HEALTH HAZARDS OF LEAD

1. Introduction

This paper contains a summary and analysis of all relevant medical and scientific evidence available to the Environmental Protection Agency (EPA) as of January 1, 1972, on the health hazards of airborne lead. As background, the paper also includes information on sources of airborne lead and lead levels in the environment. The information presented in this paper is drawn from several sources including a comprehensive report(1)prepared for EPA by the National Research Council-National Academy of Sciences; throughout this paper, the report will be referred to as the NAS Report. It should be noted that the conclusions presented in the final section of this paper are EPA's conclusions.

2. Sources

Naturally occurring lead in the atmosphere results from airborne dust, which contains on the average 10-15 ug of lead per gram of dust,(2)and from gases diffusing from the earth's crust.(3) These contribute about 0.0005 ug/m3 of lead to the atmosphere.(4)

The 1968 consumption of newly extracted and recycled lead in the United States was about 1.3 million tons.(5) The largest consumer was the electric-storage-battery industry (39%), followed by the petroleum industry, which used about 20% of the total for gasoline additives.(5) The amount of lead consumed in various products in 1968 is shown in Table 1. Table 2 shows estimated lead emissions in 1968 from most of the significant known sources. Annual consumption of lead for use in gasoline additives has increased by 65% over the past 10 years and has more than quadrupled since 1943. (6) As indicated by the data in Tables 1 and 2, only about 20% of the lead consumed in 1968 went into petroleum products, but almost 99% of the estimated lead emissions to the atmosphere were from gasoline combustion, gasoline transfer, and gasoline antiknock additive manufacturing, with over 98% of the emissions coming from gasoline combustion alone.

3. Ambient air levels

The concentration of lead in ambient air is closely correlated with the density of automotive traffic. Thus, concentrations are highest in large cities at sites of heaviest traffic and generally are progressively lower in suburbs, smaller towns, and rural areas. An extensive study of ambient air levels in Los Angeles, Cincinnati, and Philadelphia has been carried out.(7) Sampling sites were located in representative rural, residential, commercial, and industrial areas, and sampling was performed continuously from June 1961 through May 1962. The results are summarized in Tables 3 and 4.

In the three-city survey,(7) a special, less extensive study, designed to measure atmospheric lead concentrations found in heavy traffic, was conducted. This study showed a clear diurnal pattern related to commuter rush hours. The pertinent data are presented in Table 5.

The National Air Surveillance Networks (NASN) of the Environmental Protection Agency continually sample the atmosphere for lead and other

pollutants in many parts of the United States. NASN data also tend to show that lead levels are higher in cities than in rural and remote non-urban areas. In most cities of the NASN, the annual average concentration of lead in the atmosphere in 1957-66 ranged from 1 to 3 ug/m3, non-urban stations averaged 0.1 - 0.5 ug/m3 and the concentration at remote stations often less than 0.05 ug/m3. (8-12)

There is evidence (28) that short-term averages are more meaningful than annual averages as indices of significant human exposure to lead. NASN data for 1967 and 1969, as shown in Table 6, indicate that there were a minimum of 27 areas where one or more quarterly composite lead concentrations exceeded 2.0 micrograms per cubic meter. In the areas listed in Table 6, NASN sites were at locations where motor vehicle emissions clearly were primarily or largely responsible for the measured airborne lead levels. The concentration of lead in ambient air is heaviest near highways and is roughly proportional to the volume of traffic on these highways. A study by Daines, Motto and Chilko, (17)involving ambient air sampling along a 28-mile section of U.S. 1 in New Jersey in 1967-69, showed that ambient air levels approaching 10 ug/m3 occur within 10 feet of a highway with a volume of 58,000 vehicles per day but that the concentration falls off rapidly as traffic volume decreases or distance from roadway increases.

The NAS report states that available data suggest that the concentration of lead in air, even over most of the larger cities, is increasing only slowly.(13) The increase in the consumption of lead antiknock additives for gasoline from 169,000 tons in 1962 to 262,000 tons in 1968(13a) indicates that more lead is being emitted into the atmosphere and suggests that the quantity of lead in the air can be expected to be higher. Recent data show that in at least three U.S. cities, lead levels increased significantly from 1962 to 1969. Atmospheric lead concentrations were measured in 1969 at 19 of the sites where such measurements were made in 1962 in the original survey of lead in the air in Cincinnati, Los Angeles, and Philadelphia.(19) An increase of varying magnitude over the 7-year period was noted at 17 of the sampling sites. The increases were statistically significant at 14 sites, and these increases ranged from 33 to 64 percent in Los Angeles, from 25 to 36 percent in Philadelphia and from 26 to 33 percent in Cincinnati.

4. Levels in water and food

It seems likely that, today, the global mean content of naturally occurring lead in lakes and rivers lies between 1 and 10 ug/liter. (13b) The average lead content of 22 water samples of major rivers of North America was 6.6ug/liter; and 440 lake and river water samples in Maine was 2.3 ug/liter (range, 0.03-115 ug/liter).(13b) The natural concentration of lead in soils usually ranges from 2 to 200 ppm, exclusive of areas near deposits of lead ore.(14) In addition to fallout from mining, industrial operations, and motor vehicles, which cause localized increases in soil concentrations of lead, soils receive an average of 1 ug/cm2/year from precipitation and 0.2 ug/cm2/year from dustfall.(14)

The concentration of lead in atmospheric dustfall and in surface soil of large cities, presumably brought about by emissions from automobiles, can be very high. In Cincinnati at a distance of 25 feet from the roadway, the average lead concentration was 3397 ug/g, and at least 100 feet, the average was 2825 ug/g.(20) The concentration of lead in surface soils in a number of West Coast city parks ranged from 194 ug/g to 3,357 ug/g.(15)

The diet is another source of lead. Schroeder and co-workers(18) have made extensive determinations of lead in food. On a fresh-weight basis, they found about 1.2 ug/g (range, 0-1.5 ug/g) in condiments, 0.5 ug/g (range, 0.2-2.5 ug/g) in fish and seafood, 0.2 ug/g (range, 0-0.37 ug/g) in meat and eggs, 0.4 ug/g (range 0-1.39 ug/g) in grains, 0.2 ug/g (range, 0-1.3 ug/g) in vegetables, and no lead detectable by their analysis in fresh whole milk. Continuous monitoring of the water supplies of the United States since 1962 has demonstrated that their lead content has, in general, not exceeded the U.S. Public Health Service's prescribed standard of 50 ug/liter.(21) Using these data, and other information, a number of workers (16) have estimated that the dietary lead intake of an adult averages about 300 ug/day.

5. Environmental cycling

Lead is removed from the air by gravitational settling of larger particles, by clumping of smaller particles (aggregation) with subsequent settling, and by association with rain or other forms of precipitation as condensation nuclei or by wash out. Lead emitted in automobile exhaust causes a pronounced lead concentration profile in surfaces near heavily traveled highways. (22)

The lead content of precipitation is strongly associated with the use of lead additives in gasoline. Atmospheric precipitation samples from 32 U.S. stations showed a positive correlation between the number of gallons of gasoline used and the concentration of lead in the precipitation of each area. (23)

Plants do not take up substantial amounts of lead from the soil or atmosphere or from deposits on foliage. The concentration in soil has very little effect on the lead content of the tissues, (24) except under certain soil conditions including high acidity and high organic matter content.

6. Human Health Implications

A. Metabolism

The major source of lead intake for non-occupationally exposed adults is the diet. The average lead content of food is 0.2 parts per million,(25,26)and the daily average adult oral intake of lead from food is about 300 micrograms, with a range of 100 to 500 micrograms. (27,28) Limited data indicate that this intake has not changed significantly in the past thirty years. (29) The daily intake of lead from water has been estimated to be about 20 micrograms. (30)

The dietary intake of lead by children between the ages of one and three has been estimated to be 1300 micrograms/day. (31) This estimate was based on measurement of the amount of lead excreted in the feces by children who had known no unusual exposure to lead.

Only a fraction of the lead balance ingested in the diet is absorbed from the gastrointestinal tract. Lead balance studies in adults have shown that the average absorption of lead from the gastrointestinal tract over long periods of time is 5-10 percent of the ingested dose when the lead intake is not excessive. (28) Therefore, it can be calculated that about 15 to 30 micrograms of lead are absorbed from the diet each day by an adult. The exact proportion of dietary lead intake that is absorbed by infants and children has not been determined.

The contribution of lead in the air to the total amount absorbed each day has not been precisely defined. For adults, estimates of the contribution have been made by assuming that: 1. Similar percentages of lead particulate matter will be deposited in the respiratory tree, regardless of the concentration of lead in the inhaled air (Experiments with air containing 150 ug/m3 of lead particulate matter with a mass median diameter of 0.25 um showed that 36 percent of the lead was deposited in the airways.(28) It was then assumed that a similar percentage of lead would be deposited when air containing concentrations of lead in the range of 1-10 ug/m3 was inhaled), and 2. all of the lead deposited in the respiratory tract is absorbed into the body. From these assumptions, it can be predicted that the total daily absorption of lead from the air by a "standard man" engaged in light activity would be from 0.8 ug to 63 ug, depending on where he lives and works. (32) The percentage of inhaled lead that is deposited in the respiratory tract of infants and children has not been determined experimentally; however, if it is assumed that the deposition is similar to adults (i.e. about 36%), and that all the deposited lead is absorbed into the body, one can calculate the amount of lead is absorbed from the air.

During periods of normal intake of lead, about 90 percent of the lead appears in the feces. (28) A small amount of this lead may initially be absorbed and then excreted back into the gastrointestinal tract. Most of the remaining dietary lead that is not retained in the body or found in the feces can be found in the urine.

If certain simplifying assumptions are made, one can construct a dose-response curve relating the concentration of lead in the blood to the daily absorption of lead from all sources. The epidemiologic data assembled by Goldsmith and Hexter (33) are used for lead concentrations in the blood of various groups of men whose estimated exposure to atmospheric lead differed. The contribution of lead in the air to the daily absorption is estimated by using the assumptions mentioned above (i.e., that similar percentages of inhaled lead particulate matter are deposited in the respiratory tract, regardless of the concentration of lead in the inhaled air, and that all the lead that is deposited in the airways is absorbed). Respiratory deposition of inhaled lead ranges from 25 to 50%. The amount of lead that will be absorbed from inhaled air is equal to the airborne lead concentration X the volume of air inhaled per day (about 23 cubic meters) X the percent of lead deposited in the respiratory tract. For example, if a person inhales air containing 2 micrograms of lead per cubic meter, and a conservative 30% respiratory deposition is assumed, then the lead absorption from the air can be calculated as follows:

2 ug/m3 X 23m3 X 30%= 13.8 micrograms

An individual's total daily lead absorption is calculated by adding daily dietary lead absorption (about 30 micrograms) to the amount absorbed from air.

For convenience, these calculations have been summarized over a wide range of airborne lead levels (Table 7). Elevated blood leads and thus excess body burdens are associated with airborne lead levels greater than 2.0 micrograms per cubic meter. Increased urinary excretion of delta-aminolevulinic acid begins at blood lead levels of about 40 micrograms per 100 grams of whole blood. (34) Subtle signs of clinical lead poisoning have been associated with blood lead levels of about 50 to 80 micrograms/per 100 grams of whole blood. (35)

At the present time, there are no epidemiologic data which relate the lead concentration in the blood of infants and children to their estimated exposure to atmospheric lead.

B. Health Effects

Lead accumlates in humans when the amount they absorb from all sources exceeds the amount that is excreted. Clinical experiments have demonstrated that small increments in the amount of lead in the diet will result in prolonged (though not indefinite) periods of accumulation. (28) Presumably, increments in the amount of lead absorbed from the lungs will result in similar periods of accumulation of lead in the body. Certain occupational groups (garage workers, auto mechanics, and vehicular tunnel policemen) who are incidentally exposed to high atmospheric lead concentrations have been found to have mean blood levels of lead that are higher than the mean of the general population. (36,37) Furthermore, several studies have shown that urban residents have a higher average level of lead in their blood than their rural counterparts. (36,38) Although this difference might be due to differences in dietary lead intake between the groups,(39) it might also be due to differences in the intake of lead from the air. In either event, it is clear that most people are accumulating lead in their bodies. The NAS Report concluded: "The exposure of people in the general population to lead results in some accumulation in the body up to and perhaps beyond the age of 40, as determined by analysis of tissues." It goes on to say: "The biologic significance and reason for the increase are not known." (40)

Overt symptons of clinical lead poisoning in adult males have not been reported at levels of lead in the whole blood lower than 80 micrograms/100 grams. However, inhibition of the activity of certain enzymes involved in the synthesis of heme can be shown to occur when the levels of lead in the whole blood are less than 80 micrograms/100 grams. For example, the inhibition of aminolevulinic acid dehydrase (ALAD), (41) the enzyme which catalyzes the conversion of delta-aminolevulinic acid (ALA) to porphobilinogen (PBG), occurs well below 80 micrograms/100 grams of blood. There is an inverse linear relationship between the logarithm of the activity of ALAD in vitro and the concentration of lead in the blood when the concentration varies from 5 to 95 microgram/100 grams. (42,43,44) Although ALAD is essential for hemoglobin synthesis at lead concentrations below 40 micrograms/100 grams, there are currently no detectable biological effects of this enzyme inhibition.(45) This does not prove that no biological effects occur.

However, when the levels of lead in the blood exceed 40 micrograms/100 grams, the amount of ALA excreted in the urine increases exponentially.(46,47) The NAS Report hypothesized that: "The exponential increase in ALA excretion associated with blood lead content above approximately 40 micrograms/100 grams

of whole blood signifies inhibition of ALAD that is physiologically significant in vivo." (48) With respect to hematopoiesis, this inhibition of the conversion of ALA to PBG may be compensated completely when the concentration of lead in whole blood ranges between 40-80 micrograms/100 grams. However, when the level of lead in the blood exceeds about 80 micrograms/100 grams, the compensatory mechanism becomes inadequate and anemia ensues. The NAS Report states the following concerning the inhibition of ALAD (and the resulting excretion of ALA in the urine) at blood lead concentrations of 40-80 micrograms/100 grams:" ...this action must be viewed as undesirable, in that it does represent an inference with the availability of an essential metabolite required for normal body function, which in some circumstances might prove deleterious." (49) The risks from ALAD enzyme inhibition are increased among inner city children. These children are deficient in iron intake, which further decreases red blood cell synthesis, and are exposed to higher levels of carbon monoxide, which reduces the oxygen carrying capacity of their blood.

Three groups include most of the cases of clinically recognizable lead poisoning in the United States. These groups are young children living in urban areas; people who consume illicitly distilled, lead-contaminated alcohol; and workers in the lead trades. Additional sporadic cases of lead poisoning have been caused by the burning of battery casings in the home and the use of earthenware which has been improperly finished with a lead-containing glaze.

Young children living in deteriorating, dilapidated, urban housing are at risk to be poisoned by lead because of their indiscriminate eating habits and the availability of lead in their environment. The usual source of lead in cases of poisoning among such children is lead-pigment paint flaking from the interior surfaces of the dwellings in which the children live. (50) These paint flakes contain as much as 40 percent lead by weight.

Mass-screening programs conducted in New York and Chicago(51,52)have shown that between 1 and 2 percent of the children examined have blood levels of lead that are compatible with lead poisoning. In addition, 25 percent of the children have levels of lead in their blood that exceed the upper limit of normal (40 microgram/100 grams). (52) Although the direct ingestion lead-pigment paints is the principal environmental source in cases of severe lead poisoning in young children, it may not explain the rather large percentage of children with levels of lead in the blood exceeding 40 microgram/100 grams. Other potential sources of lead intake in the urban environment are lead in the atmosphere and lead that "falls and rains out" of the air and is contained in the dust on the ground. Dustfall samples fall from 77 midwestern cities contianed the following average amounts of lead: In residential areas - 1,636 micrograms/grams of dust; in commercial areas - 2,413 micrograms/grams of dust; and in industrial areas - 1,512 micrograms/grams of dust.(53) It has been calculated that daily ingestion by a one-year-old child of as little as 1/24 of a teaspoon of dust from within 100 feet of a busy roadway would, within eight months, result in lead poisoning.(54) The NAS report stated: "The swallowing of lead-contaminated dust may well account, in large part, for the higher mean blood lead content in urban children and the rather large fraction whose blood lead content falls in the range of 40-60 micrograms/100 grams of whole blood, thereby bringing them into the range in which increased urinary excretion of ALA may be observed."(55) Although the extent to which airborne lead in urban air contributes to lead absorption and

lead poisoning in children is not clearly defined, the NAS report states: "Airborne lead wastes from such sources as automotive emissions and the weathering and demolition of old buildings can be expected to have a significant additive effect on the total intake."(56) It should be remembered that combustion of gasoline containing lead additives is responsible for 98 percent(57)of the emissions of lead to the atomosphere.

The clinical manifestations of lead poisoning are variable and reflect involvement of several different organ systems. They include anemia, acute abdominal colic, acute encephalopathy, chronic encephalopathy, peripheral neuropathy, and chronic nephropathy. The syndromes of acute lead poisoning usually resolve following cessation of the exposure to lead and institution of therapy. However, acute encephalopathy in young children is followed by permanent neurological sequelae in at least 25 percent of the cases.(58,59)

In virtually all of the cases of human lead poisoning recorded to date, it has not been possible to quantitate the amount of lead absorbed prior to the onset of symptoms. Balance studies in human volunteers have shown that the blood lead level is a fairly sensitive index to recent absorption of lead. Overt symptons of clinical lead poisoning have not been reported at blood lead levels below 80 micrograms/100 grams (in occupationally exposed adult men who are not anemic and are otherwise healthy). However, symptoms and signs compatible with mild lead poisoning have been associated with whole blood concentrations of 50-80 micrograms/100 grams.(60) In 41 cases of acute lead poisoning in children, the average blood lead value was 176 micrograms/100 grams, with the lowest value being 63 micrograms/100 grams. In 99 cases of encephalopathy, the average blood lead level was 330 micrograms/100 grams.(61)

Stop

7. Summary and conclusions

A. Lead has not been shown to have any biologically useful function; all available data indicate that the metabolic effects of low concentrations of lead are of the inhibitory or adverse type. Accordingly, any increase in body burden of lead is accompanied by an increased risk of human health impairment.

B. Though the amounts of lead ingested in the diet generally are greater than the amounts inhaled, the body absorbs a greater percentage of inhaled lead (30 percent or more) than of ingested lead (about 10 percent).

C. Human blood lead levels are the most frequently used index of human exposure to environmental lead; it should be noted, however, that other tissues, e.g., hair, may provide a more accurate index of total body burden, particularly where exposure levels are relatively low.

D. Human blood lead levels begin to rise appreciable with exposure to airborne lead concentrations in excess of 2 micrograms per cubic meter. Table 1 reflects the best available data on the relationship of lead intake to absorption and blood lead levels.

E. Elevated lead intake for periods as short as, and possibly shorter than, three months is sufficient to produce an increase in blood lead levels.

F. Average blood lead levels tend to be higher among urban residents than among rural residents; they also tend to be higher among certain occupational groups, i.e., garage workers, auto mechanics, vehicular tunnel policemen, than among the general population.

G. Body burdens of lead, as measured by bone lead concentrations, are known to increase with age at least until age 40 and probably thereafter.

H. The activity of certain enzymes involved in heme synthesis (one step in red blood cell formation) is inhibited at blood lead levels significantly below those associated with clinical lead poisoning; specifically, when blood lead levels exceed 40 micrograms per 100 grams, the amount of ALA excreted in the urine begins to increase exponentially. According to the NAS report: "The exponential increase in ALA excretion associated with blood lead content above approximately 40 micro-= grams/100 grams of whole blood signifies inhibition of ALAD that is physiologically significant in vivo".(62) The NAS report also contained the observation that "...this action must be viewed as undesirable, in that it does represent an interference with the availability of an essential metabolite required for normal body function, which in some circumstances might prove deleterious."

I. Exposure to airborne lead often tends to be greatest in areas whose inhabitants, particularly children, are especially likely to be exposed to other environmental sources of lead.

J. Though ingestion of leaded paint clearly is the principal cause of lead poisoning among children, it does not necessarily account for the rather large percentage (25 percent) of children who, in mass screening programs in inner city areas, did not appear to have clinical lead poisoning but had blood lead levels exceeding 40 micrograms per 100 grams. Among such children, the high concentrations of lead found in urban street dust are another potential source of lead intake. According to the NAS report: "The swallowing of lead-contaminated dusts may well account, in large part, for the higher mean blood lead content in urban children and the rather large fraction whose blood lead content falls in the range of 40-60 micrograms per 100 grams of whole blood, thereby bringing them into the range in which increased urinary excretion of ALA may be observed."

K. The elevated concentrations of lead in dust, soil, and vegetation near streets and highways clearly can be attributed to lead emissions from motor vehicles.

L. Precipitation samples from 32 locations in the U.S. showed a positive correlation between gasoline consumption and lead content of precipitation in the areas where the sampling sites were located.

Based on the available evidence, the Administrator has concluded that airborne lead levels exceeding 2 micrograms per cubic meter, averaged over a period of three months or longer, are associated with a sufficient risk of adverse physiologic effects to constitute endangerment of public health. Since airborne lead levels in many major urban areas currently range from 2 to somewhat over 5 micrograms, and since motor vehicles are the predominant source of airborne lead in such areas, attainment of a 2.0 microgram level will require a 60 to 65 percent reduction in lead emissions from motor vehicles.

TABLE 1

Lead consumption in the U. S., 1968[a]

Product category	Tons consumed
Metal products[b]	915,500
Pigments	109,734
Gasoline additives	261,897
Other	41,659
TOTAL	1,328,790

a/ Adapted from Minerals Yearbook, 1969, p. 47
b/ Includes 513,703 tons consumed for storage batteries

TABLE 2

Lead emission in the U. S., 1968[a]

Emission source	Lead emitted, tons/year
Gasoline combustion	181,000
Coal combustion	920
Fuel oil combustion	24
Lead alkyl manufacturing	810
Primary lead smelting	174
Secondary lead smelting	811
Brass manufacturing	521
Lead oxide manufacturing	20
Gasoline transfer	36
TOTAL	184,316

a/ Data from National Inventory of Air Pollutant Emissions and Controls, on file at the Environmental Protection Agency, Stationary Source Pollution Control Program, Durham, N. C.

TABLE 3

Annual Mean Concentrations of Atmospheric Lead
June 1961 through May 1962[a]

Lead concentration, ug/m^3

Site	Cincinnati	Los Angeles	Philadelphia
Commercial	1.7	2.9	2.2
Industrial	1.8	2.3	2.2
Residential	1.1	2.0	1.1
Rural	0.9	2.8	0.9
All Stations	1.4	2.5	1.6

a/ Adapted from "Survey of Lead in the Atmosphere of Three Urban Communities," reference 7, p. 32.

TABLE 4

Seasonal Mean Concentrations of Atmospheric Lead
June 1961 through May 1962[a]

Lead concentration, ug/m^3

Season	Cincinnati	Los Angeles	Philadelphia
Summer	1.3	1.9	1.4
Fall	1.7	2.8	1.9
Winter	1.3	3.1	1.9
Spring	1.3	2.1	1.4

a/ Taken from "Survey of Lead in the Atmosphere of Three Urban Communities," reference 1, p. 32.

TABLE 5

Atmospheric Concentrations of Lead in Traffic in Cincinnati and Los Angeles[a]

Concentration, ug/m^3

City	Morning Rush Mean	Morning Rush Range	Morning Rush No. of Samples	Midday Mean	Midday Range	Midday No. of Samples	Afternoon Rush Mean	Afternoon Rush Range	Afternoon Rush No. of Samples
Cincinnati -									
Mobile Routes	14.2	7.2-19.5	20	9.1	7.0-12.3	8	15.2	9.3-21.1	8
Los Angeles									
Freeway	38.0	26.9-54.3	7	24.1	16.6-31.1	7	18.4	8.7-25.4	7
Downtown	23.6	19.1-29.9	6	10.5	8.4-12.2	4	15.3	12.4-18.6	4

a/ Taken from "Survey of Lead in the Atmosphere of Three Urban Communities", ref. 1, pp. 55-56.

TABLE 6

Maximum Quarterly Composite Lead Levels Exceeding 2.0 Micrograms Per Cubic Meter[a]

LOCATION	MAXIMUM QUARTERLY COMPOSITE
Oklahoma City, Oklahoma	2.1
Baltimore, Maryland	2.1
Miami, Florida	2.1
Kansas City, Kansas	2.2
Fort Worth, Texas	2.3
Cleveland, Ohio	2.3
Springfield, Massachusetts	2.4
Shreveport, Louisiana	2.4
Las Vegas, Nevada	2.4
Richmond, Virginia	2.6
New York, New York	2.8
Houston-Galveston, Texas	2.8
Seattle, Washington	2.9
N. W. Nevada	3.0
Detroit, Michigan	3.2
Denver, Colorado	3.4
Salt Lake City, Utah	3.6
Chicago, Illinois	3.6
El Paso, Texas	3.6
Philadelphia, Pennsylvania	3.6
Phoenix, Arizona	3.9
San Francisco, California	3.9
Puerto Rico	4.2
San Diego, California	4.2
Fairbanks, Alaska	4.8
Dallas, Texas	5.2
Los Angeles, California	5.7

a/ Source: NASN data on file at the Division of Atmospheric Surveillance, National Environmental Research Center, Environmental Protection Agency, Research Triangle Park, North Carolina.

TABLE 7

Relationship of Adult Blood Lead Levels and Body Lead Burdens to Airborne Lead Exposure

Air Lead Exposure	Daily Lead Absorption ug/day			Expected Blood Lead[3]	Relative Excess in Blood Lead (%)[4]
ug/m^3	Air[1]	Diet[2]	Total	u/gl00g	
2.0	13.8	30	43.8	21.3	0%
2.5	17.3	30	47.3	22.8	7%
3.0	20.7	30	50.7	24.3	15%
3.5	24.2	30	54.2	25.8	23%
4.0	27.6	30	57.6	27.3	30%
4.5	31.1	30	61.1	28.8	38%
5.0	34.5	30	64.5	30.3	47%
10.0	69.0	30	99.0	40.0	137%
20.0	138.0	30	168.0	53.8	284%
50.0	345.0	30	375.0	71.6	780%
100.0	690.0	30	720.0	87.2	1550%

1/ Assumes inhalation of 23 m^3/day and 30% lung retention.

2/ Assumes 10% gastrointestinal absorption of the average adult daily total dietary intake (300 ug) from food and water.

3/ Computed from regression formula: Blood lead = 69.2052 + 54.7605 x log ug Pb absorbed daily as given in Chapter 3 of Airborne Lead in Perspective by the National Research Council, National Academy of Sciences.

4/ Relative excess in blood lead is associated with ambient air exposure above 2.0 ug Pb/m^3.

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Corrections and Additions

To

Health Hazards of Lead

(Revised April 11, 1972)

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The original Table 7 on page 14 should be replaced by the following revised and corrected Table 7. The figures presented in the original Table 7 for expected blood lead concentrations and relative excess in blood lead percentages were miscalculated and have been corrected. Footnote 4 has also been revised.

TABLE 7

Relationship of Adult Blood Lead Levels to Airborne Lead Exposure

Air Lead Exposure	Daily Lead Absorption ug/day			Expected Blood Lead[3]	
ug/m^3	Air[1]	Diet[2]	Total	ug/100g	Relative Excess in Blood Lead (%)[4]
2.0	13.8	30	43.8	20.7	0%
2.5	17.3	30	47.3	22.5	9%
3.0	20.7	30	50.7	24.2	17%
3.5	24.2	30	54.2	25.8	25%
4.0	27.6	30	57.6	27.2	31%
4.5	31.1	30	61.1	28.6	38%
5.0	34.5	30	64.5	29.9	44%
10.0	69.0	30	99.0	40.1	94%
20.0	138.0	30	168.0	52.7	155%
50.0	345.0	30	375.0	71.7	246%
100.0	690.0	30	720.0	87.3	322%

1/ Assumes inhalation of 23 m^3/day and 30% lung retention.

2/ Assumes 10% gastrointestinal absorption of the average adult daily total dietary intake (300 ug) from food and water.

3/ Computed from regression formula: Blood lead = -69.2052 + 54.7605 x log ug Pb absorbed daily as given in Chapter 3 of Airborne Lead in Perspective by the National Research Council, National Academy of Sciences.

4/ According to the National Academy of Sciences, ". . . it is not possible, on the basis of available epidemiological evidence, to attribute any increase in blood lead concentration to exposure to ambient air below a mean lead concentration of about 2 or 3 ug/m^3 . . ." 64/ It follows that a relative excess in blood lead may be associated with ambient air exposure above 2.0 ug Pb/m^3.

ERRATA

1. Page 5, para. 1, line 5: Change note "35/" to read "34/."

2. Page 5, para. 1, line 5: Following the sentence ending: "...per 100 grams of whole blood 34/." insert" "Subtle signs of clinical lead poisoning have been associated with blood lead levels of about 50 to 80 micrograms/per 100 grams of whole blood.(35)"

3. Page 5, para. 4 line 9: Following the sentence ending "... accumulating lead in their bodies." insert "40/"

4. Page 8, para. E, lines 1 and 7: Change "Kg" to "kg."

5. Page 17, reference 61: After "Amer. J. Dis. Child." omit "122" and insert: "Vol. 122, October, 1971, pp. 337-340."

REFERENCES

In the "Health Hazards of Lead" paper, the references to the National Academy of Sciences (NAS) report, "Airborne Lead in Perspective," included page numbers which referred to the pre-publication copy. Recently the NAS report has been published in final form. Accordingly, the references to the NAS report have been revised and listed below to refer to the appropriate page of the published version of "Airborne Lead in Perspective."

1. National Research Council-National Academy of Sciences. "Lead, Airborne Lead in Perspective," A Report Prepared by the Committee on Biological Effects of Atmospheric Pollutants of the Division of Medical Sciences, 1971 (NAS Report). All NAS page references refer to the published copy.

13. NAS Report, p. 22.
13a ibid p. 24.
13a ibid p. 27.
14. ibid p. 28.
15. ibid p. 30.
16. ibid p. 50.
24. NAS Report, p. 46.
29. NAS Report, p. 69.
30. ibid p. 48.
32. NAS Report, p. 69. (Precise numbers deleted in final report, but general context of the quote remains the same.)

34. NAS Report p. 107.
35. ibid p. 74.
39. NAS Report p. 63.
40. ibid p. 67.
41. ibid p. 106
45. NAS Report, p. 208
48. NAS Report, p. 110.
49. ibid p. 208.
55. NAS Report, p. 139.
56. ibid p. 140.
60. NAS Report, p. 74
62. NAS Report p. 110.
63. ibid p. 132.
64. ibid p. 211.
65. ibid p. 139.
66. ibid p. 211.
67. ibid p. 208.