The Health Effects of Lead on the Human Body

Studies indicate that lead adversely impacts the neurological system, reproductive system, cardiovascular system, and kidneys of lead-poisoned individuals

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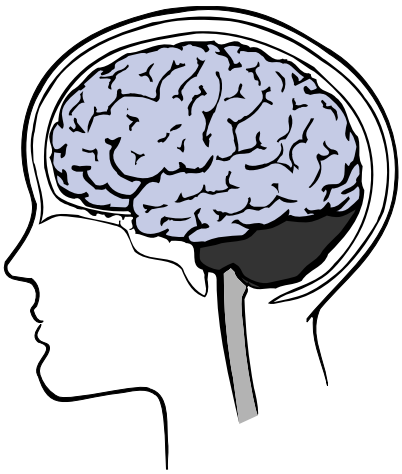
Lead is a toxic substance that attacks many different body organs and systems. Unlike other metals such as zinc or iron, lead has no beneficial effects on the body. Lead is a ubiquitous environmental contaminant, largely due to the wide variety of past and present commercial uses. It is one of the best-studied toxic substances. Lead poisoning is considered by the Centers for Disease Control and Prevention (CDC) to be the foremost environmental childhood disease today (National Academy of Sciences 1993; CDC 1991a; ATSDR 1988a).

Effects on the Neurological System

Of greatest concern is lead's effect on neurological development and behavior. Although the precise mechanism of action is not yet fully understood, it appears that lead interrupts and inhibits neural differentiation, pathway development, and learning abilities. Because learning acuity is high in the early years, interruptions in normal development in young children produce lifelong decrements in intelligence, suggesting that some effects of childhood lead poisoning are irreversible.

A recent review of a number of prospective, cross-sectional, and retrospective epidemiological studies showed statistically significant inverse relationships between blood lead levels and IQ or other measures of cognitive development. At population averages, it appears that there is a decline of 2-4 IQ points for each increase in blood lead level of 10 µg/dL, although some studies suggest that the decline may be as high as 8 IQ points/10 µg/dL (Schwartz 1994; Bellinger 1987; Bellinger 1992; Davis 1993). While this is at first glance a rather small effect, it is much larger at the ends of the population distribution. In other words, for children with IQs below 80 or above 120 (i.e., those who are retarded or gifted), the effects are extensive.



The organ of paramount concern in cases of childhood lead poisoning is the developing brain, where IQ declines of 2-4 points for every increase in blood lead levels of 10 µg/dL have been observed.

The Agency for Toxic Substances and Disease Registry (ATSDR) estimates that lead exposure doubles the number of children with low IQs, increasing the need for special education and other expensive compensatory programs (ATSDR 1988a). Similarly, the sharp decline in the number of lead-poisoned children with IQs above 120 means that lead poisoning can be associated with an absence of exemplary students and leaders in at-risk populations.

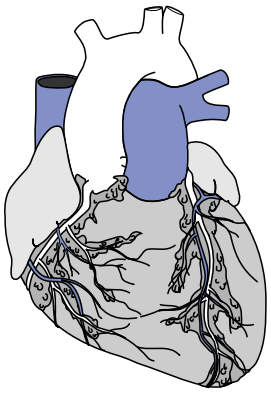
For lead-poisoned children with blood lead levels of 30 µg/dL, the predicted loss would be anywhere from 6 to 24 IQ points. Importantly, all of these studies, now numbering about a dozen, show declines in intelligence, regardless of whether exposures were high or low (Yule 1984; Bellinger 1987; Fulton 1987; Landrigan 1985; McMichael 1988; National Academy of Sciences 1993). While a few studies did not reach statistical significance, it is noteworthy that all the studies showed declines in intelligence (Davis 1993); there have not been any studies suggesting that there was an increase in intelligence as a result of lead exposure. If lead truly had no effect (or only a very small one), one would expect to see the results of these studies to be distributed in either direction due to random chance alone.

While intelligence is a difficult characteristic to measure reliably, there are similar findings for cognitive function, language and reading skills, Mental Development Index scores, school progress, and visual-spatial and visual motor skills (eye-hand coordination) (National Academy of Sciences 1993).

Unlike lead sources such as food or gasoline, which are typically associated with moderately increased blood lead levels, ingestion of lead-based paint can produce greatly elevated blood lead concentrations among young children. If blood lead concentration exceeds 30 µg/dL, the loss of intelligence becomes more severe and is accompanied by additional neurological prob-

lems. Long-term neurological outcomes among clinically poisoned children include retardation and severe behavioral disorders, seizures, cerebral edema, structural derangement in capillaries, and neuronal necrosis (Pentschew 1965; Perstein and Attala 1966).

Lead concentrations in teeth are thought to be a better measure of lead exposure than is blood lead level. Dentin lead levels have been associated with decreased reading ability and a greater likelihood of failing to graduate from high school (Needleman 1979). Another tooth lead study asked teachers to rate a series of performance measures including degree of organization, daydreaming, ease of distraction, persistence, impulsiveness, reading, hyperactivity, and other similar measures of behavior. For each of these behaviors, there was a clear dose-response relationship between tooth lead and adverse behavior (Needleman 1990). Studies have linked lead exposure to anti-social behavior, juvenile delinquency, and adult criminality (Thomson 1989; Fergusson 1993; Satterfield 1987; Denno 1990; Needleman 1996). Of course, many of these neurological and behavioral effects could be explained by other environmental or genetic factors or lifestyle variables such as nutritional status, parental involvement, or socioeconomic status. While each of the studies cited above used carefully constructed control groups that were alike in most respects except for lead exposure, confounding influences are a significant problem in human studies. Such factors are not present in animal studies, which have shown a clear association between lead intake and decreased size of neurons and dendrites, decreased complexity of dendritic processes, delayed synaptogenesis in the cerebral cortex, retarded development of neonatal cerebellar neurons, necrosis of mitotically active precursor cells, and reduced production of myelin (all reviewed in National Academy of Sciences, 1993).



It has been estimated that a reduction in blood lead levels of 50% could lead to 20,000 fewer heart attacks and 100,000 fewer cases of heart disease per year.

Animal studies have also helped reveal plausible mechanisms of the link between adverse behavior and lead exposure. For example, infant rhesus monkeys given low doses of lead showed disruptions in social behavior (Laughlin 1991). Lead is known to interfere with neurochemical inhibition in rodents (Taylor 1978), which could in turn lead to rapid uncontrolled responses to stimuli, which in turn could be seen as impulsiveness or aggression.

At higher exposures, encephalopathy, cerebral edema, neuronal necrosis, severe retardation, severe behavior disorders, and death can occur (Byers and Lord 1943; Perlstein and Attala 1966; Rummo 1979). These effects are now relatively rare events, although 3-5 deaths from lead poisoning still occur each year (Matte 1992).

Effects on the Reproductive System

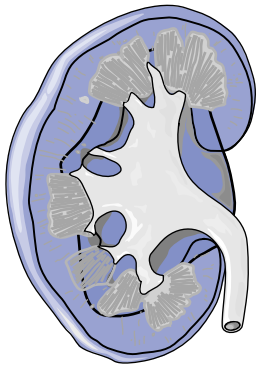
In the developing fetus, lead has been associated with decreased size of neurons and dendrites, decreased complexity of dendritic processes, delayed synaptogenesis in the cerebral cortex, necrosis of mitotically active precursor cells, and reduced accumulation of myelin. In the female reproductive system, lead has been associated with decreased fertility, increased rates of miscarriage and stillbirth, premature rupture of membranes, pre-term delivery, and decreased birthweight. Reproductive effects are not limited to women, however. In men, decreased libido, premature ejaculation, erectile dysfunction, decreased number of sperm, abnormal sperm shape and size, and reduced semen volume have all been shown to be caused by lead exposure (ATSDR 1988b; National Academy of Sciences 1993).

Effects on the Cardiovascular System

Lead exposures also are associated with small increases in blood pressure and left ventricular hypertrophy, which has important implications for heart disease. If population blood lead levels were cut in half, 20,000 fewer heart attacks per year and 100,000 fewer cases of heart disease per year could be expected (Schwartz 1991).

Other Effects

Kidney injuries can also be caused by lead exposure, causing reduced ability to reabsorb nutrients, increased blood levels of waste chemicals produced by cells, and scarring of the kidney (ATSDR 1988a). Decreased stature and reduced hearing acuity have also been observed (ATSDR 1988b).



Lead has also been shown to interfere with the kidney's normal functions of filtering out waste products from the blood and reabsorbing essential nutrients.

Relationship of Lead Distribution Within the Body and Long-Term Health Outcomes

A significant amount of lead that enters the body is stored in the bone for many years, which, together with the neurological effects described above, can also be considered yet another type of irreversible health effect. An equilibrium exists between relatively large bone lead stores on the one hand and the soft tissues and the blood stream on the other, where lead turnover levels are more rapid. Certain events such as immobilization, wasting illness, osteoporosis, and pregnancy can result in more rapid mobilization of bone stores into the bloodstream, where it again becomes available to the brain and other organs, exerting its harmful effects (ATSDR 1998b; National Academy of Sciences 1993).

Symptoms of Lead Poisoning

Effects on the nervous system are also evident in the symptoms associated with lead

poisoning. Common presenting complaints at higher exposure levels include vomiting, crampy abdominal pain, pain in the muscles and/or joints, paranoia, depression, and aggressive behavior. More obscure symptoms include malaise, fatigue, headache, irritability, anorexia, and diarrhea or constipation. Diagnosis of elevated blood lead levels in both children and adults is rarely completed by analysis of symptoms alone due to low specificity and sensitivity, necessitating a blood lead test. Most lead-poisoned children do not exhibit any obvious symptoms and most cases go undiagnosed (CDC 1991a).

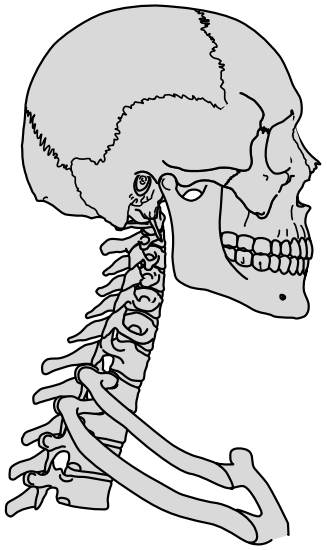
The classic symptom of high lead exposure in adults is focal palsy and peripheral neuropathy (weakness in the extremities). Wrist drop and foot drop are the common presenting signs. A lead-poisoned person often cannot keep the hands extended fully for even a short period of time (Matte 1992).

Margin of Safety and Prevalence Rates

Perhaps the most telling feature of lead toxicity and exposure is the comparatively small range between "normal" exposures to lead, its fatal dose, and the large populations exposed. In young children, fatal blood lead levels are in the range of 100-150 µg/dL, depending on the child's nutritional status, age and other factors (ATSDR 1988b). The current level of concern established by the Centers for Disease Control and Prevention (CDC) and the American Academy of Pediatrics is 10 µg/dL, which is only an order of magnitude below the fatal dose.

The most recent Health and Nutrition Examination Survey (NHANES III) showed that average blood levels in children aged 1-5 years of age are currently 2.8 µg/dL. Overall, 1.7 million young children have blood lead levels of 10 µg/dL or greater (Brody 1994; Pirkle 1994). This represents 8.9 percent of all children between the ages of one and five years. Blood lead levels in inner city neighborhoods are substantially higher. For example, 37% of young African-American urban children have blood lead levels greater than 10 µg/dL. While prevalence rates appear to be elevated among urban, low-income minority populations, there is also evidence that prevalence rates in rural areas are higher than originally thought and may be on the order of 20% (Norman 1994).

As a result of widespread lead exposure and the absence of clear symptomology associated with the disease, both CDC and the American Academy of Pediatrics now recommend that virtually all children between the ages of 1 through 6 years have their blood tested for lead (CDC 1991b; American Academy of Pediatrics 1993), unless there are data in a given geographic area showing that a more targeted screening approach is appropriate.



The tendency for lead to be stored in bone tissue often leads to chronic health effects, as stored lead can be released into the blood stream years after the initial lead poisoning event has occurred.

Prevalence rates are of course dependent upon the blood lead level of concern. Although the current level of concern of 10 µg/dL is low compared with earlier public health advisories (60 µg/dL was the level of concern for acute effects as recently as the 1970s), it is at least 300 times greater than the body burdens estimated for pre-industrial native populations in North America (Ericson 1991; Patterson 1991). There is also evidence that adverse health effects occur at blood lead levels below 10 µg/dL (CDC 1991a). Although the level of concern may decline in the future as further research is conducted, it appears to be a reasonable benchmark on which to base policy responses at this time.

In short, the margin of safety is far less for lead than for many other environmental toxicants. Death from lead poisoning has now become a relatively rare event due to improved medical management and targeted screening campaigns. From 1979 to 1988 there were 139 lead-related deaths; most of these were among adults, and three were children who reportedly had lead-based paint exposure (Staes 1995). The widespread nature of lead exposure and its narrow margin of safety make it unique among the environmental toxicants.

References for this article located on p. 32.

David E. Jacobs was appointed by Secretary Henry Cisneros to serve as Director of the HUD Office of Lead Hazard Control (formally the Office of Lead-Based Paint Abatement and Poisoning Prevention) on July 18, 1995. As Director, Mr. Jacobs is responsible for establishing policies and programs to prevent childhood lead poisoning caused by exposure to lead-based paint hazards in housing. He coordinates and oversees the Office's grant programs to state and local governments, technical assistance and guidelines development, technical studies, regulations and standards development, and public education. He is responsible for coordinating activities with other Federal agencies such as the Environmental Protection Agency and the Centers for Disease Control. Mr. Jacobs also develops initiatives designed to promote public/private partnerships that control hazards in the millions of dwellings contaminated with lead-based paint. He has also served as Deputy Director of the National Center for Lead-Safe Housing and Director of the EPA Southern Lead-Based Paint Training Consortium at the Georgia Institute of Technology, Texas A&M University, and Louisiana State University. Mr. Jacobs was one of 40 experts appointed to the HUD Task Force on Lead Hazard Control and Financing by Secretary Cisneros and is the principal author of the HUD Guidelines for the Evaluation and Control of Lead-Based Paint Hazards in Housing.

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