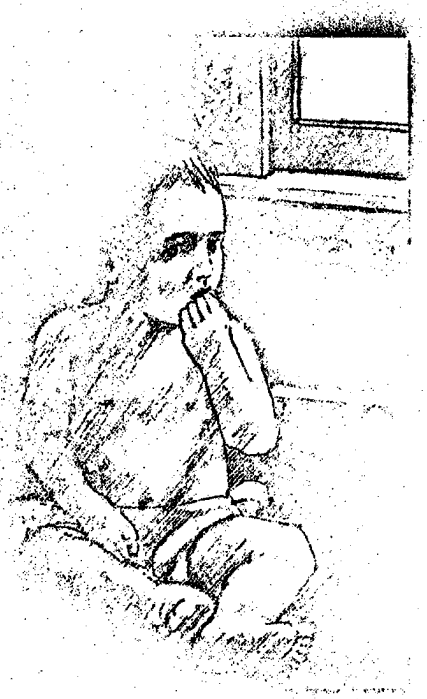
LEAD



feature

pollution and poisoning

High lead levels are dangerous to man, and ambient concentrations are presently rising

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Lead has been mined and used by man for many years. Widespread early applications of lead are readily understood in light of its many desirable properties—relatively easily refined from natural ores, ductility, high resistance to corrosion, and other properties.

In both Greek and Roman culture, lead was used for manufacturing cooking utensils of the wealthy and in the lead pipes for the extensive plumbing systems of their homes. Studies indicate that Roman civilization deteriorated largely as a result of extensive lead poisoning (high incidence of infant mortality, mental retardation, and sterility) in the ruling class. This theory is supported by data showing a high lead concentration in the bones of their remains.

Of the nonferrous metals, lead is one of the most widely used in industry and everyday life. The annual consumption in the U.S. alone is well above one million tons. The storage battery industry is the largest consumer, about 40%, and the petroleum industry consumes about 20%, producing lead alkyls as gasoline additives. These 300,000 tons of lead are, of course, added directly into the ambient air. By contrast, only about 45% of the total lead consumed is recovered from metal products and batteries.

Today lead is an ubiquitous element present in food, water, and air. Lead aerosol is a common air contaminant. Dated samples of snow in northern Greenland indicate that up to 1750, there were about 20 μg of lead per ton of ice until the Industrial Revolution. By 1860, this had increased to 50 μg/ton. The proliferation of the automobile since World War II also resulted in sharp increases—in 1940, 80 μg; in 1950, 120 μg; and in 1965, 210 μg. In the past two decades, man's continuing use of lead has produced an environmental level far above that which would exist naturally, and this could have grave consequences on human health.

Sources of lead

Lead is a natural constituent of soil, water, vegetation, animal life, and air. Significant sources of naturally occurring lead include dust from soils and particles from volcanoes. In contrast to certain other metals such as mercury, lead in its elemental form is not a major source of poisoning.

In medieval times, the practice of "sweeting" wine with lead or lead acetate became a serious toxicological problem. Even today, alcoholic beverages of illicit origin are occasionally incriminated as the source of lead poisoning. Old automobile radiators are often used as the condenser component in illicit stills and contain enough solder to cause dangerous contamination of the "moonshine." Lead encephalopathy (disease of the brain), nephritis (disease of the kidneys) with gout, and other lead-related conditions have been reported in moonshine consumers.

Until the 1950's, most interior paints contained lead pigments which were a major source of childhood lead poisoning. Since then, lead oxide has been replaced by titanium dioxide. However, some old houses were painted countless times with leaded paint, and thick chips of leaded paint fall off as the walls and ceilings peel. Putty also contains lead and is even more likely to be found in substandard dwellings. Since children between the ages of one and four or five ingest nonfood particles of all types, about 8% of children in ghetto areas suffer from some degree of lead poisoning.

Another major lead source is earthenware pottery improperly glazed with lead. Large amounts of lead may be leached out of the glaze into certain foods. Today, man continues to pollute his environment with lead from other sources such as manufacturing, use of pesticides, incineration of refuse, and combustion of coal and leaded gasoline. Available data indicate that combustion of leaded gasoline is the major source of atmospheric lead in urban areas.

Atmospheric lead

Antiknock agents of lead alkyls, in the form of tetraethyllead (TEL) or tetramethyllead (TML), have been added to most gasoline since 1923. Their use rapidly became a cause for concern because lead intoxication and even death occurred among those occupationally exposed. In 1925, the U.S. Surgeon General appointed a committee to investigate possible public health hazards in the manufacture, distribution, or use of leaded gasoline. In the following year, the committee proposed a set of regulations which were concerned only with precautions in manufacture and distribution. The proposed regulations were adopted voluntarily by the petroleum industry.

About 1930, an attack on the significance of lead as a hazard to the general public began, but for the following 30 years, there was little interest in the potential environmental pollution problem of lead. Then in 1958, the Ethyl Corp. sought and was granted an increase in the concentration of lead in gasoline by the Surgeon General. Today, the amount of TEL ranges from 2 to 4 g/gal of gasoline.

A survey in 1964 indicated only 88 cases of TEL poisoning reported in the U.S. and Canada, subsequent to adoption of the regulations in 1926,

Uses of lead

Metal products
- cable covering
- solder
- caulking lead
- ammunition
- bearing metals
- sheet lead
- pipes
- type metal
- brass and bronze

Storage batteries

Chemicals
- tetraethyllead

Pigments

Miscellaneous
- weights
- galvanizing
- annealing
- shielding

and only 16 fatalities. This record is not bad, considering that several million tons of TEL have been manufactured and distributed since 1926.

Lead exhausted by automobiles originates in the antiknock fluid in the gasoline. The fluid contains lead alkyls and organic scavengers, ethylene dichloride, and dibromide, whose function is to combine with the lead to form inorganic lead salts, chiefly bromochloride, that enters into the atmosphere as part of the exhaust gases. Since only negligible amounts of the lead alkyls in the gasoline are exhausted directly into the atmosphere, it is unlikely that the health of the general public would be affected.

The amount of lead discharged into the atmosphere in exhaust gases varies, depending on driving conditions from about 25% to 75% of the lead intake in the fuel. At low speeds, the lead tends to be retained in the exhaust system and is discharged at some later period when the engine is run at greater speeds. The level of atmospheric lead varies directly with the volume of traffic and the size of the community. Los Angeles, with a population of more than 2.5 million, has the highest lead concentration of the communities studied, with an approximate mean of 5 $\mu g/m^3$ of air. Other urban communities with a population greater than 2 million have values of about 2.5 μg. Communities smaller than a million have a mean atmospheric concentration of about 2 μg, and communities with a population of less than 100,000 have a mean value of about 1.7 μg. The mean today is rising by as much as 5% per year, and week-long averages of 8 μg now occur in San Diego.

From the atmospheric precipitation samples collected by a nationwide network of 32 stations throughout the U.S., lead concentration is correlated with the amount of gasoline consumed in the area and the amount of lead in surface water supplies. Thus, any further rise in the dissemination of lead wastes into the environment can cause adverse effects on human health.

Lead absorption

Two major routes by which lead enters the human body are the alimentary and respiratory tracts. Absorption of inorganic lead through the skin is insignificant. Trace amounts of lead are found in normal daily diets. The mean dietary intake of lead for adult Americans is approximately 300 μg/day. Approximately 6–7% of the metal ingested is actually absorbed, but the percent absorption does not appear to be materially influenced by the amount of intake.

In addition to the dietary intake, lead absorption by the respiratory tract has been studied. The amount of lead absorbed from a given air concentration varies according to the particle size. Particles of approximately 1 μ or smaller will probably be retained by the lung, with up to 50% retained in the body, and the remaining portion expired with air. Particles larger than 2 μ impinge on the mucous lining of the airways, are swallowed, and are handled as though ingested by mouth.

About 75% of particulate lead in vehicle exhaust is less than 0.9 μ in mean diameter, a size easily retained by the lung. For example, an urban adult, breathing air containing 3 μg of lead and respiring about 15 m^3 of air per day, will absorb about 20–25 μg of lead from the respiratory tract. Roughly the same amount absorbed from a normal daily diet, this atmospheric lead contribution to the body approaches, and in some communities, even surpasses the contribution made by dietary intake.

Lead metabolism and effects

Under normal conditions, more than 90% of the lead retained in the body is in the skeleton. The amount of lead which is not retained in the body is excreted back into the gut in the bile and also excreted in urine, sweat, hair, and nails. Long-term exposure at some daily level of lead is potentially hazardous because lead is a highly cumulative poison. Under conditions of abnormally high calcium metabolism, such as feverish illness or during cortisone therapy, lead may be mobilized and a toxic amount released from the skeleton. Only a small fraction of the total bone lead need be mobilized to add an appreciable increment to the small soft tissue pool.

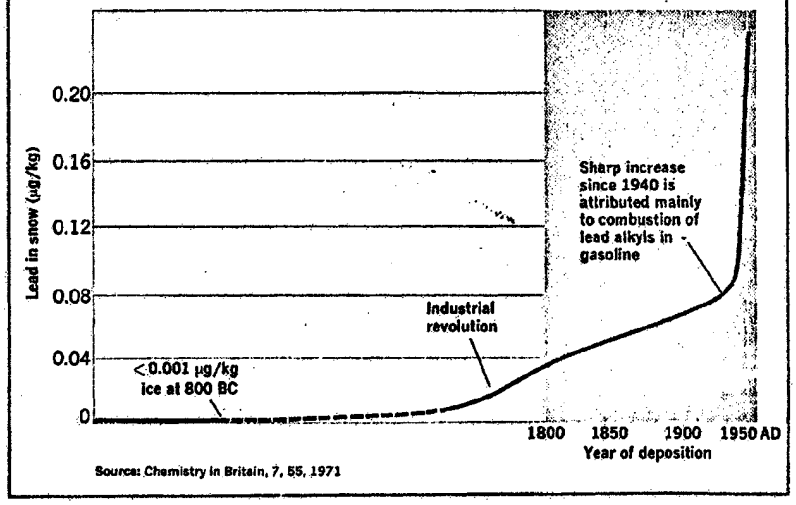
With the passage of time, absorbed lead becomes progressively more deeply buried in the bone matrix. This develops a potentially dangerous pool of exchangeable lead which can persist for months or even years. Acute lead poisoning can occur long after exposure to abnormal amounts of lead has ceased.

Lead poisoning results from very high levels of lead in the human body, particularly in the soft tissues. While in the soluble form, lead is both mobile and toxic, and most often affects the blood, kidneys, and nervous system.

The effect of excessive amounts of

Figure 1

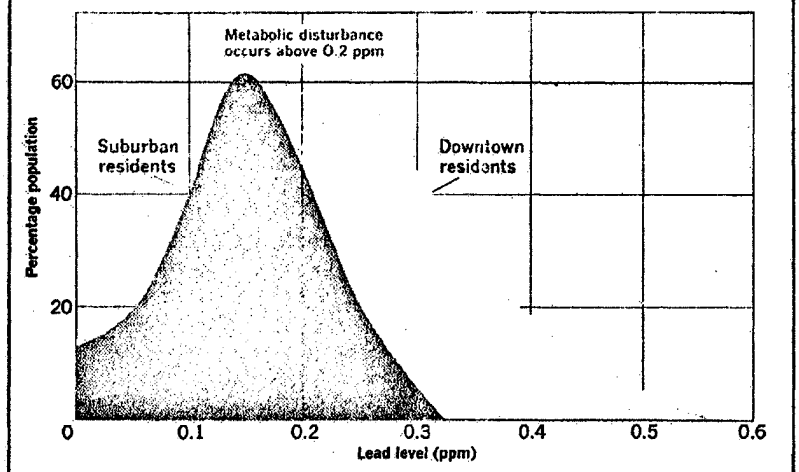
Lead content of Greenland snow layers continues to increase



Source: Chemistry in Britain, 7, 55, 1971

Figure 2

Lead levels* differ in suburban and downtown inhabitants



* Lead levels in blood of Philadelphia males 1961-2

Source: Chemistry in Britain, 7, 56, 1971

lead in the blood is anemia. The presence of stippled cells is characteristic of the blood of a patient with lead poisoning. Chronic nephritis, sometimes accompanied by gout, is a disease characterized by a scarring and shrinking of kidney tissues. In severe cases, it may lead to ultimate kidney failure.

The effects of lead on the central nervous system are seen in behavior problems characterized by convulsions or swelling of the brain. A single attack of acute encephalopathy can cause brain hemorrhage, mental retardation, and other permanent neurological injury. Similarly, repeated bouts of lead poisoning can also cause permanent brain damage ranging from subtle learning deficits to profound mental incompetence and epilepsy.

Chronic lead poisoning results from a slow buildup of lead over a period of years or the aftereffects of acute lead poisoning. Acute lead poisoning has easily recognizable symptoms, and if treatment is administered in time, permanent damage can be avoided. Chronic lead poisoning, however, without symptoms or with vague symptoms, is difficult to diagnose and may be recognized only after irreversible damage has been done.

Childhood lead poisoning

Lead poisoning in children is primarily a metropolitan disease in preschool age children who live in deteriorated housing previously painted with thick layers of leaded paint. As they are converted into slum tenements, much of the leaded paint peels and crumbles and is readily available to a young child. A chip of paint about the size of an adult fingernail can contain between 50 and 100 mg of lead. A child swallowing a few small chips a day easily ingests beyond the tolerable adult lead intake.

Lead poisoning is more dangerous in children than adults. During the first few weeks of abnormal ingestion of lead, generally no symptoms appear. After about six weeks, minor symptoms such as decreased appetite, constipation, clumsiness, fatigue, irritability, lethargy, headache, abdominal pain, and sometimes vomiting occur. Repeated ingestion of lead can lead to intermittent drowsiness and stupor, more persistent and forceful vomiting, and convulsions. With more prolonged or intense ingestion, the disease can culminate in intractable convulsions and coma (the symptoms of encephalopathy) and sometimes sudden death.

The minimum blood concentration of lead below which it is most unlikely that poisoning will occur is 80 μg of lead per 100 ml of blood for adults. In children, however, the threshold should be much lower. Mild symptoms of lead poisoning have been found in children at 60–80 μg. As the blood–lead concentration rises above 80 μg, the risk of severe symptoms increases sharply. As many as 50,000 children in the United States today suffer from lead poisoning. Even though physical recovery is possible, brain damage and mental retardation often ensue, for the ages of one to four or five are critical years in the growth and development of the brain.

Lead poisoning control

The most frequently used lead poisoning treatment is to bind the soluble lead to chelating agents which remove lead ions from soft tissues and prevent deposition of lead on the surface of the red blood cells. With chelating agents, high lead levels in tissues can be rapidly reduced to levels approaching normal, and adverse metabolic effects can be promptly suppressed. However, if there is a large residue of lead in the stomach, chelates may encourage such rapid absorption of lead that death occurs. Chelates may also release lead already stored in the bones, causing further complications. Chelating agents frequently used to treat lead poisoning are calcium disodium ethylenediamine tetraacetate (EDTA), 2,3-dimercaptopropanol or British Anti-Lewisite (BAL), and *d*-penicillamine.

Before chelating agents were available to treat lead poisoning, about two thirds of all children with lead encephalopathy died. Now the mortality rate is less than 5%. Unfortunately, treatment has not substantially reduced brain damage and mental retardation in the survivors.

Among the various substances that man concentrates in his immediate environment, lead wastes have been accumulating rapidly during the last century. As far as is known, lead is not a trace metal essential to nutrition.

One must remember that lead absorbed in seemingly harmless trace quantities over a long period of time can accumulate to exceed the threshold level for potential poisoning and produce delayed toxic effects. Concentrations of lead in soft tissues as well as in bones continues to rise from birth through the fourth or fifth decade of life. Furthermore, concentrations are higher in the U. S. than in other parts of the world.

Normal concentrations of lead in adults are below 40 μg/100 ml of blood. The average concentration of lead in Americans is 27 μg. The question is whether this average is normal or whether it represents a serious increase beginning with adding TEL to gasoline and thereby reflecting documented increase in environmental lead. If this is so, then any further increase might well be dangerous. Unfortunately, there is no baseline with which to compare contemporary populations. Low level effects are yet to be recognized.

Since 50% of airborne lead can be absorbed from diet, hopefully, the amount of airborne lead will remain constant since automobile and oil

companies have started to eliminate leaded gasoline. Some states have laws prohibiting leaded paints for interior painting. However, applying lead-free paint over old leaded paint does not solve the problem. When paint peels, the leaded paint again becomes available to the child. Old leaded paint must be removed from substandard housing. Where rehabilitation is impossible, urban redevelopment projects should replace substandard housing.

Present American industrial standards consider 200 $\mu g/m^3$ to be an acceptable level of occupational exposure. A known effect of chronic lead exposure among industrial workers is peripheral nerve disease, affecting primarily the motor nerves of the extremities, while symptoms of acute lead poisoning are strikingly absent. Federal and state governments are reponsible for instituting effective control measures in industry.

Whether or not lead concentrations measured are hazardous to human health, any further increase of lead in the environment will result in further concentration in some food chains, leading ultimately to toxic doses for man or for some other important organisms. Lead pollution, therefore, must be controlled.

Additional reading

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