be on the petroleum refining sector because of additional shifting from leaded to

unleaded gasoline. According to the American Petroleum Institute, five to ten percent more energy is required to produce unleaded than leaded gasoline at the same octane rating. For regular unleaded (98 octane), seven to eight percent more energy is needed. For super unleaded (91 octane), closer to ten percent more energy is required.

One concern with regard to the shift from leaded to unleaded gasoline is the extent of misfueling which is occurring at the present time, estimated by EPA to be 25% of fuel use. If market forces alone dictate the extent of leaded fuel use, a residual leaded fuel demand could remain as long as leaded gasoline is available at lower prices than unleaded. Regulatory action may be stimulated by this misfueling in order to meet a more stringent air standard.

Impacts upon smelters will result in increased energy consumption to operate TSP control systems. Increased blower size and air handling will result in higher energy consumption. Smelters are large energy consumers for metal refining. Air handling energy consumption is a small percentage of the total for the smelter.

In summary, the new criteria document is clearer and stronger in its documentation of health impacts of lead and of ambient air concentrations as a source of those impacts. Because of this, the document will justify a more stringent ambient standard than currently exists. The standard could be sufficiently stringent to justify a complete ban on sale of leaded gasoline as well as tighten TSP controls on stationary sources.