Lead and Lead Compounds

CAS No. 7439-92-1 (Lead)

No separate CAS No. assigned for lead compounds as a class

Reasonably anticipated to be human carcinogens

First listed in the *Eleventh Report on Carcinogens* (2004)

Also known as Pb

Introduction

The compounds lead phosphate and lead acetate were first listed in the *Second Annual Report on Carcinogens* in 1981 as *reasonably anticipated to be human carcinogens* based on sufficient evidence of carcinogenicity in experimental animals. The listing of lead and lead compounds supersedes the previous listing of lead phosphate and lead acetate in the Report on Carcinogens and applies to lead and all lead compounds.

Carcinogenicity

Lead and lead compounds are *reasonably anticipated to be human carcinogens* based on limited evidence of carcinogenicity from studies in humans and sufficient evidence of carcinogenicity from studies in experimental animals.

Cancer Studies in Humans

Lead exposure has been associated with increased risk of lung, stomach, and urinary-bladder cancer in diverse human populations (Fu and Boffetta 1995, Steenland and Boffetta 2000, NTP 2003). The strongest epidemiological evidence is for lung and stomach cancer, which are consistently but weakly associated with occupations and industries entailing lead exposure and with indices of individual lead exposure, including job history and biological monitoring of occupationally exposed and general populations. However, most studies of lead exposure and cancer reviewed had limitations, including poor exposure assessment and failure to control for confounding by other factors that could increase the risk of cancer (such as lifestyle factors and concurrent occupational exposure to other carcinogens), and did not demonstrate relationships between the level or duration of exposure and the magnitude of cancer risk. The crude exposure measures used in most studies, such as treating whole plants or occupations as having uniform exposure, may have limited the magnitude of risk estimates, most of which were modest. Evidence from epidemiological studies therefore is compatible with small increases in the risk of lung or stomach cancer; however, this evidence must be weighed against the potential for confounding by factors such as smoking, diet, or co-exposure to arsenic.

Cancer Studies in Experimental Animals

Lead compounds caused tumors in several species of experimental animals, at several different tissue sites, and by several different routes of exposure. Carcinogenicity was observed in studies with inorganic lead compounds, both soluble (lead acetate and lead subacetate) and insoluble (lead phosphate, lead chromate), and with tetraethyl lead (an organic lead compound). Lead caused cancer in rats and/or mice exposed orally, by injection, or perinatally (via the placenta or lactation). Benign and malignant kidney tumors (adenoma, carcinoma, and adenocarcinoma) were most frequently associated with lead exposure, and tumors of the brain, hematopoietic system, and lung were reported in some studies (IARC 1980, 1987).

Lead subacetate administered in the diet caused benign and malignant kidney tumors (adenoma and carcinoma or adenocarcinoma) in mice and rats of both sexes and brain tumors (glioma) in rats, and its administration by intraperitoneal injection caused benign lung tumors (adenoma) in mice. Lead acetate administered in the diet or drinking water caused benign and malignant kidney tumors (adenoma and carcinoma) in rats and increased the incidence of virus-induced lymphocytic leukemia in mice. After pregnant mice were given lead acetate in the drinking water from gestation day 12 to four weeks postpartum, the offspring showed a dose-related increase in proliferative lesions of the kidneys (including atypical hyperplasia, adenoma, and adenocarcinoma) (Waalkes *et al.* 1995). Rats exposed to lead phosphate by subcutaneous injection (alone or followed by intraperitoneal injection) developed benign or malignant kidney tumors (adenoma or carcinoma). Rats exposed to lead chromates by subcutaneous injection developed cancer at the injection site (sarcoma), and administration of lead chromates by intramuscular injection caused kidney cancer (renal-cell carcinoma) (IARC 1990). (Because lead chromate is also a hexavalent chromium compound, it is also included in the listing for Chromium Hexavalent Compounds.) Tetraethyl lead administered by subcutaneous injection caused lymphoma in female mice. Exposure to lead naphthenate, lead carbonate, lead arsenate, lead nitrate, and metallic lead (as lead powder) did not significantly increase tumor incidences in experimental animals (IARC 1980).

Studies on Mechanisms of Carcinogenesis

Exposure of rodents to lead compounds also increased the incidence or accelerated the appearance of kidney tumors induced by other carcinogens, including *N*-ethyl-*N*-hydroxyethylnitrosamine and *N*-(4′-fluoro-4-biphenyl)acetamide. Higher incidences of kidney and liver cancer were observed in rats fed diets containing lead subacetate and 2-acetylaminofluorene than in rats fed either lead subacetate or 2-acetylaminofluorene alone (IARC 1980, 1987).

Absorption of lead is affected by age, the chemical form of the lead, and minerals in the diet (e.g., iron, calcium, and zinc) (ATSDR 1999). Gastrointestinal absorption of lead is greater in children than in adults (Hammad *et al.* 1996). Once absorbed, lead is distributed to blood plasma, the nervous system, and soft tissues. It subsequently is redistributed and accumulates in bone; 75% to 90% of the lead body burden is found in bones and teeth.

In studies of humans occupationally exposed to lead, there is evidence to suggest that lead damages chromosomes or DNA. In most studies, lead caused micronucleus formation, chromosomal aberrations, and DNA damage, but studies on sister chromatid exchange gave conflicting results. Genetic studies on humans environmentally exposed to lead also gave conflicting results. Lead did not cause mutations in bacteria, and results from test systems using mammalian cells were conflicting. Lead caused chromosomal aberrations in most studies in plants or mammals, both *in vitro* and *in vivo*. It caused DNA damage or fragmentation in mammals *in vivo* and in cell-free systems (in the presence of hydrogen peroxide), but mammalian *in vitro* studies gave conflicting results. Lead also inhibited the activity of DNA and RNA polymerase in cell-free systems and in mammalian cell cultures. Conflicting results were observed for sister chromatid exchange and micronucleus formation in mammalian test systems (*in vitro* and *in vivo*) (ATSDR 1999, NTP 2003).

The mechanisms by which lead causes cancer are not understood. Lead compounds do not appear to cause genetic damage directly, but may do so through several indirect mechanisms, including inhibition of DNA synthesis and repair, oxidative damage, and interaction with DNA-binding proteins and tumor-suppressor proteins (NTP 2003).

Properties

Elemental lead is an odorless, silver-bluish-white metal that is insoluble in water (Budavari *et al.* 1996, Lide and Frederikse 1998, HSDB 2009). It is soft, highly malleable, ductile, and a relatively poor conductor of electricity. It is resistant to corrosion but tarnishes upon exposure to air. Lead exists in the valence states of +2 and +4 and has four naturally occurring stable isotopes: ^{204}Pb, ^{206}Pb, ^{207}Pb, and ^{208}Pb. Inorganic lead compounds usually consist of lead in the divalent state (+2), and the chemistry of divalent lead is similar to that of group 2 metals (beryllium, magnesium, calcium, strontium, and barium).

Lead compounds may be divided between those compounds that are relatively soluble in water and those that are relatively insoluble in water. Compounds are considered soluble or insoluble based on the following criteria: (1) If a solubility constant (K_{sp}) is available, a compound with a value greater than or equal to the K_{sp} for lead chloride (1×10^{-4}) is considered soluble. (2) If a K_{sp} is not available, a compound is considered soluble if more than 2 g of the compound dissolves in 100 mL of water. (3) If no numeric solubility data are available, the compounds are considered soluble or insoluble according to the general rules of solubility.

The major soluble lead compounds are lead acetate, lead acetate trihydrate, lead chloride, lead nitrate, and lead subacetate; all are soluble in water, and lead acetate trihydrate is miscible with water. Lead acetate exists as colorless or white crystals, granules, or powders that are soluble in glycerol and slightly soluble in ethanol. Lead acetate trihydrate occurs as white crystals that are slightly soluble in ethanol and acetone. Lead chloride exists as a white crystalline powder that is insoluble in ethanol. Lead nitrate occurs as colorless or white crystals that are insoluble in nitric acid. Lead subacetate is a white heavy powder that is soluble in ethanol (HSDB 2009).

The major insoluble lead compounds include 17 inorganic lead compounds. Lead arsenate, lead azide, lead bromide, lead fluoride, lead phosphate, lead stearate, lead sulfate, and lead thiocyanate occur as white powders, crystals, or needles. Lead carbonate occurs as colorless rhombic crystals, and lead fluoborate occurs as a colorless crystalline powder. Lead chromate, lead iodide, lead naphthenate, lead oxide, and lead styphnate occur as yellow to reddish-yellow powder, crystals, or paste. Lead sulfide occurs as metallic black cubic crystals, and lead tetraoxide is a bright-red heavy powder. Lead arsenate, lead fluoride, and lead phosphate are soluble in nitric acid, and lead arsenate, lead carbonate, lead oxide, lead phosphate, lead sulfate, and lead thiocyanate are soluble in potassium hydroxide or other alkalis. Lead bromide, lead iodide, lead oxide, lead phosphate, and lead sulfate are insoluble in alcohol, and lead fluoborate decomposes in alcohol. Lead tetraoxide is soluble in hydrochloric and acetic acids and insoluble in ethanol. The reported melting points of these compounds range from 100°C (lead naphthenate) to 1,170°C (lead sulfate). All of the insoluble inorganic lead compounds have high boiling points (up to 1,470°C); however, lead carbonate decomposes before it boils, and lead azide explodes at 350°C. Most of these compounds have high specific gravities, ranging from 6.2 for lead sulfate to 9.53 for lead oxide, but a few have lower specific gravities, including lead naphthenate (1.15), lead fluoborate (1.75), and lead thiocyanate (3.82) (HSDB 2009, Akron 2010).

Tetraethyl lead and tetramethyl lead are insoluble organic lead compounds. They both exist as colorless liquids and are soluble in benzene, ethanol, and diethyl ether. The octanol-water partition coefficients are 4.15 for tetraethyl lead and 2.97 for tetramethyl lead (HSDB 2009). The following table lists physical and chemical properties for lead, the major soluble inorganic lead compounds, and the organic lead compounds.

Substance	Specific gravity	Melting pt.	Boiling pt.
Lead	11.34	327°C	1,740°C
Lead acetate	3.25	280°C	dec
Lead acetate trihydrate	2.55	75°C	200°C (dec)
Lead chloride	5.85	501°C	950°C
Lead nitrate	4.53	470°C	dec
Lead subacetate	NR	75°C	dec
Tetraethyl lead	1.659	–136.8°C	200°C
Tetramethyl lead	1.995	–30.2°C	110°C

Source: HSDB 2009. dec = decomposes. NR = not reported.

Use

In worldwide metal use, lead ranks behind only iron, copper, aluminum, and zinc (Howe 1981). Its largest use is in lead-acid storage batteries for motor vehicles and general industry. Lead metal also is commonly used for ammunition, cable covering, piping, brass and bronze, bearing metals for machinery, and sheet lead (ATSDR 1999).

All of the major soluble lead compounds have industrial uses. Lead acetate is used as a water repellent, for mildew protection, and as a mordant for cotton dyes. Lead acetate trihydrate is used in varnishes, chrome pigments, and as an analytical reagent, and lead chloride is used in asbestos clutch or brake linings, as a catalyst, and as a flame retardant. Lead nitrate is used in the manufacture of matches and explosives, as a heat stabilizer in nylon, and as a coating on paper for photothermography. Lead subacetate is used in sugar analysis and for clarifying solutions of organic substances (HSDB 2009).

The insoluble lead compounds also have a variety of uses. Lead azide and lead styphnate both are used in munitions manufacture. Lead carbonate, lead fluoride, lead fluoborate, and lead naphthenate are used as catalysts, with additional uses in the electronic and optical industries (lead fluoride), in coatings for thermographic copying (lead carbonate), as a curing agent for epoxy resins (lead fluoborate), and as a varnish drier (lead naphthenate). Lead phosphate and lead stearate both are used as stabilizers in the plastics industry. Lead iodide and lead sulfate are used in photography; lead iodide is also used in thermoelectric materials, and lead sulfate with zinc in galvanic batteries. Lead oxide and lead sulfide are used in ceramics; lead oxide is also used as a vulcanizing agent in rubber and plastics, and lead sulfide as a humidity sensor in rockets. Lead chromate is used as a pigment in paints, rubber, and plastics; lead tetraoxide is used in plasters, ointments, glazes, and varnishes; and lead thiocyanate is used in the manufacture of safety matches and cartridges. Lead arsenate formerly was used as an insecticide and herbicide, but no current uses were found.

Organic lead (including tetraethyl lead and tetramethyl lead) was widely used in the United States as an anti-knock additive in motor-vehicle fuels until the U.S. Environmental Protection Agency initiated a phase-out of leaded gasoline in the early 1970s. By 1988, the total lead used in gasoline had been reduced to 1% of the 1970 level; in 1996, the use of lead in fuel for on-road motor vehicles was totally banned. Despite the legislated end to use of lead as a gasoline additive and reductions in some other uses of lead, overall U.S. lead consumption continued to grow until 1999, mainly because of increased production of lead-acid batteries (ATSDR 1999), but has since been on a general decline (USGS 2009, 2010, Guberman 2010).

Production

Lead is refined from mined ore, which occurs most frequently in the form of lead sulfide, also known as galena (Howe 1981). Mined lead ore is crushed and ground, and a lead concentrate is formed by separation of the various minerals. The lead concentrate is shipped to a primary smelter for refining. At the smelter, lead concentrates are sintered, roasted, and refined into lead metal that is 99.99% pure.

However, secondary lead, produced from recycled scrap (primarily from lead acid batteries), accounts for the majority of lead produced in the United States.

In 2009, 400,000 metric tons (882 million pounds) of lead was mined in the United States, a slight decline from levels over the previous four years (USGS 2010). Primary lead production in the United States has declined steadily over the past several decades, from a high of 626,000 metric tons (1.4 billion pounds) in 1970 to 115,000 metric tons (254 million pounds) in 2009 (USGS 2009, 2010). In contrast, secondary lead production has increased steadily over the same period, from 450,000 metric tons (992 million pounds) in 1970 to 1,120,000 metric tons (2.5 billion pounds) in 2009, when it accounted for about 90% of the total refined lead produced in the United States. In 2009, five lead mines in Missouri, plus lead-producing mines in Alaska and Idaho, yielded most of the mined lead in the United States. Lead was processed at one smelter-refinery in Missouri. Of the 21 plants that produced secondary lead, 15 accounted for over 99% of secondary production (USGS 2010).

From 1980 to 1999, lead consumption in the United States rose steadily from 906,000 metric tons (2 billion pounds) to 1,760,000 metric tons (3.9 billion pounds), but consumption has since generally declined; in 2009, it was 1,420,000 metric tons (3.1 billion pounds). In 2009, lead was consumed at 76 manufacturing plants, with lead-acid battery production accounting for 88% of U.S. lead consumption. U.S. imports and exports of lead have fluctuated widely over the past several decades. Imports have ranged from a low of 85,000 metric tons (187 million pounds) in 1980 to a high of 365,000 metric tons (805 million pounds) in 2000; imports in 2009 were 275,000 metric tons (606 million pounds). Exports of refined lead metal have ranged from a low of 5,000 metric tons (11 million pounds) in 1976 to a high of 164,000 metric tons (362 million pounds) in 1980; exports in 2009 were 85,000 metric tons (187 million pounds) (USGS 2010).

Lead acetate was first produced in the United States in 1944; however, little production information was found. Three companies reported production of an undisclosed amount of lead acetate in 1977. Production volumes were estimated at over 6,810 kg (15,000 lb) in 1978 and over 2,270 kg (5,000 lb) in 1982, and U.S. imports were 113 kg (250 lb) in 1978 and 39,300 kg (87,000 lb) in 1982 (IARC 1980, HSDB 2009). Lead nitrate was first commercially produced in the United States in 1943, and imports of 480,000 kg (1.06 million pounds) were reported in 1978. Commercial production of lead subacetate was first reported in the United States in 1947; no production data were found (IARC 1980).

Lead carbonate has been produced commercially in the United States since the 1600s; in 1976, U.S. production was 1.48 million kilograms (3.3 million pounds), with imports in 1978 of 178,000 kg (392,000 lb) (IARC 1980). U.S. exports of lead carbonate in 2002 were 779,071 kg (1.7 million pounds) (USITC 2003). U.S. production of lead oxide in 1976 was 120 million kilograms (260 million pounds), with imports of 20 million kilograms (44 million pounds) (IARC 1980). U.S. imports of lead oxides in 2002 totaled 3.9 million kilograms (8.6 million pounds), and exports totaled 1.7 million kilograms (3.7 million pounds) (USITC 2003). Commercial production of lead naphthenate in the United States was first reported in 1944. Production of lead naphthenate was 8.2 million kilograms (18.1 million pounds) in 1969, dropping to 2.2 million kilograms (4.9 million pounds) in 1977. U.S. production of lead tetraoxide in 1976 was 18 million kilograms (39.7 million pounds), with imports of 800,000 kg (1.8 million pounds) in 1976 and 1 million kilograms (2.2 million pounds) in 1979, and exports were estimated at 1 million to 15 million kilograms (2.2 million to 33 million pounds) in 1977 (IARC 1980).

Tetraethyl lead was first produced commercially in the United States in 1923. Production was 266 million kilograms (590 million pounds) in 1964, dropping to 148 million kilograms (330 million pounds) in 1977. U.S. imports of tetraethyl lead in 1978 were 17,000 kg (37,500 lb). Commercial production of tetramethyl lead in the United States began in 1960; 54 million kilograms (119 million pounds) was produced in 1977, and 13,800 kg (30,400 lb) was imported in 1974 (IARC 1980).

Exposure

The routes of environmental exposure to lead resulting in its absorption into the body are inhalation (with 30% to 50% of the inhaled dose absorbed into the bloodstream), ingestion (with 8% to 15% of the ingested dose absorbed into the bloodstream) and, to a limited extent, dermal contact. Lead is released to the environment from both natural and anthropogenic sources; however, most exposure results from anthropogenic sources (e.g., mining, smelting, industrial uses). Lead exists in various inorganic and organic forms, which affect its environmental fate, transport, and bioavailability. Regardless of the form, however, lead is not degraded and remains available for exposure. In the mid 1980s, combustion of leaded gasoline contributed about 90% of all anthropogenic lead emissions, but the percentage decreased sharply through the late 1990s as a result of the phase-out of leaded gasoline (ATSDR 1999, EPA 2003). Over 90% of the lead released from the combustion of leaded gasoline was in the form of inorganic lead halides (e.g., lead bromochloride), while less than 10% was in the form of organic lead alkyls (e.g., tetraethyl lead). Tetraalkyl lead compounds once accounted for 5% to 10% of the total particulate lead present in the atmosphere but are no longer present in significant quantities. Industrial processes, particularly lead smelters, are now the primary source of lead emissions and accounted for more than 78% of emissions in 2001 (EPA 2003).

According to EPA's Toxics Release Inventory, over 4,000 facilities released almost 22 million pounds of lead and 482 million pounds of lead compounds to the environment in 2007 (TRI 2009). Concentrations of lead in the air in the United States declined by 97% between 1976 and 1995 and by 57% between 1993 and 2002 (ATSDR 1999, EPA 2003). Ambient concentrations are highly variable but may exceed 10 $\mu g/m^3$ near industrial sources such as smelters (ATSDR 1999). A 1991 survey of lead levels in U.S. urban air found a maximum quarterly mean concentration of approximately 0.08 $\mu g/m^3$. Lead concentrations typically are lower in rural areas. In 1995, the estimated U.S. mean air lead concentration was 0.04 $\mu g/m^3$ (EPA 1996). The estimated daily average intake of lead by inhalation in 1991 was 2 µg for an adult living in a U.S. urban setting, significantly lower than estimates from the early 1980s (ATSDR 1999).

Lead concentrations in U.S. drinking water generally are below 5 µg/L. Lead also is found in food, cigarette smoke, and alcoholic beverages. Levels in food have declined since the elimination of lead-soldered food cans between 1979 and 1989 (ATSDR 1999). In 1990, the estimated daily intake of lead from consumption of food, water, and beverages was approximately 4 µg for children aged 2 years or younger, 6 to 9 µg for children aged 14 to 16, 6 to 9 µg for adults aged 25 to 30, and 2 to 8 µg for adults aged 60 to 65. For young children, the most common source of environmental lead exposure is direct ingestion of paint chips and lead-laden dust and soil released from aging painted surfaces. These sources can contribute an additional daily intake of 5 µg for a toddler engaging in normal hand-to-mouth activity (CDC 1997, Lanphear *et al.* 1998).

The most common route of occupational exposure to lead is inhalation of lead fumes or lead-laden dusts in air and absorption of lead through the respiratory system. Lead may also be ingested and ab-

sorbed via the gastrointestinal tract (Bress and Bidanset 1991, Stauber *et al.* 1994). The National Institute for Occupational Safety and Health has estimated that more than three million Americans potentially are occupationally exposed to some form of lead (Staudinger and Roth 1998). Occupations having frequent high exposure to lead include battery-production worker, battery-recycling worker, foundry worker, lead chemical worker, lead smelter and refinery worker, leaded-glass worker, pigment worker, and radiator-repair worker. Occupations with a moderate frequency of high exposure include firing-range instructor, house renovator, lead miner, newspaper printer, plastics worker, rubber worker, and steel welder or cutter. Occupations with a low frequency of high exposure include automobile-repair worker, cable-production worker, construction worker, demolition worker, firing-range participant, flame-solder worker, plumber or pipe fitter, pottery-glaze producer, ship-repair worker, and stained-glass producer (Fu and Boffetta 1995, ATSDR 1999). For U.S. industries identified by the Occupational Safety and Health Administration as having significant airborne lead in the workplace, the mean concentration ranged from 165 μg/m^3 at secondary smelters to 200 μg/m^3 at storage-battery plants and brass, bronze, and copper foundries (Froines *et al.* 1990).

Regulations

Consumer Product Safety Commission (CPSC)

Accessible parts of products designed or intended primarily for children 12 and younger may not contain more than 100 ppm of lead; products exceeding this level are banned hazardous substances.

Paint or any other surface-coating materials for consumer use shall not contain lead at levels greater than 90 ppm.

Toys and other items for child use that bear paint with lead at levels greater than 0.009% of the total weight of the solid or dried paint film are banned.

Furniture articles for consumer use that bear paint with lead at levels greater than 0.009% of the total weight of the solid or dried paint film are banned.

Metal-cored candlewicks containing more than 0.06% lead by weight in the metal, and candles with such wicks, are banned.

Department of Transportation (DOT)

Numerous specific lead compounds, and lead compounds not otherwise specified, are considered hazardous materials and marine pollutants, and special requirements have been set for marking, labeling, and transporting these materials.

Environmental Protection Agency (EPA)

Clean Air Act

National Ambient Air Quality Standards: National primary and secondary ambient air quality standard = 1.5 μg/m^3 for lead and lead compounds.

National Emission Standards for Hazardous Air Pollutants: Lead compounds are listed as a hazardous air pollutant.

New Source Performance Standards: Manufacture of tetraethyl lead and tetramethyl lead is subject to provisions for the control of volatile organic compound emissions.

Prevention of Accidental Release: Threshold quantity (TQ) = 10,000 lb for tetramethyl lead.

Urban Air Toxics Strategy: Lead compounds are identified as one of 33 hazardous air pollutants that present the greatest threat to public health in urban areas.

Mobile Source Air Toxics: Lead compounds are listed as a mobile source air toxic for which regulations are to be developed.

As defined by the Clean Air Act, gasoline which contains lead additives or contains lead at a concentration greater than 0.05 g/gal shall not be sold for use in motor vehicles.

Clean Water Act

Biosolids Rule: Limits have been established for lead in biosolids (sewage sludge) when used or disposed of via land application or incineration.

Effluent Guidelines: Lead and lead compounds are listed as toxic pollutants.

Numerous lead compounds are designated as hazardous substances.

Comprehensive Environmental Response, Compensation, and Liability Act

Reportable quantity (RQ) = 10 lb for lead, lead acetate, lead chloride, lead fluoborate, lead fluoride, lead iodide, lead nitrate, lead phosphate, lead stearate, lead subacetate, lead sulfate, lead sulfide, lead thiocyanate, and tetraethyl lead; = 1 lb for lead arsenate.

Emergency Planning and Community Right-To-Know Act

Toxics Release Inventory: Lead and lead compounds are listed substances subject to reporting requirements.

Reportable quantity (RQ) = 10 lb for tetraethyl lead; = 100 lb for tetramethyl lead.

Threshold planning quantity (TPQ) = 100 lb for tetraethyl lead and tetramethyl lead.

Federal Insecticide, Fungicide, and Rodenticide Act

All registrations for pesticides that have lead arsenate as an active ingredient have been canceled.

Resource Conservation and Recovery Act

Characteristic Hazardous Waste: Toxicity characteristic leaching procedure (TCLP) threshold = 5.0 mg/L.

Listed Hazardous Waste: Waste codes for which the listing is based wholly or partly on the presence of lead or lead compounds = F035, F037, F038, K002, K003, K005, K046, K048, K049, K051, K052, K061, K062, K069, K086, K100, K176, P110, P116, U144, U145, U146.

Lead and lead compounds are listed as hazardous constituents of waste.

Safe Drinking Water Act

Treatment technique, action level = 0.015 mg/L for lead.

Numerous requirements have been established to reduce exposure to lead in drinking water due to lead leaching from lead pipes and lead fittings.

Toxic Substances Control Act

A seller must disclose to the purchaser of a home any known lead-based paint hazards.

Comprehensive regulations have been developed to prevent lead-based paint poisoning in certain residential structures.

Food and Drug Administration (FDA)

A conspicuous label shall be on the surface of ornamental or decorative ceramics that contain lead warning that the vessel is not for food use and may be harmful if used for such.

A number of food additives generally recognized as safe are permitted for use in foods for human consumption providing maximum lead levels do not exceed concentrations prescribed in 21 CFR 184.

Action levels for lead in ceramic ware, hollowware, cups, mugs, and pitchers range from 0.5 to 7 μg/mL of leaching solution.

Lead acetate hair coloring must provide warning labels and may be safely used in cosmetics intended for coloring hair on the scalp if lead levels do not exceed 0.6% (weight to volume).

Lead solder may not be used in food packaging.

Maximum allowed levels of lead in various color additives used in food, drugs, cosmetics, and medical devices are provided 21 CFR 73 and 74.

Maximum permissible level of lead in bottled water = 0.005 mg/L.

Select food additives are permitted for use in animal feed with maximum lead levels ranging from 10 to 30 ppm.

Restrictions on the use of lead in various food additives are prescribed in 21 CFR 172.

Limits on the use of lead in feed and drinking water of animals are prescribed in 21 CFR 584.

Tin-coated lead foil capsules shall not be used for wine bottles.

Department of Housing and Urban Development (HUD)

HUD's Lead-Based Paint Disclosure Rule requires that a seller or lessor disclose to the purchaser the presence of any lead-based paint in a home for sale, provide an EPA pamphlet on the health effects of lead, provide records on lead-based paint used in home, and provide a 10-day period to conduct a home inspection for lead-based paint or lead-based paint hazards.

HUD has established regulations to implement the provisions set forth in the Residential Lead-Based Paint Hazard Reduction Act. In part, the goals of these regulations are to develop a national strategy to build the infrastructure necessary to eliminate lead-based paint hazards in all housing as expeditiously as possible, and to ensure that the existence of lead-based paint hazards is taken into account in the development of government housing policies and in the sale, rental, and renovation of homes and apartments.

Occupational Safety and Health Administration (OSHA)

While this section accurately identifies OSHA's legally enforceable PELs for this substance in 2010, specific PELs may not reflect the more current studies and may not adequately protect workers.

Permissible exposure limit (PEL) = 0.050 mg/m^3 for metallic lead, inorganic lead compounds, and organic lead soaps.

Comprehensive standards have been developed for occupational exposure to metallic lead, all inorganic lead compounds, and organic lead soaps.

Guidelines

American Conference of Governmental Industrial Hygienists (ACGIH)

Threshold limit value – time-weighted average (TLV-TWA) = 0.05 mg/m^3 for lead, inorganic lead compounds, and lead chromate; = 0.15 mg/m^3 for tetramethyl lead; = 0.1 mg/m^3 for tetraethyl lead.

Biological Exposure Index (BEI) (sampling time not critical) = 30 μg/100 mL for lead in blood.

Consumer Product Safety Commission (CPSC)

Manufacturers are requested to eliminate the use of lead that may be accessible to children from products used in or around households, schools, or in recreation.

It is recommended that before purchasing products for resale, importers, distributors, and retailers make assurances that those products do not contain lead that may be accessible to children.

National Institute for Occupational Safety and Health (NIOSH)

Recommended exposure limit (REL) = 0.05 mg/m^3 (as metallic lead) for metallic lead, lead oxides, and lead salts (including organic salts such as lead soaps but excluding lead arsenate); = 0.002 mg/m^3 (as arsenic) for lead arsenate (15-min exposure) (listing for inorganic arsenic compounds).

Immediately dangerous to life and health (IDLH) limit = 100 mg/m^3 (as metallic lead).
Air concentrations should be maintained so that worker blood-lead levels remain at less than 0.06 mg Pb/100 g of whole blood.

References

Akron. 2010. The Chemical Database. The Department of Chemistry at the University of Akron. http://ull.chemistry.uakron.edu/erd and search on CAS number. Last accessed: 3/23/10.

ATSDR. 1999. *Toxicological Profile for Lead (Final Report)*. Agency for Toxic Substances and Disease Registry. http://www.atsdr.cdc.gov/toxprofiles/tp13.pdf.

Bress WC, Bidanset JH. 1991. Percutaneous *in vivo* and *in vitro* absorption of lead. *Vet Hum Toxicol* 33(3): 212-214.

Budavari SM, O'Neal J, Smith A, Heckelman PE, eds. 1996. *The Merck Index*, 12th ed. Whitehall, NJ: Merck & Company.

CDC. 1997. Update: blood lead levels—United States 1991-1994. *Morbid Mortal Wkly Rep* 46(7): 141-146.

EPA. 1996. *National Air Quality and Emissions Trends Report, 1995*. U.S. Environmental Protection Agency. http://www.epa.gov/airtrends/aqtrnd95/report.

EPA. 2003. Lead. In *Latest Findings on National Air Quality: 2002 Status and Trends.* U.S. Environmental Protection Agency. http://www.epa.gov/airtrends/aqtrnd02/2002_airtrends_final.pdf. p. 17.

Froines JR, Baron S, Wegman DH, O'Rourke S. 1990. Characterization of the airborne concentrations of lead in U.S. industry. *Am J Ind Med* 18(1): 1-17.

Fu H, Boffetta P. 1995. Cancer and occupational exposure to inorganic lead compounds: a meta- analysis of published data. *Occup Environ Med* 52(2): 73-81.

Guberman DE. 2010. Lead [Advance Release]. In *Minerals Yearbook, Vol. I, Metals and Minerals.* U.S. Geological Survey. http://minerals.usgs.gov/minerals/pubs/commodity/lead/myb1-2008-lead.pdf.

Hammad TA, Sexton M, Langenberg P. 1996. Relationship between blood lead and dietary iron intake in preschool children. A cross-sectional study. *Ann Epidemiol* 6(1): 30-33.

Howe HE. 1981. Lead. In *Kirk-Othmer Encyclopedia of Chemical Technology*, 3rd ed, vol. 14. New York: John Wiley & Sons. pp. 98-139.

HSDB. 2009. *Hazardous Substances Data Bank.* National Library of Medicine. Last updated: 5/20/99. http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB and search on CAS number or compound name.

IARC. 1980. Lead and lead compounds. In *Some Metals and Metallic Compounds*. IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans, vol. 23. Lyon, France: International Agency for Research on Cancer. pp. 325-415.

IARC. 1987. Lead and lead compounds. In *Overall Evaluations of Carcinogenicity.* IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans, suppl 7. Lyon, France: International Agency for Research on Cancer. pp. 230-232.

IARC. 1990. Chromium and chromium compounds. In *Chromium, Nickel, and Welding*. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, vol. 49. Lyon, France: International Agency for Research on Cancer. pp. 49-256.

Lanphear BP, Matte TD, Rogers J, Clickner RP, Dietz B, Bornschein RL, *et al.* 1998. The contribution of lead-contaminated house dust and residential soil to children's blood lead levels. A pooled analysis of 12 epidemiologic studies. *Environ Res* 79(1): 51-68.

Lide DR, Frederikse HPR, eds. 1998. *CRC Handbook of Chemistry and Physics*. New York: CRC Press.

NTP. 2003. *Report on Carcinogens Background Document for Lead and Lead Compounds*. National Toxicology Program. http://ntp.niehs.nih.gov/ntp/newhomeroc/roc11/Lead-Public.pdf.

Stauber JL, Florence TM, Gulson BL, Dale LS. 1994. Percutaneous absorption of inorganic lead compounds. *Sci Total Environ* 145(1-2): 55-70.

Staudinger KC, Roth VS. 1998. Occupational lead poisoning. *Am Fam Physician* 57(4): 719-726, 731-732.

Steenland K, Boffetta P. 2000. Lead and cancer in humans: where are we now? *Am J Ind Med* 38(3): 295-299.

TRI. 2009. *TRI Explorer Chemical Report.* U.S. Environmental Protection Agency. http://www.epa.gov/triexplorer/ and select Lead.

USGS. 2009. Lead statistics. In *Historical Statistics for Mineral and Material Commodities in the United States.* U.S. Geological Survey. Last updated: 11/5/09. http://minerals.usgs.gov/ds/2005/140/lead.pdf.

USGS. 2010. Lead. In *Mineral Commodity Summaries*. U.S. Geological Survey. http://minerals.usgs.gov/minerals/pubs/commodity/lead/mcs-2010-lead.pdf. 2 pp.

USITC. 2003. *USITC Interactive Tariff and Trade DataWeb.* United States International Trade Commission. http://dataweb.usitc.gov/scripts/user_set.asp and search on HTS nos. 283670 and 2824. Last accessed: 2003.

Waalkes MP, Diwan BA, Ward JM, Devor DE, Goyer RA. 1995. Renal tubular tumors and atypical hyperplasias in B6C3F$_1$ mice exposed to lead acetate during gestation and lactation occur with minimal chronic nephropathy. *Cancer Res* 55(22): 5265-5271.