ENVIRONMENTAL LEAD AND PUBLIC HEALTH

U. S. ENVIRONMENTAL PROTECTION AGENCY

AP90

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ENVIRONMENTAL PROTECTION AGENCY
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Cover: The symbol on the cover was used by 18th century alchemists to indicate lead, according to Bergman's alchemy table, dated 1783.

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FOREWORD

Environmental Lead and Public Health, a summary of the major public health problems associated with lead, was written for a broader group of readers than the professional scientific community alone. For those who wish more detailed information than that provided in this report, appropriate references to the literature are provided.

For each problem, the report succinctly discusses the scope and nature of the problem; the present scientific and technological knowledge relevant to its solution; the significant gaps in this state of knowledge; the predictions on the growth of the problem; and, in some cases, alternative approaches to its solution.

Several other reports on lead are being published by the Federal Government. A technical state-of-the-art report on lead is being prepared for APCO by the National Academy of Sciences. An interim report, *Implications of Lead Removal from Automotive Fuels: Interim Report of Commerce Technical Advisory Board Panel on Automotive Fuels and Air Pollution,* published in June 1970 by the U. S. Department of Commerce, outlines a phased plan for removal of lead from gasoline. The final report is scheduled for publication in 1971. Community guidelines for eliminating the problem of lead poisoning among children in older urban areas will be detailed in *Control of Lead Poisoning in Children* to be published by the Bureau of Community and Environmental Management in 1971.

The interdisciplinary nature of the lead problem required the cooperative efforts of many services within the U. S. Department of Health, Education, and Welfare (DHEW) in the preparation of this report. The scientific staff within the Air Pollution Control Office, in particular, Drs. Ronald E. Engel, Robert A. Horton, and Douglas I. Hammer were responsible for obtaining and integrating the material presented in this document. Coordination in the Environmental Protection Agency (EPA) was handled by Dr. Lawrence A. Plumlee. Individual contributions were prepared by Public Health Service Drs. Barry King, Herbert Stokinger, Harold Wolf, Jane Lin-Fu, Granville Lipscomb, and Mr. George Morgan. Additional contributions via telephone came from Drs. Benjamin Pringle, Frances Marzulli, Dale Lindsay, and Miss Helen M. Reynolds.

The EPA Lead Liaison Committee, the DHEW Intra-Departmental Committee on Lead Poisoning in Children, and the National Clearing House for Smoking and Health provided guidance and review in the initial stages of preparation.

The assistance of all the persons in EPA, Health Services and Mental Health Administration, Food and Drug Administration, and other government and private agencies who have made this document possible despite many other pressing demands is most gratefully acknowledged. We hope that all who read this report will find it a useful guide.

John T. Middleton
Acting Commissioner
Air Pollution Control Office
U. S. Environmental Protection Agency

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ENVIRONMENTAL LEAD AND PUBLIC HEALTH

INTRODUCTION

Elements that comprise less than 0.01 percent of an organism are commonly called trace elements. Some of these elements, such as copper, iron, and zinc, are known to be essential for optimal biological function. Others, such as cadmium, lead, and mercury, are not presently known to be necessary for normal growth and metabolism. Large amounts of either essential or nonessential trace elements may be toxic, however.

Natural lead deposits are ubiquitous; man has been aware of the uses and hazards of lead since the time of the ancient Greeks.[1] In its natural state lead has never been a major source of poisoning. The common forms of lead poisoning result from the mining, processing, and commercial dissemination of the metal. Because as a metal it has several desirable properties such as relative ease of refining from natural ores, malleability, and resistance to corrosion, lead is used extensively in industry and in consumer goods. Industrialization and urbanization have caused ecologic shifts resulting in increased general population exposures to lead from man-made sources. Any health effects caused by the increased lead exposures appear to be subtle, since they do not cause any apparent clinical lead poisoning. Although lead poisoning does occur in animals,[2,3] further discussion in this report is confined to human effects.

Because lead is a known poison, it is necessary to identify and control those conditions that result in excessive human exposure. For the same reason, it is important to learn man's tolerance by determining what level of intake causes the earliest signs of intoxication so that the appropriate margins of safety can be developed. To fully understand the effects of lead, one must consider both its role in human physiology and its sources.

The purpose of this document is to briefly summarize the major public health problems associated with lead in the environment and the role that the Department of Health, Education, and Welfare and EPA fulfill with respect to these problems. Following a basic discussion of lead metabolism and toxicology, the sections on diet and consumer goods and on ambient air deal primarily with general population exposures. Because industrial workers and children constitute two distinct exposure groups, they are discussed in separate sections.

LEAD METABOLISM AND TOXICOLOGY IN MAN

INTRODUCTION

The chemical form of lead is an important determinant of its biological behavior. Except in contaminated water, lead is rarely encountered in the ionic form. Alkyl lead compounds, used primarily as automotive-fuel additives, are readily absorbed by skin and mucous membranes and are preferentially distributed to the lipid phases of the body, including the brain. Such properties make this organic form highly toxic, though in a different way from inorganic forms.[2] Since virtually all alkyl lead fuel is normally destroyed during combustion, organic lead poisoning is a special problem limited to a small group of occupationally exposed workers. Further mention of lead in this document refers to the *inorganic* forms, unless otherwise specified.

ABSORPTION

Lead is primarily absorbed through the gastrointestinal and respiratory tracts. Not all ingested or inhaled lead is absorbed or retained in the body, however. The quantities of lead absorbed into the human body may be estimated with fair accuracy from experimental evidence, which shows (1) that somewhat less than 10 percent of the lead ingested daily under ordinary circumstances is absorbed from the gastrointestinal tract and (2) that from 25 to 50 percent of the inhaled lead, dependent upon the size, shape, and density of the particles inhaled, is retained and absorbed in the respiratory system. The average quantity absorbed daily in the gastrointestinal tract of the adult is of the order of 30 micrograms, and a reasonable estimate of that absorbed in the respiratory system would be less than 20 micrograms for urban dwellers.[4, 5, 6] In the United States, lead intake in food and beverages ranges from 100 to 2000 micrograms per day for an individual, with long-term averages of 120 to 350 micrograms per day.

As lead intake is increased, the amount of lead retained also increases. Clinical lead poisoning becomes apparent when the tolerance level is exceeded. In a series of careful feeding experiments, Kehoe showed that a healthy adult man could eat 1 milligram of lead chloride per day for several years without apparent ill health even though his body pool and blood lead were increasing. In contrast, a subject eating 3 milligrams of lead chloride per day developed signs and symptoms of clinical lead poisoning within 8 weeks.[5] Lead retention is relatively poor, and prolonged exposure is required for development of clinical symptoms of lead poisoning.

INTOXICATION

Although all organs contain some lead, about 90 percent of the body pool is found in the skeleton. The total body pool for a 70-kilogram man is estimated between 100 and 400 milligrams.[7] In practice, it is often necessary to use the lead level in a readily accessible tissue, such as blood, as an index of the body pool. Sometimes urinary lead is also used because specimens are easy to obtain, but urine levels vary more than blood levels under similar conditions.

Individuals with low levels of exposure have between 10 and 30 micrograms per 100 grams of blood; highly exposed individuals frequently have blood-lead levels over 100 micrograms per 100 grams. Only persons exposed to concentrated sources of lead are likely to have such high levels.

Clinical lead poisoning, or plumbism, is commonly characterized by severe abdominal cramps, headaches, constipation, loss of appetite, fatigue, anemia, motor-nerve paralysis, and encephalopathy. Almost exclusively confined to children, lead-induced encephalopathy often leaves permanent brain damage after both exposure and acute illness have ceased. To date, no cases of plumbism have been reported in adults with blood-lead levels below 80 micrograms per 100 grams or in children with blood-lead levels below 50 micrograms per 100 grams.[8]

Clinical lead poisoning should be apparent to the physician. Such a condition should have a subclinical stage wherein some early signs of disturbance are found by appropriate tests. Unfortunately, published reports of clinical lead poisoning are still much more extensive than those on the early subclinical or subtle changes.

Lead causes damage to the blood-forming system by altering both hemoglobin synthesis and red-blood-cell-survival time. Although clinical anemia has not been detected at blood levels under 50 and 80 micrograms per 100 grams, in children and adults, respectively, recent work on hemoglobin formation and red-cell survival indicates that early signs of disturbance can be detected below this level in adults, but not in children.[1,7] Examination of plasma delta-aminolevulinic acid levels [δ-ALA] may eventually prove useful as an indicator of lead toxicity.[9,10,11] Plasma delta-aminolevulinic acid dehydrase (δ-ALADH) also seems to be a sensitive indicator of blood lead. The activity of this enzyme correlates negatively with blood-lead levels ranging from 5 to 95 micrograms of lead per 100 grams of blood.[12]

AREAS FOR RESEARCH

In contrast to the considerable amount of research directed toward detection of subclinical lead poisoning, there has been little investigation of early and late effects on organ systems other than blood, such as bone,

kidney, liver, and the nervous system.[13-16] Blood-lead levels do not accurately reflect the body lead pool and may not be the optimal measures of exposure in the general population. Knowledge of the factors that favor the release of lead from body storage sites is essential for determining the contribution of the various lead sources to the total environmental hazard. Also necessary is the estimation of the total body burden that can be tolerated considering possible shifts of body stores.

Other gaps in the knowledge of the etiology of lead poisoning fall into three main categories. The first is the lack of information regarding possible long-term, or chronic effects of lead at environmental levels of exposure, especially at levels that do not produce apparent clinical lead poisoning. Although it is known that lead accumulates with age in the skeleton, kidneys, liver, aorta, pancreas, and lungs, few follow-up studies have been made on the present health status of people exposed to excessive lead levels 20 to 50 years ago.[6] The few reported studies are contradictory.

The second gap is the paucity of information on the effects of exposure on groups of individuals in the general population who might be especially sensitive to lead. This sensitivity might be permanent or temporary. Metabolic abnormality such as glucose-6-phosphate dehydrogenase (G6PD) deficiency and sickle cell disease are examples of permanently altered sensitivity. Temporary increased sensitivity may be associated with a particular age period of growth, of pregnancy, or of illness. Particular attention should be directed toward elucidation of the mechanisms by which lead produces the central-nervous-system syndrome in children and not in adults. The possibility that lead exposure from conception could enhance the onset or severity of disease should be explored. Emphasis should be placed on determining whether the fetus of a mother exposed to elevated environmental concentrations of lead is even more sensitive to lead encephalopathy than the 2-year-old child.

The third gap in our knowledge is that little scientific information on the possible effects of interaction of lead with other environmental chemicals is known. The chemical diversity and complexity of our environment necessitates the study of such interactions.

Although blood and urine are commonly used to determine clinical illness and increased exposure, additional indices more closely related to the total body-lead pool are needed to evaluate low-level exposures. The use of hair and teeth, which are easily accessible tissues, has been recommended, and a few pilot studies have indicated their potential usefulness.[13, 17, 18] More detailed studies are needed.

Improved methods of treatment should reduce, not only the number of deaths, but also the residual effects in survivors. Studies of children who have elevated body-lead levels and who appear clinically asymptomatic would help to determine the subtle effects of lead poisoning that are not immediately apparent. Such studies could provide biomedical tools for determining

the effectiveness of treatment, and for detecting and preventing high body burdens of lead.

LEAD IN DIET AND CONSUMER GOODS

INTRODUCTION

The diet is the principal source of lead uptake among the general population. All natural foods contain small quantities of lead, the level varying according to their growing environment. There is no food source known, however, to concentrate lead selectively from the soil. This is in distinction to other trace elements, such as copper and selenium.

Since lead is widely used in equipment and containers involved in food processing, prepared foods and beverages may become contaminated. Historically, direct contamination of food and drink has been the major source of lead poisoning among general populations. Acidic foods, in particular, can leach lead from cooking, serving, and storage utensils made from metal alloys, or from pottery improperly glazed with lead compounds. It has recently been suggested that the widespread use of lead in the cooking utensils of the Roman upper classes contributed to the fall of Rome.[19]

NATURAL SOURCES

Fruits and Vegetables

Past concern with lead arsenate used as a pesticide on fruits and vegetables resulted in establishment of Federal Tolerance Limits for lead arsenate residues on selected agricultural raw products. Except for citrus fruits, whose acidity requires a limit of 1 part per million (ppm), the limit is 7 ppm where the regulation applies.[20] Since 1945 the use of lead arsenate has been declining, but over 5 million pounds was still used on fruit* and tobacco in 1968.[21] Moreover, soils in some areas still have evidence of heavy contamination with the stable lead arsenate compound.[22] Preliminary evidence from limited studies of crops growing near highways suggests that, other things being equal, plant residues and uptake decrease exponentially with the distance from the highway.[23]

Lead in milk poses no current threat to public health even in areas with pollution problems.[24]

In a recent study of dietary trace metals the total lead content found in sample American diets was approximately 0.03 microgram per gram. The

*Seventy percent of the 5 million pounds was used in apple orchards.

lead levels in individual foods varied from 0.08 to 0.3 microgram per gram in some local market products.[25] Certain foods consistently contain more lead than others.[26] The assay techniques have not yet been adapted to include lead among the pesticide residues routinely monitored in the U. S. Food and Drug Administration's Market Basket Survey of dietary components.

Fish

Bacterial decomposition of organic matter is inhibited by lead concentrations in water equal to or greater than 0.1 milligram per liter.[27] As with many other elements, lead is more toxic to fish in soft water than in hard water.[28] Little is known about the ability of fish, other than shellfish, to concentrate lead in their edible portions, but levels found by limited surveys of marketed oysters and clams were not considered dangerous for human consumption.[29-32] Since oysters can concentrate up to 328 ppm lead from 0.2 ppm lead in sea water after 10 weeks exposure in a controlled environment, it can be assumed that oysters living in waters with a high-lead content will have high concentrations of lead in their edible portions.[30] If environmental pollution at the shellfish resources is allowed to continue unheeded, levels in such food could present a serious public health problem in the very near future.

DRINKING WATER

Water Supplies

Drinking water supplies have been monitored for lead by the Water Pollution Surveillance System since 1962, and the lead content has not generally exceeded the prescribed standard of 0.05 milligram per liter of water.[28] In one recent 5-year summary of trace metals in rivers and lakes of the United States, sponsored by the Federal Water Pollution Control Administration, the maximum recorded lead concentration was 0.140 milligram per liter, with about 2 percent (27/1700) of the samples containing lead in excess of the limit.[33] The sample locations that had the higher levels could usually be related to metal working industries situated along a river. Data from the Bureau of Water Hygiene of the Environmental Control Administration, collected from each of the nine Public Health Service regions across the country, showed that only 2 percent of the drinking water samples exceeded the PHS limit in 1969, but over 75 percent of the samples analyzed by atomic absorption specrometry had detectable lead levels.[34] Some of the elevated lead levels in the tap water samples can probably be attributed to lead pipes.

Most samples for such monitoring surveys are taken from or near water plants. More extensive surveillance of consumer tap samples would be desirable, and would provide more assurance that the level of lead in drinking water was truly reflecting the compliance with the standards.

Water Pipes

There have been serious outbreaks of lead poisoning in Europe, where lead pipe has been commonly used in exterior and interior plumbing. Most of these outbreaks, however, occurred many years ago. In this country, iron, galvanized, and copper pipes are used; however, the use of lead pipe is still permitted by many plumbing codes. Some lead is also used at the joints in caulking or soldering, but not in quantities to cause any hazard. The use of lead pipes is generally harmless with most water supplies, but some waters dissolve lead from plumbing. The reasons for this are not all known, but naturally soft waters, which are slightly acid and low in mineral content, are the most corrosive.[35]

MANUFACTURED SOURCES

Ceramic Glazes

Recently, the Food and Drug Administration (FDA) has been concerned with the lead in ceramic glazes, inserts on tableware, and pottery serving dishes. Most of the items inspected have been of foreign, rather than domestic, origin. In fact, the recent episodes of lead poisoning from glazes on imported pottery initiated an FDA study of all suspect foreign ceramic products. Having found several import items in that category, the FDA has designed a parallel study of domestic dinnerware, to be completed in the near future.

In one study of random market samples, pottery with glazes in cones three to five times the temperature at which most commercial domestic dinnerware is fired, released only 0.02 to 0.45 microgram of lead per milliliter of leaching solution.[36] That test, however, was based on a 30-minute exposure to a hot (60° C) leaching solution.[37] Experience in FDA laboratories has shown that length of exposure is a critical factor in determining the amount of lead released.[38]

As of July 1970, the working action level with respect to extractable lead in clear ceramic glazes was 7 micrograms per milliliter, released after soaking the piece for 24 hours at room temperature in 4 percent acetic acid. The Division of Compliance in the Bureau of Foods, FDA, now attempts to remove from the market any dinnerware that releases more lead than allowed by this interim standard.

Moonshine

Historically famous episodes of lead poisoning from direct contamination of alcoholic beverages have been attributed to lead cooling coils in rum stills (West Indies Dry Gripes), lead linings and joints in cider presses (Devonshire Colic), and lead compounds used for cleaning and preserving wine (Poitou Colic). Although none of these situations now exist in licensed alcoholic beverage industries, illegal whisky production in some areas of this

country is still a cause of lead poisoning. In 1969, thirty percent of the moonshine whiskey samples taken by the Atlanta regional office of the Treasury Bureau's Alcohol and Tobacco Tax Unit contained more than 1 milligram of lead per liter.[39] Most of these stills are made from old lead-coated automobile radiators with lead solder connections. There is some evidence that "moonshine" lead is the cause of sterility, spontaneous abortion, and neonatal malformation among steady female consumers.[40]

Color Additives and Hair Dyes

Some foods, cosmetics, and drugs contain lead in color additives. The Food and Drug Administration currently permits this use of lead at a working limit of 10 ppm in food and 20 ppm in drugs and cosmetics.[20]

In a number of men's hair coloring preparations on the market the principal active ingredient is lead acetate; the average lead concentration is about 2 percent.[41] As of July 1970, lead acetate was neither provisionally nor permanently listed as an official color additive by the Food and Drug Administration. There is no current official objection to the use of lead acetate in hair dyes provided the labels bear cautionary statements against use on cut or abraded scalps, and a warning to wash hands throughly after use.

On the basis of animal and human studies with lead acetate hair dyes the industries have issued a preliminary report stating that the hazard of lead absorption among the users is negligible. FDA toxicologists are still evaluating the industrial report.[42] Meanwhile, the Dermal Toxicity Branch is conducting studies to determine the degree of dermal absorption.

Cigarettes

Like most plants, tobacco contains some lead. Although most tobacco lead remains in the ash, a little less than 1 microgram is present in the smoke of one cigarette.

Examination of blood lead levels in smokers and nonsmokers has produced differing results.[43, 44] The disparity may have been due to difference in lead analysis techniques. Even so, average blood-lead levels in all subjects in all studies have been well within the normal range. It is still possible, however, that cigarette smoking may contribute to an increased body burden of lead since higher mean lead concentrations have been found in the calvarium, rib, and lungs of smokers than in nonsmokers.[44]

CURRENT STATUS

The Federal Government maintains a limited program of surveillance for lead in consumer goods primarily based on isolated incidents of poisoning

due to excessive contamination. Regulations are, therefore, set for specific items, but not based on total dietary consumption. There is at present no standarized monitoring system, nor a standardized lead assay technique for mass sampling although both are on the FDA's list of planned projects and on-going research.

Drinking water supplies are monitored by the Water Quality Office of the Environmental Protection Agency and certified only when their lead content is less than 0.05 milligram per liter. The major drawback in the system is the fact that the water taps themselves are not usually monitored. Tap water monitoring would assure the consumer that the water lead level actually reaching him was no greater than 0.05 milligram per liter and would act as a monitoring system for the delivery network.

Although little is known about the degree to which fish concentrate lead in the edible portions, lead is known to concentrate in oysters. It is, therefore, reasonable to conclude that oysters living in a water environment high in lead could constitute a public health hazard if marketed and consumed.

Illegal moonshine whiskey made in stills using automobile radiators as condensors is the principal recognized source of excess lead in dietary consumer products. This source of lead comsumption is being reduced through increased public education and through full enforcement of regulations banning illegally distilled whiskey.

The cosmetics industries, the U. S. Pottery Association, and the International Lead-Zinc Research Organization in cooperation with the FDA are studying the physiologic, pharmacologic, and toxicologic effects of lead compounds in both ingestion and topical exposures encountered in consumer products.

LEAD IN AIR

INTRODUCTION

Most airborne lead is associated with particles ranging between 0.1 and 5.0 microns in diameter, with a mean for urban air of 0.25 micron.[45, 46] Particle size distribution is an important consideration since it determines both the amount and the nature of airborne contamination. Although larger and heavier particles settle out within a short distance from the source, smaller particles remain airborne and may be carried great distances depending on meteorological conditions. Size and shape also determine the deposition, retention, and absorption of lead particles in the human lung.

DISTRIBUTION OF AMBIENT LEAD PARTICLES

Data on the size distribution of particulate lead indicate that most of the ambient lead particles are less than a micron in diameter. A study at Berkeley, California, showed that 50 to 80 percent of atmospheric lead occurred in particles of less than 1 micron in diameter; in Los Angeles 90 percent of the atmospheric lead was found in particles smaller than 1.6 microns in diameter.[47]

Particulate lead in urban Cincinnati had an average mass median diameter (MMD) in the 0.18- to 0.30-micron range, whereas an average MMD of 0.74 micron was found at a remote site 40 miles from Cincinnati.[45] The average MMD of lead particles in such "background" air had a large variance. An average MMD similar to those in urban areas indicated "young" background particles, those that were transported or dispersed to the site by rather high-velocity winds. A relatively high average MMD, like the 0.74-micron example, indicates "old" particles, which probably resulted from mid-transit coagulation.[48] The average MMD of particulate lead ranged from 0.23 to 0.31 micron in samples collected by means of the Goetz aerosol spectrometer in Chicago, Cincinnati, Philadelphia, Los Angeles, Pasadena, Vernon, San Francisco, Cherokee, and Mojave in 1963.[46]

RELATIONSHIP OF PARTICLE SIZE TO DEPOSITION IN HUMAN LUNG

When airborne particles are inhaled, they may be deposited on the mucus membranes of the nose and throat or in the parenchyma of the lungs.

About half of the inhaled particles reaching the lung are deposited; the rest are exhaled. The quantity and location of the deposition depends principally on the size and chemical composition of the particles.[49] Particles over 5.0 microns in diameter are primarily deposited in the nose and throat. In general, as the size of the particle decreases, the deeper the particle is deposited in the respiratory tract.[49]

In the submicron size range, 36 to 51 percent of experimentally inhaled particles were deposited from an experimental dose of 150 micrograms of lead acetate per cubic meter of air. Following these exposures, an increase in detectable blood lead with little, if any, increase in fecal lead[50] indicates that some retention and absorption of the smaller particles did occur.

Men chronically exposed to 10 micrograms per cubic meter of 0.05-micron-diameter lead particles showed inconclusive evidence of increased lead absorption as measured by blood and fecal lead.[51] Similar experiments using 20 micrograms per cubic meter have been completed, but results have not been reported. Mice exposed for 15 months to low levels of nonirradiated auto exhaust in a cyclic diurnal pattern contained more lead in their bones than corresponding groups exposed to equal or slightly lower concentrations of irradiated auto exhaust even though the daily average lead concentrations were about the same in both atmospheres. Mice exposed to as little as 2.6 micrograms per cubic meter of lead in nonirradiated exhaust had bone lead concentrations higher than control mice.[52]

SOURCES OF ATMOSPHERIC LEAD

Total estimated United States lead consumption increased 9.6 percent (1,202,283 to 1,318,809 tons/year) from 1964 through 1968.[53] Lead used as gasoline antiknock additives increased an estimated 17.1 percent during this same time period. These fuel additives represent a high potential atmospheric lead source and accounted for 19.9 percent of 1968 consumption; 58.0 percent was probably recycled and 22.1 percent was insignificant with respect to atmospheric pollution. In this same year lead from gasoline combustion represented an estimated 96.4 percent (180,000 of 186,614 tons) of lead emissions. Thus 68.7 percent (180,000 of 261,897 tons) of fuel additives was emitted to the atmosphere as opposed to 0.6 percent (6,614 of 1,056,912 tons) of all other lead consumption. Not surprisingly, urban ambient lead levels ranged from 5 to 50 times higher than those of nonurban areas.

Primary and Secondary Lead Smelters

About 980 tons of lead was emitted to the atmosphere from primary

and secondary lead smelting operations during 1968, i.e., approximately 0.5 percent of the total lead emitted to the atmosphere. Whereas particulates of metallurgical dust and fume cover a particle size range of roughly 0.001 to 100 microns, the fume has a particle size range from 0.01 to 2 microns.[55, 56] Concentrations of lead reaching several milligrams per cubic meter have been observed at ground level downwind from smelters. The average lead content of the earth's crust is 16 ppm, but values of several thousand parts per million may be found near smelters. Because of these high air and soil levels, smelters represent potential point-source public health problems in addition to their known risk of occupational exposures.

Other Industries

Excluding gasoline antiknock additives, industrial uses have accounted for approximately 85 percent of the total United States consumption. Emissions from industrial sources other than primary and secondary lead smelters account for approximately 2 percent of the total atmospheric emissions in 1968. In brass founding, for example, molten lead that is overheated during production processes is emitted as lead fumes or particles, in the size range of 0.01 to 2 microns in diameter.

The manufacturing of lead alkyl contributes approximately 810 tons of lead per year to the atmosphere. Nationally this represents only 0.43 percent of the total, but the emissions are concentrated and can present localized health problems. Also in the event of an accident, high local concentrations can occur. The transfer of gasoline from one container to another accounted for release of 40 tons of lead in the air in 1968.

Combustion of Coal and Fuel Oil

Because coal and fuel oil contain lead, airborne particulates derived from their combustion may also contain appreciable quantities of lead. Coal combustion contributed approximately 920 tons of lead (0.5%) to the atmosphere in 1968; fuel oil combustion contributed 24 tons (0.01%).[54] Concentrations of lead as high as 150 ppm in fly ash and 358 ppm in soot have been reported near coal-burning electric power plants.[46]

Incineration

Burning of waste crankcase oil in 1968 produced 3,000 tons of lead emissions, amounting to 1.6 percent of the total estimated emissions. Incineration of solid wastes in municipal incinerators released 320 tons of lead to the atmosphere in 1968–roughly one-third of fuel oil and coal combustion. Like most other general sources of ambient air lead, other than automobile exhaust, emissions on a national basis have probably been underestimated.

Automotive Exhaust

Production of gasoline antiknock additives represent about 20 percent of the total lead used in the United States in 1968. In contrast, the exhaust from gasoline-fueled vehicles accounted for approximately 97 percent of the airborne lead. Nearly all engine gasolines sold in the United States contain lead alkyl compounds added to improve the antiknock quality of fuels without expensive additional refining. The maximum concentration of lead permitted in gasoline is set at 4.2 grams per gallon, a value agreed upon by the producers of gasoline and accepted by the Surgeon General of the United States in 1965. Average concentrations of lead in gasoline have risen from 2.32 grams per gallon in 1963 to 2.59 grams per gallon in 1970.[57]

Lead alkyl compounds are converted to lead halides and lead oxyhalogenates in the internal combustion engine, and 70 to 80 percent of that lead is emitted as particulate matter from the tailpipe.[46] The age and condition of the vehicle as well as the design of the engine and exhaust system govern the particle size and the amount of lead emitted to the atmosphere. About 95 percent of total lead in the particulate components in hot, concentrated automobile exhaust and in cooled diluted exhaust was associated with particles having aerodynamic diameters below 0.5 micron.[58] The relatively high concentration and submicron size of particulate lead in automobile exhaust are consistent with atmospheric measurements.[45, 46]

Lead alkyl compound also reaches the atmosphere directly by evaporation from gas tanks, from carburetor vents, and during filling at the service station. Lead alkyl additives also interfere with catalytic exhaust treatment systems designed to reduce hydrocarbon emissions. Principally because of this, and not from health considerations alone, the Department of Commerce, Technical Advisory Board Panel on Automotive Fuels and Air Pollution, and the Department of Health, Education, and Welfare have recommended that legislation be enacted to establish the authority of the Federal Government to regulate the use of automotive fuel additives, requiring the availability of low-lead fuel by 1972 and unleaded fuel by 1974.[59] Tax incentives on production of both of these fuels have been recommended by some agencies because of the slightly higher cost of producing nonleaded high-octane gasoline. The economic burden of producing an unleaded gasoline of the desired octane will fall heavily on small refiners.[59]

METHODS FOR MEASURING ATMOSPHERIC LEAD

Sample Collection

The most commonly used methods for collecting air particulates for lead analysis involve filtration through either a glass fiber or membrane filter for 24 hours.[60] Cellulose filters may be used, but they are not recommended because of variation in collection efficiency. Particles collected by the filtration method fall in the 0.1-micron-diameter range; the usual practice is to

sample for a 24-hour period. In order to determine lead content in the larger, settleable particulates, samples are collected by means of a dustfall procedure, usually over a period of 30 days.

Special sampling devices that can separate airborne particulates by size are available if there is a need to determine the particle size distribution of lead. These samplers are not in common use because of their high cost, low sampling rate, and complexity of operation.

Organic lead—lead tetraethyl, and lead tetramethyl—can be trapped by passing pre-filtered ambient air through an absorbing train containing crystalline iodine or high-purity activated carbon. Although the system is cumbersome and time consuming, and requires constant attention, it performs adequately for the collection of gaseous lead compounds in the microgram-per-cubic-meter range.

Sample Analysis

By suitable processing, both the particulate and gaseous lead samples are converted into solutions of lead salts that may be analyzed by any one of several acceptable methods. Although lengthy and tedious, the colorimetric Dithizone method, which has been the method of choice for a number of years, is very specific, accurate, and precise.[61] The use of atomic absorption spectrometry has gained widespread acceptance within the past 5 years because its high initial instrumentation cost is offset by its speed and accuracy. Satisfactory results can be obtained by the use of emission spectrometry or polarographs, but these methods require a greater degree of analytical skill than atomic absorption spectrometry. An instrumental method that would permit the direct, simultaneous measurement of gaseous and particulate atmospheric lead on a continuous basis is currently under development.

Air samplers currently available for sampling a variety of size ranges of airborne particulates differ in their capabilities and sampling rates. A recently developed air sampler, presently being field tested, promises to provide adequate samples for determination of size distribution of lead particulates over a wider range than is currently possible.[61] At present the Air Pollution Control Office (APCO) uses the Lee modification of Anderson Cascade impactor* and electrostatic fractionator.

To fully assess this aspect of ambient air lead levels, the National Air Surveillance Network (NASN) of APCO instituted a network early in 1970 to determine the size distribution of lead and other components in the air at six major urban areas. Special short-term studies are being conducted to gather additional data relative to particle size distribution and ambient levels at selected sites.

*Use of a company or product name does not constitute endorsement by the U. S. Environmental Protection Agency.

Limited studies of particle size distribution have been carried out in the San Francisco Bay area. Ordinarily, two fractions are obtained with a centrifugal separator, but Cascade Impactors are sometimes used to provide four or more fractions. The Kettering Laboratory of the University of Cincinnati College of Medicine conducts intermittent particle size distribution studies as part of a long-term lead project. Short-term studies are frequently done in the Los Angeles area by the University of California, Riverside, with the Lundgren Impactor sampler. Stanford Research Institute uses the Goetz Aerosol Spectrometer in special contract studies relating to atmospheric lead.

ATMOSPHERIC SURVEILLANCE

Routine Surveillance of Particulates

The National Air Surveillance Network operates about 270 glass-fiber filter samplers for measuring airborne particulate pollutants in urban and nonurban areas throughout the United States. Samples are collected over a 24-hour period every 2 weeks on a random schedule and analyzed for lead and 35 other trace metals by emission spectrometry.[62-66] National Air Surveillance Network data through 1966 have been published, and more recent data are scheduled for publication early in 1971. Many state and local air pollution control agencies conduct particulate surveillance programs similar to that of APCO. Some of the samples generated by these networks are analyzed for lead, and the data are contributed to APCO's National Aerometric Data Bank.[67]

The combined Federal, State, and local effort provides reasonably adequate definition of ambient lead concentrations for all cities over 50,000 population, for many with less than 50,000 population, and for a cross section of nonurban areas. New York City also conducts special particulate surveillance programs, which include measurement of lead.

Routine Surveillance of Organic Lead

There is presently no organized routine surveillance program for determining organic lead in ambient air. The current method for assaying lead in samples of particulate pollutants does not distinguish between organic and inorganic lead compounds. Organic lead is thought to contribute only trivial amounts to total ambient air levels.

Urban Versus Nonurban

The effect of civilization on lead emission becomes obvious when concentrations at urban stations are compared with those at nonurban stations. Average particulate lead concentrations at urban stations for 1965 ranged quarterly from 0.1 microgram per cubic meter in Paradise Valley, Arizona, to as much as 2.3 micrograms per cubic meter in Burbank, California.[66] In

contrast, the range of averages at nonurban stations was an order of magnitude lower than that at the urban sites. They ranged from no detectable concentration at the White Pine County, Nevada, station to 0.17 microgram per cubic meter at the Washington County, Rhode Island, station. Nonurban stations may be perceptibly influenced by surrounding urban areas and/or highway traffic. Even the "remote" stations are located in national park areas, where electricity is available, and where there is tourist traffic during at least some seasons. These remote stations may therefore owe some portion of the lead detected there to man's activities.

Special Studies of Selected Cities

In 1961-1962, a 12-month study of ambient-air- and human-blood-lead levels was made in Cincinnati, Los Angeles, and Philadelphia.[68] Extensive measurements of lead in the three cities showed variations with season of the year and location in the city; these data were found to conform to the range of urban concentrations reported by the national network.[68] The highest seasonal lead concentrations typically occur in the fall and winter. Ambient-air lead concentrations are also a direct function of traffic density and speed. Cholak and co-workers reported that the lead content of soil in the Cincinnati area varied from 16.4 to 360 ppm.[69]

A similar study in the same cities and four additional large cities—Chicago, Houston, New York, and Washington—will be completed by the end of 1971. In these studies, six to eight sampling stations are operated in each city at sites selected to provide a reasonable lead profile for the city. For each city, a nearby area with a minimum of lead sources is selected to provide comparable information of suburban background lead levels, when possible.

CURRENT STATUS

Lead air pollution problems still occur in areas near lead smelting and reprocessing plants, lead-using industries, and coal-burning electric power plants. Small amounts of airborne lead come from soils and from solid waste incineration.

Although particulate sampling devices currently used by NASN to measure ambient pollutants do not have the capability of collecting total atmospheric lead, various components of the Government and industry are deeply involved in the development of an acceptable sampling device. Techniques to provide accurate, economical, and rapid measurements of atmospheric lead are also being developed.

Limited research has indicated that ambient lead concentrations are a function of season, location, and traffic density and speed. More intensive studies are being conducted to fully define the role of other pollutants and

variables in determining the lead concentrations in ambient air and relating the levels to health effects.

Lead in automobile fuels is the major source of atmospheric lead. Various committee studies, research and development, and other activities continue to develop the Federal position relating to reduction of the atmospheric lead levels contributed by the automobile.

LEAD IN OCCUPATIONAL EXPOSURES

INTRODUCTION

Industry will probably remain a major source of excessive lead exposure because of the wide use and versatility of the metal. Where most severe exposures among the general populace have been due to lead ingestion, exposures among industrial workers have been primarily due to lead inhalation from fumes and dusts produced by heating, pulverizing, and grinding the metal. Industrial hygiene measures for controlling workers' exposure have been developed for nearly all recognized processes. Incidence of clinical lead poisoning in the past has usually been attributed to unrecognized exposures introduced with new industrial processes. The incidence, however, has decreased as knowledge of lead toxicity and hygienic technology has increased. Efforts have been made to reduce exposure in many work categories below the minimum acceptable limits through the enforcement of safe work standards for lead in air and urine set by the Walsh-Healy Act.[70]

In a 1964 U. S. summary of industrial exposure, measurements of lead in inplant air and employee urine samples showed that since 1934, exposure had decreased by several magnitudes for workers in seven of eight categories representing the major uses of lead.[71] The report did not note that overexposure to lead was common in small shops nor include data on munitions plants.

To up-date this survey, the Bureau of Occupational Safety and Health telephone-interviewed 24 key individuals across the country who were responsible for the control of industrial lead exposures. The 1969 survey indicated that overexposure to lead still occurs: (1) in small shops, (2) among itinerant workers (e.g., construction workers), and (3) where new or unusual lead-using processes had been introduced. Repeated lead poisoning has occurred, for example, in crews spraying lead-based paint to renovate missile silos.[72] Problems of inadequately staffed and supported occupational health facilities noted in 1964 were also found unimproved in the 5-year period. The survey found that improved control of lead exposures had resulted from (1) removal of the source of exposure, for example, substituting titanium for lead in paint or using construction materials not requiring lead paint; (2) orientation of supervisors; (3) improvement of monitoring; and (4) improvement of control techniques.

SMALL SHOPS

Small-shop operations, in particular battery recovery and welding, are

responsible for the largest group of uncontrolled lead exposures. The types of plants, operations, and sources that should be monitored for suspected cases of lead exposure are legion. One state health department surveyed 23 small shops* from 1966 through 1969.[73] About 20 percent (118 of 568) of all blood samples drawn during these 4 years exceeded 80 micrograms of lead per 100 grams of blood. Blood levels exceeding this limit were considered presumptive evidence of overexposure to lead since these workers were not clinically examined by a health department physician. This same health department was effective in preventing one company with a particularly bad record of overexposures to lead from locating in its jurisdictional area.

Another state set threshold limit values of 200 micrograms per cubic meter of industrial air based on a 40-hour work-week exposure, but found levels above this in 35 percent of 537 samples taken in 1967.[74] Most of these were also in small-shop operations.

Itinerant construction workers, though fewer in number, pose an even more formidable problem for control agencies because the workers are usually not available for regular checkup.

REPORTING AND DIAGNOSIS OF POISONING

Most industrial hygiene laboratories now use blood-lead levels in addition to air-lead levels to monitor and regulate worker exposures. Some use stippled-cell counts; a few, urinary lead and/or coproporphyrins levels; fewer use urinary delta-aminolevulenic acid (δ-ALA) levels routinely; and none routinely use blood δ-ALA dehydrase levels.[12] Some laboratories sample workers' blood either quarterly or annually; others sample when overexposures are suspected during and after surveys or on a demand basis. Quarterly sampling is done when the surveys indicate a need for more information. Medical examinations are performed in addition to the biological and environmental analyses when physicians are available.

Blood-lead levels are generally accepted as superior to urine levels as indicators of lead intoxication. Nevertheless, industrial hygienists and physicians comparing medical reports with laboratory data have commonly noted that many workers have blood-lead levels far in excess of recommended limits, but show no signs or symptoms of lead intoxication. Hence an elevated blood-lead level *per se* is not conclusive evidence of clinical lead poisoning. The number of reported "mild, chronic" cases may therefore be exaggerated because the diagnosis is usually based on blood levels alone.

Lead intoxication, particularly in its early stages, is difficult to diagnose.

*Battery manufacture and rebuilding, can manufacture, chemicals, communications, electrical work, exterminating, food processing, glass manufacture, junk yards, newspaper and print shops, machining, metal working, paint manufacture and application, fuel blending, plastics, phonograph records, police rifle range, packaging, refrigerator manufacture, ship salvage, smelting and refining, trucking, and vocational training.

The development of refresher courses in occupational medicine and the establishment of a nationwide compulsory reporting system for occupational exposures would both help to identify and consequently reduce this problem.

Cases of acute lead poisoning are rarely reported in large industries with well-staffed medical or industrial hygiene departments or in states and local jurisdictions with well-staffed occupational health departments. Unfortunately cases are rarely sought in areas with less well-staffed health departments and have even been found in certain branches of the U. S. Armed Forces where widespread small operations are common. Inadequate investigations because of understaffed agencies, opening of new small shops, failure to diagnose early lead poisoning, and problems in blood lead analysis have all mitigated against improvement.

CURRENT STATUS

Industries with good occupational health departments have minimized lead exposure problems. Nevertheless problems still occur in places with inadequate control by reason of peculiar work situations, itinerant workers, new operations, small shops, or inadequate medical laboratory and/or occupational health services. There is still need for more uniformity and vigor in the control of industrial lead exposures.

The problems of industrial lead exposure are being reduced by better enforcement of present legislation, enacting new legislation where needed, increasing the number of occupational health physicians and industrial hygienists, establishing refresher courses for both industrial and non-industrial physicians, and certifying medical laboratories for tissue lead analyses.

LEAD POISONING IN CHILDREN

INTRODUCTION

Lead poisoning among children in the United States is almost invariably caused by repeated ingestion of chips and flakes of lead containing paint and plaster from the walls, windowsills, and woodwork of pre-World-War-II houses. It has a high incidence only among children living in city slums, where accessibility to flaking and peeling lead paint and broken plaster, lack of knowledge among parents that ingestion of lead paint is dangerous and even lethal, frequent inadequate parental supervision of young children, and a high incidence of pica (an appetite for nonfood items such as dirt, paper, paint, and plaster) all favor lead poisoning.

EXTENT OF PROBLEM

Children under 6 years of age are the main victims and those between 1 and 3 years comprise 85 to 90 percent of the childhood cases.[75] Boys and girls are affected equally. Lead poisoning occurs year round, but lead encephalopathy (brain injury caused by lead), a very serious complication, is much more frequent during the summer. Some cases have been reported in winter when leaded battery casings are burned for fuel and the fumes are inhaled or when there is prolonged contact with the ashes.

Nobody knows exactly how many children in the United States are exposed to this health hazard, or how many are actually poisoned, for many cases of childhood lead poisoning go undiagnosed. Since the problem is closely related to poor housing conditions, an educated guess could be based on the number of old, deteriorating houses in the United States and the known rate of lead poisoning among children living in such houses. According to the 1960 Housing Census, 7.5 million of the occupied housing units in the United States built in or before 1939, when lead paint was commonly used on interiors, were classified as deteriorating or dilapidated. Approximately one-eight of the population, or 1.25 million children ages 1 to 3 years old, lives in these houses.[75]

Although since the 1940's lead pigment has been replaced by titanium in interior paints, recent surveys in Baltimore, Philadelphia, and Minneapolis revealed selected slum areas where from 40 to 80 percent of houses still contained quantities of flaking lead paint. Among children ages 1 to 6 years, 10 to 25 percent living in high-risk areas are found to have higher than

normal blood-lead levels and 2 to 5 percent have clinical symptoms of lead poisoning. The latter projected to a national level represents roughly 40,000 children, ages 1 to 3 years old.[75]

Effects of Childhood Poisoning

Until the advent of chelating agents—therapeutic agents that bind the lead ions and increase their excretion rate—about two-thirds of the children with lead encephalopathy died. Even with the use of chelating agents, the fatality rate remained 30 percent for many years. More recently, with the use of the chelating agents and supportive therapy, the fatality rate has been reduced to less than 5 percent; but many of the survivors are left severely handicapped.

In Chicago a follow-up study of 425 children who were treated for lead poisoning revealed that 165 had some kind of neurological sequela. Among the 59 children in this group who had symptoms of lead encephalopathy before treatment, 48 were left with various handicaps: 32 had recurrent seizures, 22 were mentally retarded, 8 had cerebral palsy, and 4 had optic atrophy.[76]

Epidemiological studies in Queensland, Australia, have demonstrated a high incidence of chronic kidney disease among patients who had lead poisoning in childhood 10 to 40 years previously. Half of these patients with kidney damage also suffered from gouty arthritis, and many had severe high blood pressure, mental impairment, and psychiatric disorders.[77] A similar study in the United States did not find this high incidence of chronic renal disease and pointed out that the Queensland cases were not typical of those seen in the United States.[78]

Detection Difficulties

Clinical lead poisoning in children is prevalent only among families who are least able to improve their living conditions and who are not generally well-informed. The well-informed segments of the population are seldom affected. Many health agents who work among the poor are not aware that lead poisoning in children is still a problem. Childhood lead poisoning is a disease that health workers may not recognize, even though it exists in epidemic proportions, because it has no distinctive clinical features. Signs and symptoms of lead poisoning such as anemia, anorexia, abdominal pain, constipation, increased irritability, and vomiting can all be easily mistaken as manifestations of other childhood illnesses.

Unless a physician inquires specifically as to whether the child has eaten chips of paint or plaster and draws a blood specimen for lead determination, he is likely to miss the diagnosis altogether. He may treat the child for something else, only to be confronted later with the same patient, who may then exhibit signs of brain injury which may already be irreversible. The

probability of this happening is supported by the fact that in some cities with a high incidence of lead poisoning, certain hospitals serving children from known "lead belts" report few or no cases of the disease.[75]

Inadequacy of Housing Codes

Many cities, where lead poisoning is a public health problem, do not have health or housing codes adequate to protect tenants from exposure to lead paint. Even in cities with codes specifically prohibiting lead paint in the interior of dwellings, enforcement of such codes is generally far from satisfactory. Where city officials are interested in enforcing codes, they frequently do not have enough inspectors and sanitarians to carry out the necessary procedures for enforcement—inspection of houses, collection of paint specimens, testing for lead content, and reinspection.[75]

Another reason housing codes are not an effective solution is that enforcement relies primarily on the criminal process, usually in the form of misdemeanor prosecution in the lower criminal courts where procedural and conceptual difficulties can delay action indefinitely.

The recurrence rate of lead poisoning is high. Failure to remove lead paint from a house where a child is known to have developed lead poisoning usually means that a treated child returns to the same environment to be exposed again. Among survivors of acute lead encephalopathy who are re-exposed to an environment that contains lead paint, the incidence of severe permament brain damage is almost 100 percent.[75] Development of inexpensive methods for paint covering and removing is essential.

PROPOSED COMMUNITY CONTROL PROGRAM

The public must be alerted to the dangers of eating paint, the symptoms of lead poisoning, and the course of action to follow when lead poisoning is suspected. So that they will always have an index of suspicion, physicians, nurses, social workers, and all other health workers must also be made aware of the prevalence of lead poisoning among children. The Surgeon General has recently stated that lead poisoning should be a reportable disease.

Mass screening programs must be conducted in "lead belts" for all children between 1 and 6 years of age. Children found to have elevated body-lead levels should be referred to a medical center for further diagnosis and treatment if necessary. Steps should be taken to prevent their re-exposure to lead in the home, and all children treated should be re-examined periodically for possible re-exposure.

Effective health and housing codes concerning lead and lead poisoning must be established and diligently enforced. Where codes are not enforced, court action may be necessary.

ECONOMIC CONSIDERATIONS

The estimated cost of treatment and institutionalization to the age of 60 of a person who incurs severe permanent brain damage from lead poisoning in childhood has been calculated to be around $220,000.[79] Complete removal of lead paint from an average rowhouse with 10 windows, two doors, and baseboard would cost $250 to $300; replacement of window and door units and baseboards in such a house would cost $600 to $1,200. These figures show only the difference in dollar costs between preventing lead poisoning through paint removal and permitting severe brain damage to occur in children. They do not take into consideration the intangible loss of useful or potential manpower.

CURRENT STATUS

Many comprehensive health care projects for children sponsored by the U. S. DHEW Children's Bureau include programs to control lead poisoning. Pilot projects are currently operating at the Hill Health Center in New Haven, Connecticut, the Children's Hospital of the District of Columbia; and several community hospitals in New York City and Chicago. Volunteer groups are also working in Baltimore, New York, Chicago, New Haven, Cleveland, and the District of Columbia.[74]

In 1969, the Lead Industries Association published a booklet entitled "Facts About Lead and Pediatrics," in which seven steps to the prevention of lead poisoning were presented. The booklet is distributed to physicians, public health authorities, social workers, city officials, and others who can help achieve prevention and control of the disease in children.

The Bureau of Community Environmental Management is preparing guidelines for local childhood-lead-poisoning control programs based on its own active collaboration with several communities. These guidelines should be published in 1971. The Bureau is also supporting studies at the University of Minnesota on lead metabolism following single doses and at the John Hopkins University on sequelae of lead encephalopathy and lead poisoning treatment with chelates.

The Comprehensive Health Care for Children and Youth Program of the Health Services and Mental Health Administration supports demonstration community action programs against lead poisoning in Chicago and New York City. They also support research on the development of improved screening methods.

The Johns Hopkins University School of Medicine has received a grant from the U. S. DHEW Children's Bureau to do an urgently needed study of tests used in screening children for lead poisoning. The goal is to develop a simple, quick method of determining the amount of lead in human blood for use in large-scale screening programs. At present, a physician or skilled

technician must puncture the vein to draw enough blood for blood-lead determination. The successful testing of more than 120,000 children in Chicago is evidence that this method of screening for lead poisoning is feasible on a large scale. Scientists are, however, seeking a quicker, but equally reliable, method that will require only the small amount of blood obtained from a finger prick.

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