

Lead—General Toxicity

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Prior to the industrial exploitation of lead, there was almost no human exposure to the metal. Investigations of skeletal remains indicate that the modern individual typically has 500-1000 times more lead burden than a person who was born in a pre-industrial era. The largest widespread source of lead in the environment is from the prior use of tetraethyl lead in internal combustion engines, residual lead paint pigments, leaded water pipes and from electronic solder. Hot spots of high lead contamination still occur where lead is mined, smelted or recycled. Despite efforts to reduce the widespread sources of lead, older urban areas still have lead contamination and much of the developing world lacks the controls to stop the use of lead in their manufacturing and consumer streams.¹

Adult exposure to lead is usually via workplace exposure and therefore acute. Most workplaces are aware of the exposure routes and how to mitigate them. Typical industries working with lead would be mining and smelting operations, battery and electronics manufacturing and the soldering of equipment.²

The easiest way lead can enter the body is by inhalation or ingestion of dust containing lead or lead compounds. Shortly after ingestion, the lead is distributed in soft tissues and organs, but longer-term storage is in the bones and teeth. For a typical adult, about 94% of the body burden of lead is stored in this manner, but for children it may only be 73%. Lead can be stored in the body for decades, hence the long-term ramifications of lead exposure in childhood.

In adults, the primary effects of lead toxicity are in the peripheral nervous system, while in children lead primarily affects the central nervous system.³ Encephalopathy may present with symptoms of dullness, irritability, poor attention span, headache, muscular tremor, loss of memory or hallucinations. More extreme exposure may result in delirium, lack of coordination, convulsions, paralysis, coma and ataxia.⁴ Children may be inattentive, hyperactive and irritable at low lead levels. Greater lead levels may exhibit delayed growth, decreased intelligence, short-term memory and hearing loss. At higher levels, lead can cause permanent brain damage and even death.⁵

Lead exposure can also cause anemia by inhibiting key enzymes involved in the

heme synthesis pathway and reducing the life span of circulating erythrocytes by weakening the cell membranes.⁶ High levels of lead can cause renal dysfunction but damage has been reported at lower levels as well.⁷ Lead can affect reproductive health, causing abnormal spermatogenesis in men and infertility and miscarriage in women.⁸ The primary storage site for lead in the body is in the bones. In adults, 85-95% of blood lead is found in bone, but only 70% for children, leaving children with a higher lead concentration in soft tissue.

Lead is bivalent, like calcium, magnesium and iron, and can therefore substitute in many fundamental biochemical processes.⁹ Lead is very effective at inactivating antioxidant enzymes such as glutathione, superoxide dismutase and catalase, which prevent lipid peroxidation and hemolysis. Also, lead is believed to hinder the proper development of the blood-brain barrier and neurotransmitters such as protein kinase C.

The best defense against lead poisoning is prevention from exposure and monitoring on a regular basis to check for exposure from non-obvious sources.

For more information on lead testing, contact your laboratory.

??? Did You Know ???

Individuals, families, and communities that have experienced social and economic disadvantages are more likely to face greater obstacles to overall health. Characteristics such as race or ethnicity, religion, low socioeconomic status, gender, age, mental health, disability, sexual orientation or gender identity, geographic location, or other characteristics historically linked to exclusion or discrimination are known to influence health status.

SAMHSA is committed to addressing these health disparities by providing culturally and linguistically appropriate prevention, treatment, and recovery support programs.

SOURCE: SAMHSA

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Question of the Month

Question: *How long will urine stay "fresh" for drug-testing purposes? What if I need a confirmation test?*

Answer: At point of collection, a urine sample contains the drugs that have been consumed within the detection period for each particular drug (i.e. Opiates: 2-4 days). Drugs are stable in urine for at least 30 days at room temperature except Alcohol, which can evaporate from urine just like it evaporates from skin, sweat, etc. However, if the sample is frozen Alcohol evaporation is eliminated. All other drugs remain stable in an unfrozen urine sample for 30 days so GC/MS or TLC Confirmatory testing can be performed during this time (GC/MS can detect even very small amounts of the drugs).

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References

1. IPEN. Global Lead Paint Elimination by 2020: A Test of the Effectiveness of the Strategic Approach to International Chemicals Management. Berkeley, CA:International POPs Elimination Network (2012).
2. World Health Organization, Lead Poisoning and Health Fact Sheet, September 2016 (<http://www.who.int/mediacentre/factsheets/fs379/en/>)
3. Brent JA. Review of: "Medical Toxicology" Clin Toxicol. 2006;44:355–355.
4. Flora SJS. Structural, chemical and biological aspects of antioxidants for strategies against metal and metalloid exposure. Oxid Med Cell Longev. 2009;2:191–206.
5. Cleveland LM, Minter ML, Cobb KA, Scott AA, German VF. Lead hazards for pregnant women and children: Part 1: immigrants and the poor shoulder most of the burden of lead exposure in this country. Part 1 of a two-part article details how exposure happens, whom it affects, and the harm it can do. Am J Nurs. 2008;108:40–49.
6. Guidotti TL, Ragain L. Protecting children from toxic exposure: three strategies. Pediatr Clin North Am. 2007;54:227–235.
7. Grant LD. Environmental Toxicants. John Wiley & Sons, Inc.; 2008. Lead and compounds; pp. 757–809.
8. Flora SJS, Pachauri V, Saxena G. Academic Press; 2011. Arsenic, cadmium and lead. Reproductive and Developmental Toxicology; pp. 415–438.
9. Lidsky TI, Schneider JS. Lead neurotoxicity in children: basic mechanisms and clinical correlates. Brain. 2003;126:5–19.