Lead

Lead is a gray-white, soft metal with a low melting point, a high resistance to corrosion, and poor electrical conducting capabilities. It is highly toxic. In addition to its highly concentrated ores, lead is naturally available in all environmental media in small concentrations. From the atmosphere, lead is transferred to soil, water, and vegetation by dry and wet deposition. A significant part of lead particles from emissions sources is of submicron size and can be transported over large distances. Larger lead particles settle more rapidly and closer to the source. Lead in soil binds hard, with a half-life of several hundred years. New depositions, primarily atmospheric, therefore contribute to increased concentrations. Atmospheric deposition is the largest source of lead in surface water, as well. Only limited amounts are transported to water from soil. Terrestrial and aquatic plants show a strong capability to bioaccumulate lead from water and soil in industrially contaminated environments (WHO 1989). Lead can also be taken up by grazing animals, thus entering the terrestrial food chain.

Natural atmospheric lead concentrations are estimated to be in the range of 0.00005 micrograms per cubic meter (µg / m3). Urban concentrations are around 0.5 µg / m^3, and annual average concentrations may reach 3 µg / m^3 or more in cities with heavy traffic (WHO 1987).

Sources and Uses

Mining, smelting, and processing of lead and lead-containing metal ores generate the greatest part of lead emissions from stationary sources. In addition, the combustion of lead-containing wastes and fossil fuels in incinerators, power plants, industries, and households releases lead into the atmosphere. Airborne ambient lead concentrations reaching over 100 µg / m^3 have occasionally been reported in the vicinity of uncontrolled stationary sources, decreasing considerably with distance from the source due to the deposition of larger lead particles.

As a result of the extensive use of alkyl-lead compounds as fuel additives, vehicular traffic is the largest source of atmospheric lead in many urban areas, accounting for as much as 90% of all lead emissions into the atmosphere (Brunekreef 1986). High concentrations of lead in urban air have been attributed to vehicular emissions in various countries (Lovei and Levy 1997). Traffic-generated lead aerosols are mostly of the submicron size; they can penetrate deeply into the lungs after inhalation, and they are transported and dispersed over large distances (Brunekreef 1986). With the phase-out of leaded gasoline, the relative contribution of traffic to environmental lead concentrations is changing.

Due to its special physical characteristics, lead has been used in a variety of products. Water distribution systems frequently contain lead pipes or lead solder, contaminating drinking water. Lead carbonate ("white lead") was highly popular as a base for oil paints before its use was banned in most countries in the first half of the twentieth century. Lead-based paint and dust contaminated by such paint still represent significant sources of human exposure in several countries. Lead-acid batteries contribute to the contamination of all environmental media during their production, disposal, and incineration. Lead compounds may be also used as stabilizers in plastics. Other lead-based products include food-can solder, ceramic glazes, crystal glassware, lead-jacketed cables, ammunition, and cosmetics.

Health Impacts of Exposure

The main pathways of lead to humans are ingestion and inhalation. Children up to about six years of age constitute the population group at the highest risk from lead exposure through ingestion: their developing nervous systems are susceptible to lead-induced disruptions; their intake of food is relatively high for their body weight; they are exposed to high intake from dust, dirt, soil, and lead-containing paint due to their hand-to-mouth behavior; and their absorption through the gut is very efficient. (According to WHO 1987, the proportion of lead absorbed from the gastrointestinal tract is four to five times higher in children than in adults.) The main sources of lead exposure of children are dust and dirt; the role of dissolved lead in water supply systems, lead-based paint, and other sources varies across locations. The contribution of drinking water to exposure is highest in infants under one year of age and children under five years of age. Lack of essential trace elements such as iron, calcium, and zinc and poor nourishment may increase the absorption of lead by the human body.

Inhalation poses the highest risk of exposure to environmental lead in adults. Inhaled airborne lead represents a relatively small part of the body burden in children, but in adults it ranges from 15 to 70%. About 30–50% of lead inhaled with particles is retained in the respiratory system and absorbed into the body (WHO 1987). In addition to environmental exposure, alcohol consumption and tobacco smoking have been shown to contribute to human exposure to lead. On the basis of a review of epidemiologic studies, Brunekreef (1986) concluded that a 0.1 µg / m^3 change in the ambient air concentration of lead was associated with a change in blood lead level—the best indicator of exposure—of 0.3 to 0.5 micrograms per decaliter (µg / dl).

Lead affects several organs of the human body, including the nervous system, the blood-forming system, the kidneys, and the cardiovascular and reproductive systems. Of most concern are the adverse effects of lead on the nervous system of young children: reducing intelligence and causing attention deficit, hyperactivity, and behavioral abnormalities. These effects occur at relatively low blood lead levels without a known lower threshold (Schwartz 1994). Many of these symptoms can be captured by standardized intelligence tests. Various studies have found a highly significant association between lead exposure and the measured intelligence quotient (IQ) of school-age children (Needleman et al. 1979; Bellinger et al. 1992). Reviews of studies concluded that a 10 µg / dl increase in blood lead can be associated with a 2–2.5 point decrease in IQ (CDC 1991; WHO 1995). The negative impact of lead exposure is generally stronger on verbal IQ than on performance IQ. (WHO 1995)

Prenatal exposure to lead was demonstrated to produce toxic effects in the human fetus, including reduced birth weight, disturbed mental development, spontaneous abortion, and premature birth. Such risks were significantly greater at blood lead levels of 15 µg / dl and more (WHO 1995).

High lead concentrations, generally due to occupational exposure or accidents, result in encephalopathy, a life-threatening condition at blood lead levels of 100 to 120 µg / dl in adults and 80 to 100 µg / dl in children (ATSDR 1990). An acute form of damage to the gastrointestinal tract known as “lead colic” is also associated with high lead levels. The hematological effects of lead exposure are attributed to the interruption of biosynthesis of heme by lead, severely inhibiting the metabolic pathway and resulting in reduced output of hemoglobin. Reduced heme synthesis has been associated with blood levels over 20 µg / dl in adults and starting from below 10 µg / dl in children (WHO 1987).

Several studies (Schwartz 1988, 1995; Pocock et al. 1988, Hu et al. 1996; Kim et al. 1996) have shown that increased blood pressure and hypertension in adults are also related to elevated blood lead levels, even at lower levels of exposure, increasing the risk of cardiovascular diseases (Pirkle et al. 1985).

Ambient Standards and Guidelines

Ambient standards and guidelines are aimed at protecting human health. Table 1 includes EU, USEPA, and WHO reference standards and guidelines for ambient levels of lead in air and water.

Table 1. Reference Standards and Guidelines for Mean Ambient Lead Concentrations in Air and Water

Medium	*EU limit values*	*USEPA standard*	*WHO guide values*
Air (micrograms per cubic meter)	2	1.5	0.5–1.0[b]
Drinking water and surface water intended for drinking (micrograms per liter)	10	50	10

a. Maximum arithmetic mean over a calendar quarter.
b. Annual mean.
Sources: Air: CEC 1982 (EU), United States, 40 CFR, Part 532 (USEPA); WHO 1987. Water: CEC 1980 (EU); USEPA 1987; WHO 1993.

Conclusions

People are exposed to lead from a variety of sources and in a variety of ways, and ambient guidelines and standards for individual media may not provide sufficient protection. A comprehensive approach and strategy is therefore necessary to protect human health. Ambient environmental quality guidelines and standards should be only the starting point for such a strategy. Environmental monitoring of ambient concentrations in soil, air, and drinking water should help to identify highly polluted areas and high-risk population groups. This step should be followed by targeted biological screening and policy intervention. Such an approach should be the core of a comprehensive policy intervention that deals with lead exposure from all sources.

Recommendations

Stationary sources that contribute to the increase of lead in the environment should not exceed the lead emissions referred to in the relevant industry section of this *Handbook.* These emissions are normally achievable through good industrial practices.

In addition, the impacts of new stationary sources on ambient concentrations of lead should be considered. When the use of certain processes results in emissions that contribute to a significant increase in ambient lead concentrations, or in areas where significant background concentrations exist, the environmental assessment should ensure that lead emissions are properly abated, taking into consideration alternative technologies and control measures. Intermittent monitoring of ambient air, water, and soil should ensure that lead concentrations do not impose an increased health threat to the population in the vicinity of the industrial plant.

References and Sources

ATSDR (Agency for Toxic Substances and Disease Registry). 1990. *Toxicological Profile for Lead.* Washington, D.C.: United States Public Health Service in collaboration with United States Environmental Protection Agency.

Bellinger, D., et al. 1992. "Low-Level Lead Exposure, Intelligence, and Academic Achievement: A Long Term Follow-up Study." *Pediatrics.*

Brunekreef, B. 1986. *Childhood Exposure to Environmental Lead.* MARC Report 34. London: Monitoring and Assessment Research Centre, King's College, University of London.

CDC (Centers for Disease Control). 1991. *Strategic Plan for the Elimination of Childhood Lead Poisoning.* Washington, D.C.: U.S. Department of Health and Human Services.

CEC (Commission of the European Communities). 1975. *Official Journal of the European Communities* 194(26). Luxembourg.

————. 1980. *Official Journal of the European Communities* 229(11). Luxembourg.

————. 1982. *Official Journal of he European Communities* 378(15). Luxembourg

Hayes, Edward B., et al. 1994. "Long-Term Trends in Blood Lead Levels among Children in Chicago: Relationship to Air Lead Levels." *Pediatrics* 93(2).

Hu, H., et al. 1996. "The Relationship of Bone and Blood Lead to Hypertension." *Journal of the American Medical Association* 27(15): 1171–76.

Kim, R., et al. 1996. "A Longitudinal Study of Low-Level Lead Exposure and Impairment of Renal Function." *Journal of the American Medical Association* 275(15): 1177–81.

Lovei, Magda, and B. S. Levy. 1997. "Lead Exposure and Health in Central and Eastern Europe: Evidence from Hungary, Poland and Bulgaria." In Magda

Lovei, ed., *Phasing Out Lead from Gasoline in Central and Eastern Europe: Health Issues, Feasibility, and Policies.* Washington, D.C.: World Bank.

National Research Council, Committee on Measuring Lead in Critical Populations. 1993. *Measuring Lead Exposure in Infants, Children, and Other Sensitive Populations.* Washington, D.C.: National Academy Press.

Needleman, H. L., et al. 1979. "Deficits in Psychologic and Classroom Performance in Children with Elevated Dentine Lead Levels." *New England Journal of Medicine* 300: 584–695.

Pirkle, J. L., et al. 1985. "The Relationship between Blood Lead Levels and Blood Pressure and U.S. Cardiovascular Risk Implications." *American Journal of Epidemiology* 121: 246–58.

Pocock, S. J., et al. 1988. "The Relationship between Blood Lead, Blood Pressure, Stroke and Health Attacks in Middle-Aged British Men." *Environmental Health Perspectives* 78.

Schlag, R. D. 1987. "Lead." In Lawrence Fishbein, Arthur Furst, and Myron A. Mehlman, eds., *Genotoxic and Carcinogenic Metals: Environmental and Occupational Occurrence and Exposure.* Advances in Modern Environmental Toxicology, vol. 11. Princeton, N.J.: Princeton Scientific Publishing Co.

Schwartz, Joel. 1988. "The Relationship between Blood Lead and Blood Pressure in the NHANES II Survey." *Environmental Health Perspectives* 78.

————. 1994. "Low Level Lead Exposure and Children's IQ: A Meta Analysis and Search for a Threshold." *Environmental Research* 65(1): 42–-55.

————. 1995. "Lead, Blood Pressure and Cardiovascular Disease in Men." *Archives of Environmental Health* 50(1): 31–37.

United States. CFR (*Code of Federal Regulations*). Washington, D.C.: Government Printing Office.

USEPA (United States Environmental Protection Agency). 1986. *Reducing Lead in Drinking Water: A Benefits Analysis.* EPA-230-09-86-019. Washington, D.C.: Office of Policy, Planning and Evaluation.

————. 1987. *Quality Criteria for Water 1986.* EPA 440/5-86-001. Washington, D.C.: Office of Water Regulations and Standards.

WHO (World Health Organization). 1987. *Air Quality Guidelines for Europe.* Copenhagen: WHO Regional Office for Europe.

————. 1993. *Guidelines for Drinking Water Quality,* vol. 1, no. 15: *Recommendations.* 2d ed. Geneva.

————. 1995. *Inorganic Lead.* International Programme on Chemical Safety. Geneva.