

Lead and Lead Poisoning from Antiquity to Modern Times¹

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ABSTRACT. Lead, because of its low melting point, was one of the first metals used by man. It was probably isolated soon after the ancients discovered the use of fire. Archeological discoveries indicate the presence of lead objects and pigments during the early Bronze Age. In ancient Egyptian and Greco-Roman times metallic lead was produced as a by-product of silver mining. Extensive evidence of ancient mining and smelting exists in both the Orient and Mediterranean regions. Although generally thought of as a disease of the Industrial Revolution, lead poisoning has been documented for 6,000 years. As early as the 4th century BCE, Hippocrates accurately described the symptoms of lead poisoning. However, it was not until the 19th and 20th centuries that lead as an occupational health factor became a public issue. During Greco-Roman and Medieval times *sapa*, a sweet lead acetate syrup, was added to both wines and food. This resulted in widespread lead intoxication among the affluent and has been suggested as a probable reason for the fall of the Roman Empire. Fortified wines from Spain and Portugal, rum from the Colonies, and cider precipitated epidemics of lead poisoning. Since 1970 the National Institutes of Occupational Safety and Health have monitored environmental lead and significantly reduced lead exposure in air, water, and food.

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INTRODUCTION

One of the remarkable characteristics of lead is its low melting point (Table 1) which is approximately 328 to 347°C, depending on minor impurities in the ore. Thus, it must have been one of the first metals known to and used by man, and was probably isolated by the ancients soon after they discovered the use of fire. The most widely distributed lead ore is galena (PbS); however, lead

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²PRESIDENTIAL ADDRESS—Presented at the 96th Annual Meeting of The Ohio Academy of Science held at Malone College, Canton, Ohio on 25 April 1987. Dr. Milton A. Lessler, Professor Emeritus of The Ohio State University, College of Medicine, served as President of the Academy during 1986-1987. He received a B.S. in Botany from Cornell University in 1937 and an M.S. in Plant Physiology in 1938. He taught elementary and junior high school for several years as a substitute teacher, and then worked as a research technician for the New York State Health Department in Albany and Saratoga Springs in the early forties. During World War II he participated as a paratrooper in five invasions with the 82nd Airborne Division in Africa and Europe. He resumed his education at New York University in 1946 and received a Ph.D. in Cell Physiology in 1950. During his studies on the quantification of nuclear DNA at New York University he was a pre- and post-doctoral Fellow of the National Institutes of Health. He joined the faculty of The Ohio State University in 1951 as an Assistant Professor, became an Associate Professor in 1957 and a Professor of Physiology in 1963. He was a National Science Foundation Faculty Fellow at the University of Michigan in 1958-1959 and a Visiting Professor at Northern Michigan University in 1967. Dr. Lessler became a member of The Ohio Academy of Science in 1963, was elected as a Fellow in 1966 and became a Life Member in 1982. He served as Editor of *The Ohio Journal of Science* from 1974-1981, and was a member of the Executive Committee of the Academy during that period. Dr. Lessler is a member of Sigma XI, a Fellow of the New York Academy of Science and the AAAS, and is a member of several Scientific Societies. His research in the areas of nuclear histochemistry, the biological effects of radiation, and the effect of lead poisoning on cellular metabolism resulted in the publication of more than 100 papers in national and international journals. He contributed chapters to books on histochemistry, the biological effects of radiation, and the use of oxygen electrodes for biological and biochemical analyses. He served as a Visiting Scientist for the American Physiological Society from 1962-1966, the FASEB program for minority institutions from 1982-1984, and was a consultant on the use of oxygen electrodes with the Yellow Springs Instrument Company for many years.

TABLE 1

Metals of antiquity arranged by melting point (MP).

Element	Latin name	Symbol	MP (°C)
Tin	stannum	Sn	232
Lead	plumbum	Pb	328
Zinc	zincum	Zn	420
Silver	argentum	Ag	961
Gold	aurum	Au	1063
Copper	cuprum	Cu	1083
Iron	ferrum	Fe	1535

also occurs in many ores as an oxide or carbonate and is widely distributed throughout the world wherever copper, gold, silver, and zinc occur. There are major lead-ore deposits in Africa, Asia, the Mediterranean regions, Spain, Southern England, the Americas, and the Orient (Nriagu 1983a).

LEAD MINING AND USE IN ANTIQUITY

The use of lead probably preceded the use of copper and bronze. This is based on archeological discoveries, dated approximately 7000 BCE on the use of cast lead objects and lead pigments. The presence of lead objects as well as cast copper artifacts during the Bronze Age indicated that ancient man had discovered the smelting process and was rendering and purifying metals to make useful and decorative objects (Nriagu 1983a). It can be seen from Table 1 that of the seven metals of antiquity only tin has a melting point lower than that of lead. Tin, although known to the ancients, was sometimes confused with lead (Pliny). Not as widely available as lead and copper, tin was not chemically isolated and described until the 18th century. The use of the other six metals of antiquity date back to very early archeological records of civilized man.

In ancient times, metallic lead was probably produced as a by-product of silver mining and smelting. Studies of metallurgy, as practiced 5,000 to 7,000 years ago, suggest that lead sulfide ores which contained silver sulfide were melted into an alloy with a reasonably hot fire. This lead-silver alloy was reoxidized by a secondary process

called cupellation that separated the lead from the silver, which was then used for ornaments, coinage and other purposes (Aronson 1983). Among the oldest metallic lead objects discovered in archeological digs is a string of decorative lead beads found in a tomb in Anatolia that was dated at approximately 6000 BCE. It is well known that the Pre-Greeks on the Aegean Islands had an extensive trade in lead and silver and used these metals for a variety of purposes (Nriagu 1983a). This is particularly well documented on Crete where the palaces of Knossos (3000-2000 BCE) contained artifacts indicating the use of lead pigments, lead and bronze castings for doors, and "T" forms to fasten building stones together. Early Egyptian tombs also yield ample evidence of advanced metallurgy and the use of lead pigments for glazes, paints, and cosmetics. The metals of antiquity are mentioned in various books of the Old Testament and in many of the early writings from Phoenician times onward (Nriagu 1983a). There is also evidence (as yet not well documented) that the mining and use of lead and silver occurred in the Orient as early or earlier than in the Mediterranean regions.

LEAD POISONING AS A DISEASE

Obviously, this early mining, smelting, and fabrication with lead must have resulted in environmental contamination and lead poisoning. Indeed, the lead mines in Cartagena of southern Spain, which were exploited by Carthage and later by the Romans, left a legacy of over 10,000,000 tons of slag and evidence of many deaths among the 40,000 men who worked the mines. Among the earliest records, whether on papyrus, copper, parchment or cuneiform writing on clay, there are notes that lead miners and individuals who worked with lead developed ailments that resulted in their early demise. This was first well documented by the Egyptians who used slaves in their mines and later by the Pre-Greeks, Greeks, and Romans (Nriagu 1983a). The largest known silver mines in Greece, the infamous Laurion Mines of classical Greece (near present-day Ergastiri), generated great material wealth for Athens and were known as an environmental problem as early as 2500 to 2000 BCE. I have visited these mining shafts in southern Attica, which still exist as mute evidence of the many slaves and condemned criminals who worked these mines and died there.

Hippocrates (460-377 BCE) described the symptoms of lead poisoning as appetite loss, colic, pallor, weight loss, fatigue, irritability, and nervous spasms. These are the same symptoms that are observed and categorized for lead poisoning today. It also was observed that cows and horses could not be pastured near the mines, or they would soon become sick and die. Recent research by Zmudski et al. (1983) indicated that the fatal dose of lead for farm animals is much lower than that for humans. Cattle fed 6-8 mg Pb per Kg per day died in 1 to 2 weeks. Horses fed as little as 3-4 mg Pb per Kg per day died in a few weeks. Humans can tolerate up to 20-30 mg Pb per Kg per day, whereas rodents can tolerate up to 250 mg per Kg per day. Landrigan et al. (1975) studied a group of 919 children living near a lead smelter in Shoshone, Idaho. They noted that 42% of the children had elevated blood lead, and that 99% of the children

living within 1.6 km of the smelter had toxic levels of lead in their blood.

Although generally thought of as a disease of the Industrial Revolution, the symptoms of lead poisoning were known, described, and documented for at least 5,000 years prior to that period. The Middle Ages saw a marked increase in the use of lead and lead-containing products, and there were occasional publications on the toxic dangers involved in the preparation and use of various metals. For example, U. Ellenberg in 1473 published a tract titled "On the Poisonous and Noxious Vapors and Fumes of Metals;" later G. Agricola (1556) published "De Re Metallica." It was not until the late 19th and early 20th centuries that occupational health was recognized as an important governmental public health issue. It was during this period that the United States and several European countries (Britain, France, Germany) passed occasional legislation designed to protect industrial workers from potentially dangerous toxic environments. As the evidence for lead and other types of industrial poisoning accumulated, the need for the protection of the public became more evident (Woolley 1984). The United States Congress, recognizing the severity of the problem, passed the Occupational Health Act in 1970. This legislation created the National Institutes of Occupational Safety and Health (NIOSH) and was soon followed by legislation for the regulation and clean-up of toxic wastes. Limits for the acceptable levels of lead in air, water, and food were set by NIOSH. These levels have been revised downward as new evidence became available on the vulnerability of infants and developing children to relatively low levels of lead in the environment (Johnson and Mason 1984).

During the Middle Ages, there was a marked increase in the use of lead, but the Industrial Revolution saw a tremendous expansion in the fabrication of metals and in the number of industries using lead and lead products (Table 2). It was not until the 18th century that B. Ramizzini (an Italian physician) linked the diseases shared by potters, guilders, glass makers, and metal workers with lead poisoning. The symptoms of lead poisoning were often given quaint local or industrial names (Table 3). Development of concern for occupational and environmental health, although noted in ancient times,

TABLE 2

Partial list of industries disseminating environmental lead.

Industry	Products
Electrical	Storage batteries, solder
Fermented beverages	Fortified wines, moonshine
Glass	Leaded glass and art objects
Glazing	Litharge, caulk
Metal reclamation	Gold, silver, lead, iron, etc.
Metal tubing	Pipes and solder
Mining	Lead release from ores
Munitions	Guns and ammunition
Oil	Leaded gasoline, tetraethyl lead
Paint	Red and white lead pigments
Patent medicines	Herbal medicines
Pottery	Lead glazes and tableware
Printing	Leaded types and inks
Ship and bridge repair	Flakes of leaded paint
Silverware	Silver and pewter objects
Smelting	Atmospheric lead and slag

TABLE 3
Some local and special names for lead poisoning.

Name	Period	Area	Source of lead
Colic Pitonus	Middle Ages	Europe	Sapa in wine and food
Colic of Poitou	Middle Ages	France	Adulterated wine
Derbyshire belain	Middle Ages	England	Hard cider
Dry gripes	Colonial	Americas	Distilled rum
Entrabado	Middle Ages	Spain	Fortified wine
Huttenkatze	Middle Ages	Germany	Adulterated wine
London gout	After 1703	England	Port and sherry
Miner's colic	Ancient to modern	Worldwide	Mineral ores and slag
Moonshiner's colic	Recent	Southern U.S.	Illegal whiskey
Painter's pallor	Recent	Worldwide	Paints and caulks
Potter's disease	Industrial	Worldwide	Clays and glazes
Saturnism	Ancient	Greco-Roman	Sapa in food and wine

was not important as long as slaves and cheap labor were available. As the evidence accumulated through the years, many areas adapted some type of legislation for the protection of workers and the population.

ADULTERATION OF WINE

During the Middle Ages, the writings of medieval physicians indicated an awareness of both the sources and symptoms of lead poisoning. Recognizing the dangers of fortified wines, both the French and Spanish authorities issued edicts in 1427 prohibiting the addition of sapa to sweeten wine. Indeed, in 1478, German authorities made wine adulteration a crime punishable by death. Sapa, which had been in use since very early Greco-Roman times, was prepared by boiling acidic wine in lead-lined vessels. This yields a sweet syrup due to the formation of lead acetate $[\text{Pb}(\text{C}_2\text{H}_3\text{O}_2)_2]$. The addition of sapa not only sweetens the wine, but also inhibits spoilage by interfering with enzyme activity. Most early Greek and Roman wines contained sapa which also was used to sweeten food because these civilizations had no readily available source of sugar. Recent analyses of ancient Greek and Roman wine vessels indicated that wine stored in them had a considerable lead content. In 1883, K. B. Hofmann (a German physician) followed Roman recipes for making sapa in lead-lined pots and obtained a syrup of wine with approximately 240 mg Pb per L. Additions of 2 teaspoons of this sapa to a liter of wine yielded 20-30 mg Pb per L. He found that with a longer boiling time sweet sapa with 1000 mg Pb per L could be prepared with little additional effort.

LEAD POISONING AND THE FALL OF ROME

In 1965, Gilfillan published a paper titled "Lead Poisoning and The Fall of Rome," which has been discussed widely. He suggested that the widespread behavioral and neurological deficiencies of the Roman Emperors were due to lead intoxication. It is well documented that the aristocracy and patricians made extensive use of wine and food sweetened by sapa, of lead plumbing, and of lead-lined storage vessels. These are things that the plebians and slaves could not afford. In fact, recent studies of presumed patrician and slave skeletons show a much higher bone lead content among the patricians (Mackie et al. 1975). Analyses of the residue in ancient Roman wine vessels gave estimated lead contents of approximately 750 mg Pb per L of wine. Gilfillan's contention is controversial as historians, militarists, and economists have

cited other factors as being more important than lead poisoning in the fall of Rome. These objections to Gilfillan's thesis certainly do not negate the ever increasing evidence that there was an epidemic of gout and rampant lead intoxication among the patricians of ancient Rome, and that this may have been a factor in the fall of the Roman Empire (Nriagu 1983b).

LEAD IN RUM AND CIDER

England signed the treaty of Methuen in 1703. This made it possible to import "fortified wines" (port and sherry) from Spain and Portugal. As a result of this trade, there was widespread lead poisoning and a great gout epidemic among the upper classes in England, who imbibed liberally of these beverages (Green 1985). Recent atomic absorption analyses of English port bottled during the period 1770 to 1830 showed lead contents of up to 1900 mg Pb per L (Nriagu 1983a). Wine was not the only culprit, however. Sir Geroge Baker (1767) studied an illness called Devonshire Colic and found that this was lead poisoning, "due to the use of lead pounds and presses to make apple cider." James Hardy later reinvestigated and confirmed Baker's work, and noted experimentally that cider allowed to stand in glazed earthenware pots (to make hard cider) was rapidly impregnated with lead (Waldron 1969).

Not only fortified wine and hard cider resulted in lead poisoning. During the Colonial Period, "the demon rum" became an important source of lead intoxication. Ben Franklin (then a Philadelphia printer) published a monograph by Thomas Cadwalader in 1745 titled "Essay on West India Dry Gripes." This essay by the Philadelphia physician clearly described the enteric and neuropathic sequelae of drinking lead-laced rum and other spirits (Aronson 1983). At this time rum from the Caribbean Island of Barbados sold for 6 shillings per gal. When later made in other British Colonies, rum sold for as little as 1 shilling per gal. Imported into England, where it was sometimes called "Kill-devil," rum became a standard ration for sailors in the British navy. The name rum probably originated from the Devonshire term "rumbullion" (indicating tumult) which was then shortened to "rumbo" and then to rum (Felton 1965).

On 3 September 1723, the Massachusetts Bay Colony passed an act prohibiting the distillation of rum or other strong liquor with leaden heads (covers) or worms (pipes). This was the result of extensive sickness (lead poisoning)

caused by the imbibing of distilled rum. Chapter II of the Massachusetts Bay Colony Act of 1723, passed under the charter granted by their Majesties King William and Queen Mary to the inhabitants of the Province of Massachusetts-Bay in New England, reads as follows:

"An Act for preventing Abuses in distilling of Rum and other strong Liquor with Leaden Heads or Pipes.

Whereas the strong Liquors and Spirits that are distilled thro' leaden Heads or Pipes, are judged on good Grounds to be unwholesome and hurtful; notwithstanding which some Persons to save Charge may be led into making or using such Heads, Worms, or Pipes:

For Remedy and Prevention Whereof:

Be it enacted by the Lieutenant Governer, Council and Representatives in General Court assembled, and by the same, That no Person whatsoever, shall make Use of any such Leaden Heads or Worms for the future; and that whosoever shall presume to distill or draw off any Spirits or strong Liquors through such Leaden Heads or Worms, upon legal Conviction thereof before any of his Majestie's Courts of Record, shall forfeit and pay a sum of *One Hundred Pounds*.

And Be It Further Enacted By the Authority aforesaid, That no Brazier, Pewterer, or other Artificer whatsoever, shall presume to make any Worm or Head for distilling of coarse and base pewter, or such as hath any Mixture of Lead in it; under the Penalty of *One Hundred Pounds*."

During the Colonial Period, in addition to the trade in rum, there was extensive manufacture and use of glazed earthenware, pewter, lead pipe, lead shot, and lead type for printing. Red and white lead were widely used as pigments for paints (also as an addition to red pepper); lead acetate and lead oxide were used to sweeten and whiten bread. Litharge (a PbO product) was used as a putty to install windows and, according to colonial records, was sometimes added to snuff. Apparently, lead intoxication was rampant during the Colonial Period in America and may have been involved in accusations of witchcraft because individuals with lead poisoning neuropathy often show weird behavior. Recently, Aufderheide et al. (1985) analyzed the exhumed skeletal remains of four Colonial Virginia populations and found that bones from graves of black individuals averaged 35 ppm of lead (8-96 ppm), whereas white families from the same plantations had a mean of 185 ppm (125-250 ppm). This indicates a much lower exposure to lead in the food and water consumed by blacks, as compared to the higher lead content of the food, wines and rum available to the landowners.

ENVIRONMENTAL LEAD

Evidence of lead poisoning became increasingly common in industrialized countries during the 20th century, when world-wide studies indicated that the human population needed protection. Industrial engineers became

interested in the problem and used systems analysis techniques to study the movement of lead from the workplace, into the environment, and to the population (Fig. 1). These studies along with monitoring of lead in air, water, and food in the every-day environment have allowed us to follow the path of lead to humans and to devise ways to reduce significantly our exposure to lead and other toxicants. The reduction of lead in gasoline, controlling the use of lead pigments in paints and printing inks, and banning of lead-based glazes on pottery and ceramic ware (Wallace et al. 1985) have resulted in a reduction of both industrial and population exposure to lead. There is no way that an important metal like lead can be removed from the environment, but with increased vigilance and control we can markedly reduce the exposure of animals and humans to lead. Industrial management of toxic wastes from plants using lead and lead products (e.g., the assembly and disposal of automobile batteries) can have a marked effect on the release of lead into the environment.

There always will be sources of environmental lead release, because wherever mining occurs the debris often contains lead that can be leached out by rain water. The burning of coal releases lead into the air; the area around metal smelters (as noted earlier) shows lead contamination for several miles downwind. Even if we control all of these factors, individuals will still find ways to expose themselves to lead. For example, in Birmingham, Alabama the majority of patients admitted to the Veteran's Administration Hospital with anemia, gout, or severe colic are suffering from lead poisoning because of the consumption of "moonshine" distilled with lead pipes or containers. This type of lead poisoning is common also in rural southern Ohio, Kentucky, and Tennessee. Studies of "moonshine" obtained from these areas indicate lead levels as high as 1,000 to 10,000 mg Pb per L (Batuman et al. 1981).

NIOSH AND CDC MONITORING

Extensive animal experimentation and detailed studies of adults and children suffering from lead poisoning have enabled NIOSH and the Center for Disease Control (CDC) to establish the following toxic limits for blood lead content in humans (Johnson and Mason, 1984):

- <20 μgPb per 100 mL — little or no effect in adults but may represent a danger to embryos, newborns, or infants;
- >40 μgPb per 100 mL — overexposure and danger for both children and most adults; and
- >80 μgPb per 100 mL — frank lead poisoning; patients need immediate treatment and may require hospitalization.

These limits have since been revised downward for infants and developing children (CDC 1985).

Since industrial and urban lead screening programs have been in place (about 16 years), surveys by the CDC have shown a significant drop in both environmental lead (approximately 37%) and the incidence of lead poisoning. This is due in part to renewed vigilance in the disposal of toxic wastes, removal of lead from paints and ceramic glazes, the decreased use of leaded gasoline, controlling the use of lead-arsenate for insect and mold control in orchards and vineyards, and better control of

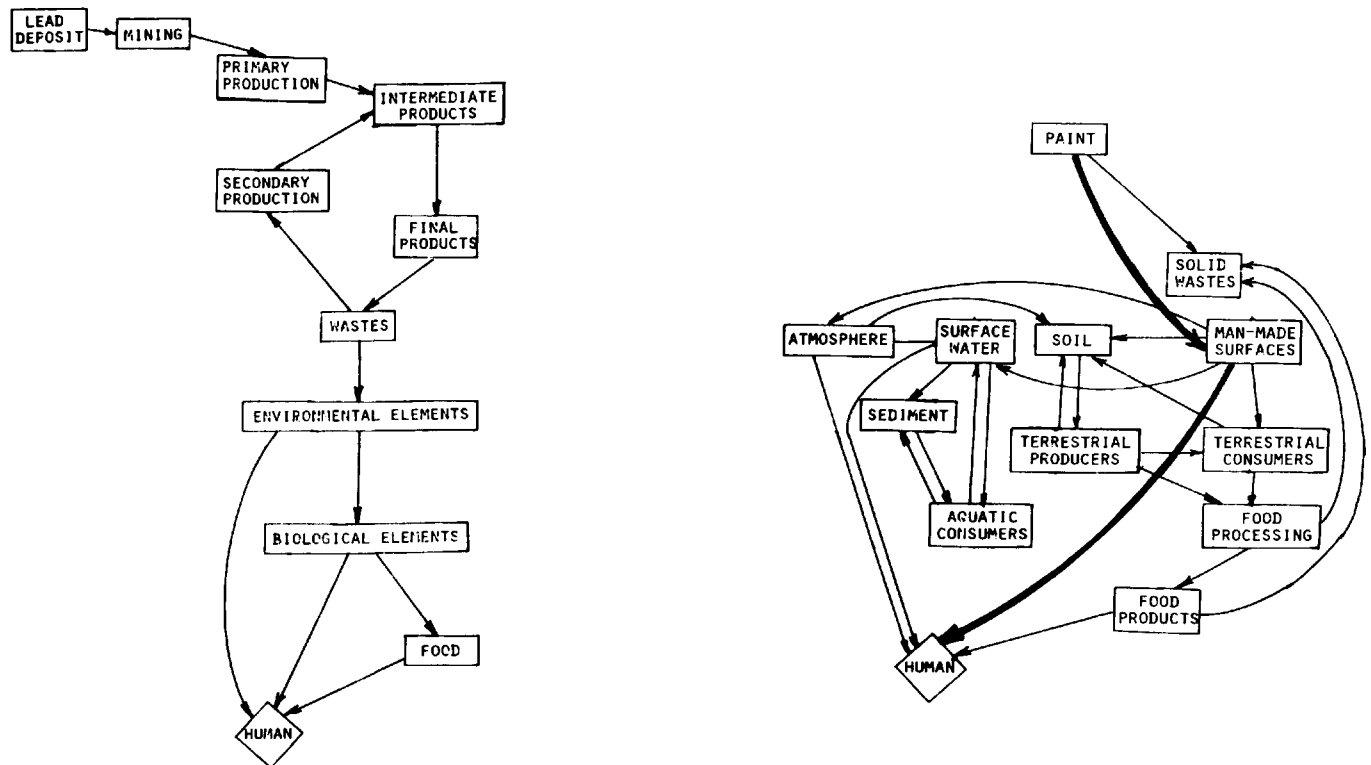


FIGURE 1. Systems analysis diagrams of the movement of lead into the environment via lead mining and production (left) or paint (right), (Courtesy of Heavy Metals Task Force, Institute for Environmental Studies, Univ. of Illinois at Urbana-Champaign.)

industrial emissions and wastes. A ban on the manufacture of lead-based products and paints for home use, childrens' furniture, and toys has reduced markedly the exposure of children to lead in the home. It should be remembered, however, that lead is essentially a non-degradable metal, and that most of the anthropogenic accumulated lead will continue to remain in the environment for an extended period of time.

LEAD AND DEVELOPMENT

When lead is absorbed, it is distributed first to the soft tissues such as the liver and kidneys. In adults, approximately 80 to 90% of ingested lead is excreted; the lead that remains may be stored in bone where it does little harm. Apparently, the bone-forming cells can arrange for an interchange of calcium and lead (both divalent ions) and thus for the storage of lead in a bound form for long periods of time. Studies of house painters, who have used lead based paints, indicate that bone-stored lead can be released into the blood stream during illness or prolonged acidosis resulting in severe lead poisoning. Both infants and adults have the ability to store lead in bone in an insoluble form, but the more active absorption and small bone mass of infants and children allow them to store only small amounts of lead as compared to adults. Infants and children exposed to toxic levels of lead during their early years show a marked reduction in growth and development. If the exposure is for a prolonged period, they may have peripheral neurological, central nervous system, and kidney damage (Chisolm et al. 1975, Lin-Fu 1972).

Lead intoxication inhibits the development of red cells in the bone marrow and markedly reduces the synthesis

of hemoglobin by developing red blood cells, resulting in an anemia (Fig. 2). When children become anemic, it stunts their body growth and the normal development of the nervous system. Experiments with young rats or chicks in which lead was mixed with their food showed significant retardation in growth and development (Figs. 3 and 4). The retardation is greatest if the lead intoxication occurs during early periods of growth, and is not as evident after the major growth phase has been completed. The risk of lead poisoning may not be as great for adults as for children, but continued exposure to lead can result in serious consequences. Thus, continued vigi-

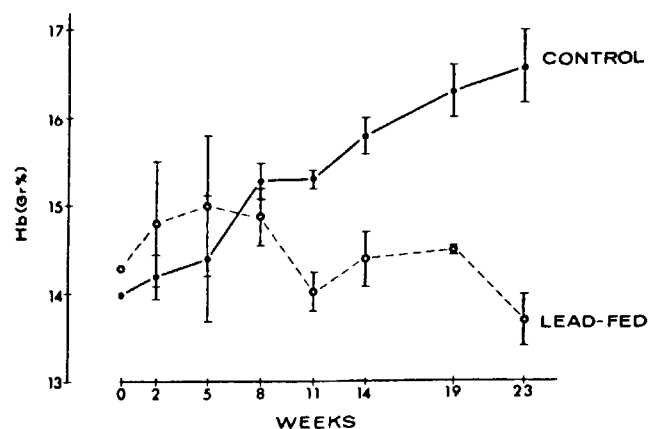


FIGURE 2. Changes in the percent of hemoglobin (Hb) in the blood of Sprague-Dawley rats fed a normal diet or one with 1% lead acetate added. Note that the animals were in reasonably good Hb balance for the first 8 weeks of the experiment before they became anemic. The bars indicate the standard errors of the means (Cardona 1970).

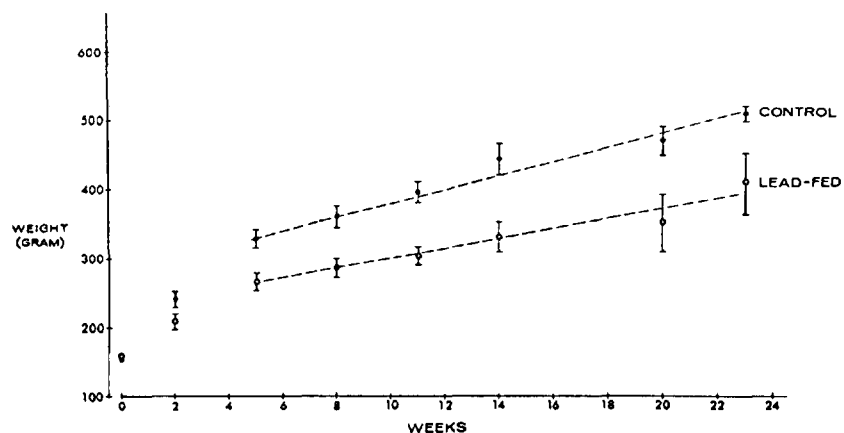


FIGURE 3. Body weight changes of Sprague-Dawley rats weighing 160 ± 10 g at the start of a 24-week growth period. The controls were fed Purina Lab Chow *ad libitum*; the lead-fed animals were fed the same diet with 1% lead acetate mixed in the food. The dashed lines are linear regressions based on the mean weights (10 animals) obtained weekly from weeks 5 through 24 of the experiment. The bars indicate the standard deviations of the means (Lessler and Wright 1976).

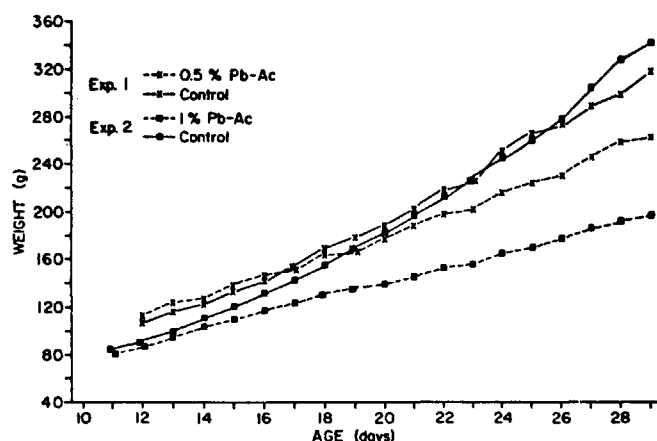


FIGURE 4. Body weight changes of white leghorn cockerels with either 0.5% or 1% lead acetate added to their diet of Purina Startina Mash compared to chicks not exposed to lead. Mean weights are based on 20 chicks per group. Note that growth inhibition started earlier and was more profound at the higher lead level (Ray 1981).

lance for reduction of lead in the environment is an important goal for all populations.

To end on a positive note, it is evident that world-wide environmental exposure to lead has been reduced by new laws and constant environmental surveillance. This is particularly true in the United States, many European countries, and Japan which have instituted national programs for control of the use and disposal of lead and lead products. This is not true of Third World countries where monitoring of air, water, and toxic waste contamination is at a minimum. Thus, occasionally we hear of serious epidemics of lead poisoning in countries where mining, smelting, and use of lead products are unregulated. The cost of maintaining uncontaminated air, water, and food worldwide is constant vigilance in order to maintain adequate control of the penetrance of toxic substances into the environment.

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