OHSAH

TOXICOLOGY PROFILE

Lead (Pb) IN THE HEALTH CARE INDUSTRY

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Summary

This report includes information related to lead, including its chemical and physical properties, analytical methods, exposure routes, health effects, exposure limits, regulations, and control measures.

Element lead is a kind of heavy metal which was used in manufacturing pipes and storage batteries. Organic lead was an additive to gasoline, but was banned in Canada and internationally wide since 1980s, and its level in the environment has decline significantly. Workers may be exposed to lead through inhalation, dermal contact and ingestion.

The main target for lead toxicity is the nervous system. It may also cause weakness in fingers, wrists, or ankles. Long-term exposure of adults can result in decreased performance in some tests that measure functions of the nervous system. Lead has effect on the cellular component of the immune system. Lead is one proven animal carcinogen, and it increased cancer rate in many epidemiology studies. In addition, lead has proven reproductive effects, such as pre-term delivery in women, and in alterations in sperm and decreased fertility in men. Lead also has developmental effects on anthropometric indices and sexual maturation.

The preferred control measures of occupational exposure to lead is engineering methods, including isolation and ventilation. In addition, there are Canadian and US national standard concerning the establishment of personal protective equipment program against occupational exposure to lead.

Part 1 General Information

Name

Lead (Pb)

CAS Number

7439-92-1

Chemical and Physical Properties

Lead is a heavy, low melting, bluish-gray metal that occurs naturally in the Earth's crust [9]. Lead exists in three oxidation states: Pb(0), the metal; Pb(II); and Pb(IV). In the environment, lead primarily exists as Pb(II) [9]. The extensive use of lead is largely due to its low melting point and excellent corrosion resistance in the environment.

Measurement Methods

Standard measurement methods for lead are summarized in **table 1** as below.

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Table 1 Standard measurement method for lead

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	Sampling	Analysis	LOD [A]	Ref.
U.S.OSHA	Personal sampling pump with mixed-cellulose ester (MCE) membrane filter and back-up pad (BUP) contained in a polystyrene cassette	Open Vessel Microwave Digestion/ICP-MS Analysis	0.0042 ppb	[16]
	0.8-μm mixed-cellulose ester (MCE) filter and backup pad using a calibrated personal sampling pump	Digested and analyzed by atom adsorption spectrometry (AAS)		[17]
	Air samples are collected using mixed-cellulose ester filters using a calibrated sampling pump.	Samples are desorbed or digested using water extractions or mineral acid digestions. Elemental analysis of the prepared sample solutions is performed by atomic absorption or emission spectroscopy	0.01 μg/ml (LOQ [B])	[18]
	A calibrated personal sampling pump is used to draw a known volume of air through a mixed-cellulose ester membrane filter contained in a styrene cassette.	Filters are digested with nitric acid, sulfuric acid and hydrogen peroxide. Dissolution of the elements is facilitated by addition of hydrochloric acid. Analysis is performed using Inductively Coupled Argon Plasma-Atomic Emission Spectroscopy (ICAP-AES).	2.1 μg per sample	[19]

A personal sampling pump is used to draw a known volume of air through a mixed cellulose ester membrane filter contained in a polystyrene cassette. Wipe (smear tab) and bulk material are collected by grab sampling techniques.	Filters are digested with hydrochloric and nitric acids. Analysis is performed using Inductively Coupled Plasma-Atomic Emission Spectroscopy.	305 μg per sample	[20]
Filter (0.8μm cellulose ester membrane)	Flame atomic absorption spectrophotometer	2.6μg/sample	[11]
Filter (0.8μm, 37-mm, mixed cellulose ester membrane)	X-Ray fluorescence (XRF), portable L-Shell excitation	17 to 1500μg/ sample	[12]
Filter (0.8μm cellulose ester membrane)	Graphite furnace atomic absorption spectrometry	0.02μg/ sample	[13]
Filter (37-mm, 0.8μm pore, mixed cellulose ester membrane)	Portable anodic stripping voltammetry	0.09μg/ sample	[14]
Cyclone with filter (10-mm nylon or Higgins Dewell cyclone + 5-μm PVC membrane)	X-ray powder diffraction (for lead sulfide only)	5 μg/sample	[21]
Filter (0.8-μm, cellulose ester membrane, or 5.0-μm, polyvinyl chloride membrane)	ICP-AES	2.5 ng/ml	[84]
Filter (0.8-:m, cellulose ester membrane)	ICP-AES	0.023 μg/ml	[85]
Wipe	ICP-AES	Not determined	[86]
Filter (0.8-μm cellulose ester membrane)	Chemical spot test kit (Rhodizonate-based)	Not given	[87]
Use gloves, templates and pads to collect surface samples	Screening of all samples by flame AAS or ICP, followed by graphite furnace AAS for those samples giving "Not Detected" is an efficient scheme.	2 μg/sample for AAS or ICP; 0.1 μg/sample for graphite furnace AAS	[88]
Wipe sample of human skin	Chemical spot test, Rhodizonate-based solution or spot test kit applied to wipe samples	Positive in the range of 5 -15 μg/sample.	[89]

(Left column label, vertical: U.S. NIOSH)

[A] Limit of Detection
[B] Limit of Quantification

Part 2 Exposure

(I) Use of Lead in Industry

Metallic lead is resistant to corrosion (i.e., not easily attacked by air or water). When exposed to air or water, thin films of lead compounds are formed that protect the metal from further attack. Lead can be combined with other metals to form alloys, and lead and lead alloys are commonly found in pipes, storage batteries, weights, shot and ammunition, cable covers, and sheets used to shield us from radiation. The largest use for lead is in storage batteries in cars and other vehicles [9]. Lead compounds are used as a pigment in paints, dyes, and ceramic glazes and in caulk. The amount of lead used in these products has been reduced in recent years. Organic lead, such as tetraethyl lead and tetramethyl lead were once used in North America as gasoline additives to increase octane rating. However, with the introduction of unleaded gasoline in Canada in 1975, lead concentrations in the air have declined significantly, falling 76% between 1973 and 1985 [83]. Leaded gasoline in cars was banned in Canada in 1990. Since then levels of lead in the air of most Canadian cities have dropped below detectable limits [83].

(II) Exposure Routes

Inhalation
Inorganic lead in ambient air consists of aerosols of particulates that can be deposited in the respiratory tract when the aerosols are inhaled [9]. Amounts of lead deposited in the respiratory tract depend on the size of inhaled particles, breathing patterns (e.g., nose breathing vs. mouth breathing), airway geometry, and air-stream velocity within the respiratory tract [22]. It is estimated that 95% of deposited inorganic lead that is inhaled as submicron particles is absorbed [23,24]. Both human and animal studies showed that *organic lead* is absorbed through inhalation rapidly and near completion [25-27].

Oral
Gastrointestinal absorption of water-soluble lead is higher in children than in adults: dietary balance studies conducted in infants and children showed approximately 40-50% of ingested lead is absorbed [28,29], by comparison, in adults, the estimated absorption rate ranged from 3 to 10% [30-33].

Ingestion
Lead ingestion is possible when eating food or drinking water that contains lead. Water pipes in some older homes may contain lead solder. Lead can leach out into the water.

Part 3 Health Effects

(I) Death

Cohort studies showed that mortality rate among workers occupationally exposed to lead (mean blood-Pb ranged from 63 µg/dL to 80 µg/dL) was significantly greater than general population [37,38]. The increased mortality cases results in large part from malignant neoplasms, chronic renal diseases, hypertension, nephritis and so on.

(II) Immunological Effects

There are lots of studies concerning the effect of lead exposure on immunological parameters in lead workers as well as general population. Although these studies showed mixed results, they gave certain indication that lead may have an effect on the cellular component of the immune system. Results of some of the studies are summarized in **table 2** as below.

Table 2 Studies concerning the immunological effects of lead

Study Group	Lead Exposure Level	Immunological Effects	Reference
Lead workers	Mean blood-Pb 59 µg/dL, ranged from 21 to 90 µg/dL.	Statistically significantly more colds and influenza infections per year, as well suppression of serum IgM compared with reference group.	[39]
Lead workers occupationally exposed for 4-30 yrs	Mean blood-Pb 38.4 µg/dL (range: 25-53 µg/dL)	Serum concentration of IgG, IgA and IgM not significantly different from reference group.	[40]
145 lead-exposed male workers	Median blood-Pb 39 µg/dL (range: 25-55 µg/dL)	No significant difference in serum immunoglobulin levels compared with unexposed worker.	[41]
3 group of lead workers	The three groups of workers had blood-Pb of 6.5, 17.8, and 128 µg/dL respectively.	Inhibited lymphocyte proliferation to phytohemagglutinin (PAH), but no abnormalities on natural killer cell activity.	[42]
Lead workers	Mean blood-Pb of 74.8 µg/dL.	Decreased T-cell subpopulation ($CD4^+$).	[43]
71 male workers in the manufacturing of lead stearate	Mean blood-Pb of 19 µg/dL.	No significant alternation in the number or percentages of $CD4^+$ or $CD3^+$ T-cells.	[44]

(III) Neurological Effects

Encephalopathy

Lead encephalopathy is the most severe neurological effect of lead among **adults**, which refers to various diseases that affect brain function [9]. Early syndromes within weeks of initial exposure include dullness, irritability, poor attention symptoms, headache, muscular tremor, loss of memory, and hallucination [9]. After the early syndromes, the conditions may worsen, sometimes abruptly, to delirium, convulsions, paralysis, coma, and death [45]. Severe signs of encephalopathy may occur in some adults at blood-Pb levels ranging from approximately 50 to >300 µg/dL, which are extremely high [46].

Neurobehavioral Effects

There are numerous case reports and small cohort studies which associate occupational lead exposure with abnormalities in neurobehavioral syndromes, including malaise, forgetfulness, irritability, lethargy, headache, fatigue, impotence, decreased libido, dizziness, weakness, and paresthesia at blood-Pb levels ranging from 40 to 120 µg/dL [47-50]. One study [51] found that lead workers exhibited greater levels of conflict in interpersonal relationships compared with unexposed workers. Another study [48] showed that lead workers with blood-Pb of 45-60 µg/dL performed much worse than workers with lower blood-Pb on neurobehavioral tests, with impaired ability to perform cognitive and visual-motor coordination tasks. Other studies showed other neurobehavioral effects of lead, such as deficits in hand-eye coordination and delayed response time [52], disturbances in hand dexterity and IQ test [53], and impaired verbal concept formation and memory [54].

Peripheral Physiological Effects

There are many studies concerning the effect of lead exposure on peripheral nerve function, which is quantified by measuring the conduction velocity of electrically stimulated nerves in the arm or leg of lead workers. Some of the studies are summarized in **table 3** as below. The results of these studies indicate that NCV effects occur in adults at PbBs <70 µg/dL, and possibly as low as 30 µg/dL.

Table 3 Studies concerning the peripheral physiological effects of lead

Exposed Workers	Peripheral Physiological Effects	Reference
High-exposure workers after 1 year of exposure, whose blood-Pb level ranged from 30 to 48 µg/dL.	Decreased nerve conduction velocities (NCVs) in the median and ulnar nerves	[54]
55 lead workers with blood-Pb ranging from 60 to 80 µg/dL.	Decreased NCVs in ulnar and peroneal nerves.	[55]
70 female and 58 male ceramic painters with blood-Pb level of 2.1-69.5 µg/dL.	No significant association was found between blood-Pb level and median NCVs.	[56]
Workers with blood-Pb >70 µg/dL	Decreased NCVs of ulnar and median nerves	[57]

72 male workers from a lead-battery manufacturing factory and 83 unexposed referents, whose mean blood-Pb levels were 36.9 μg/dL and 10.5 μg/dL respectively.	Decreased NCVs in the third test of the serial measurements.	[58]

(IV) Carcinogenicity

Human Studies

Almost all information regarding lead exposure and cancer in humans is derived from studies of lead workers exposed to inorganic lead. Results of some of these studies are summarized in **table 4** as below. Lead increased rate of cancer of the digestive tract, respiratory tract, bladder and kidney in some of the studies.

Table 4 Human studies concerning lead's carcinogenicity

Exposed Workers	Carcinogenicity Effects	Reference
Cohort study of 1,898 retired lead acid battery workers during 1925-1976 in UK.	The only significant finding regarding cancer was a small but significant excess of malignant neoplasms of the digestive tract among workers with highest lead exposure.	[59]
Same cohort as the above study.	No association between lead exposure and deaths from malignant neoplasms.	[60]
4,519 battery-plant workers.	An increased standard mortality rate SMR was found to be statistically significant, attributed to digestive and respiratory cancers. No adjustment was made for other concomitant occupational exposure or smoking.	[38]
Case-control study at a battery plant that had 30 stomach cancer deaths.	No association was found between lead exposure and stomach cancer.	[61]
437 Swedish smelter workers with verified high lead exposure for at least 3 years.	An increased SMR only for lung cancer, but not statistically significant.	[62]
A follow-up study of 1,992 workers at the same smelter as the above study.	An increased SMR for all malignancies among a group with the highest level of exposure (1.5, 95% CI 0.8-2.4), and a much higher SMR for lung cancer (4.1, 95% CI 1.5-9.0).	[63]
20,700 Finnish workers exposed to lead during 1973-1983.	A 1.4-fold increase in the overall cancer incidence and a 1.8-fold increase in the incidence of lung cancer among workers who had ever had a blood-Pb ≥21 μg/dL were found.	[64]
1,388 workers of a lead-smelting plant in Italy.	By comparing with the national mortality rates, stomach cancer and lung cancer were significantly decreased, while deaths from cancer of the liver and biliary tract, bladder cancer and kidney cancer were increased insignificantly.	[65]
Workers exposed to tetraethyl lead.	A statistically significant association was found between exposure to this compound and rectal cancer (odds ratio 3.7, 95% CI 1.3-10.2).	[66]
Meta-analysis of lead-workers studies focusing on overall cancer.	A significant excess risk of overall cancer, stomach cancer, kidney cancer and bladder cancer was found.	[67]

Animal Studies

The available data on the carcinogenicity of lead following ingestion by laboratory animals indicate that lead is carcinogenic, and that the most common tumors that develop are renal tumors [1-3]. The mechanism of lead-induced carcinogenicity in animals is not known, but some non-genotoxic mechanisms that have been proposed include inhibition of DNA synthesis and repair, alterations in cellto-cell communication, and oxidative damage [4].

(V) Reproductive Effects

The available evidence suggest that occupational and environmental exposure resulting in moderately high blood-Pb might result in abortion and pre-term delivery in women, and in alterations in sperm and decreased fertility in men.

Reproductive Effects in Females

Studies concerning the reproductive effects of lead in females are summarized in **table 5** as below.

Table 5 Studies concerning the reproductive effects of lead in females

Exposed Group	Reproductive Effects	Reference
Female workers at a lead smelter in Sweden	Among 294 workers who were employed at the smelter during pregnancy, 13.9% ended in spontaneous abortion, among 176 pregnancies employed at the smelter prior to pregnancy, 17% ended in spontaneous abortion. Both groups have a abortion rate higher than general population.	[5]
Nested control-case study of a cohort of 668 pregnant women in Mexico City	The risk of spontaneous abortion increased with increasing blood-Pb level. There was a 1.13-fold increase in the risk of spontaneous abortion per µg/dL increase in blood-Pb.	[6]
Cohort study of women living in Port Pirie, which was a lead smelter community in South Australia.	No association was found between blood-Pb and spontaneous abortions. Mean mid-pregnancy blood-Pb in and outside of the town were 10.6 and 7.6 respectively.	[7]
Same cohort as the above study	Rate of preterm delivery (delivery before the 37th week) was significantly higher in women living in the smelter town than in women not living in the town.	[8]
Pregnant females	Preterm births were almost 3 times more frequent in women with umbilical blood-Pb ≥5.1 µg/dL than in women with blood-Pb <5.1µg/dL.	[10]
121 women biologically monitored for exposure to lead at the Finnish Institute of Occupational Health	No alternations in the time-to-pregnancy or decreased fecundability was found.	[34]

Reproductive Effects in Males

Reproductive effects of lead exposure among males includes reduced fertility, lowered sperm quality, reduced serum testosterone. Studies concerning lead's reproductive effects in males are summarized in **table 6** as below.

Table 6 Studies concerning reproductive effects of lead in males

Exposed Group	Reproductive Effects	Reference
2,111 Finnish workers occupationally exposed to inorganic lead, and reference group was 681 unexposed one	Significant reduction in fertility relative to the unexposed group was found. And the risk ratio (RR) for infertility in exposed men appeared to increase with increasing blood-Pb level.	[35]
74 exposed workers with mean blood-Pb of 46.3 μg/dL, and control group was 138 men with blood-Pb of 10.4μg/dL	A significant reduction in fertility was observed in the exposed group.	[36]
163 Taiwanese male lead battery workers	Decreased fertility in men with PbB in the range of 30–39 and ≥40 μg/dL was found, but there was no significant reduction in fertility in men with PbB of ≤29 μg/dL.	[68]
81 lead smelter workers	An association between blood-Pb and sperm concentration was found; in addition, a reduction in serum testosterone with increasing semen lead concentration was observed.	[69]
149 industrial workers in Zagreb, Croatia	98 men who had moderate occupational exposure to lead (mean blood-Pb of 36.7 μg/dL) had significantly lower sperm density and lower counts of total motile and viable sperm.	[70]
503 European workers	The study showed a 49% reduction in the median sperm concentration in men with blood-Pb ≥50 μg/dL, whereas there was no significant difference in sperm concentration between the reference group of men (mean blood-Pb ≤10 μg/dL) and men with mean blood-Pb of 10–50 μg/dL	[71]

(VI) Developmental Effects

Anthropometric Indices

There are a number of epidemiological studies which reveal the association between blood-Pb and anthropometric dimensions since the first case report of lead-poisoned children in 1929, and some of them are summarized in **table 7** as below.

Table 7 Studies concerning the relationship between blood-Pb level and anthropometric dimension

Exposed Group	Anthropometric Indices	Reference
1-month-old Mexican infants	Infant blood-Pb level was inversely associated with weight gain, with an estimated decline of 15.1 grams per µg/dL	[72]
Children aged 18-36 months with a mean blood-Pb of 6.4 µg/dL	Blood-Pb was inversely related with head circumference	[73]
148 Russian mothers and 114 Norwegian mothers with maternal and cord blood-Pb levels as low as 1.2 µg/dL	Blood-Pb had a negative impact on birth weight and child's body mass index (BMI, weight in kg divided by the square of the height in meters) with or without adjusting for gestational age	[74]
2,695 children ≤7 years old	Blood-Pb (range, 4–35 µg/dL) was a statistically significant predictor of children's height, weight, and chest circumference, after controlling for age, race, sex, and nutritional covariates	[75]
1,454 Mexican-American children aged 5–12 who were participants in the Hispanic Health and Nutrition Examination Survey	Blood-Pb in the range of 2.8–40 µg/dL were related with decreased stature	[76]

Sexual Maturation

Two studies concerning the effect of lead exposure on sexual maturation are summarized in **table 8** as below. IN both studies, lead caused delayed sexual maturation.

Table 8 Studies concerning the effect of lead exposure on sexual maturation

Exposed Group	Developmental Effects in terms of Sexual Maturation	Reference
2,741 U.S. female children and adolescents, ages 8–18 years	Increasing blood-Pb was significantly associated with decreasing stature (height) and delayed sexual development	[77]
1,706 girls 8–16 years old with blood-Pb ranging from 0.7 to 21.7 µg/dL	A significant and negative association between blood-Pb and delayed sexual maturation was found	[78]

Part 4 Regulations and Guidelines

(I) Exposure Limit

WorkSafeBC

WorkSafeBC exposure limit and notations for lead and lead compounds are summarized in **table 9** as below.

Table 9 WorkSafeBC exposure limit and notations for lead and lead compounds

Substance		Formula	CAS No.	TWA	STEL/Ceiling	Notations [A]
Lead (elemental and inorganic)		Pb	7439-92-1	0.05 mg/m^3	N/A	Elemental: 2B (possibly carcinogenic to humans)
						Other inorganic: 2A (probably carcinogenic to humans); R (adverse reproductive effect)
Lead arsenate		$Pb_3(AsO_4)_2$	3678-31-8	0.15 mg/m^3	N/A	2A (probable human carcinogen); R (adverse reproductive effect)
Lead chromate	as Pb	$PbCrO_4$	7758-97-6	0.05 mg/m^3	N/A	A2 (Suspected human carcinogen); 2A (probably carcinogen to humans); R (adverse reproductive effect)
	as Cr			0.012 mg/m^3		

[A]: ACGIH notations A1 and A2 and IARC notations 1, 2A and 2B
[B]: Under section 5.57(1) of the OHS Regulation

US ACGIH

ACGIH exposure limits and notations for lead and lead chemicals are summarized in **table 10** as below.

Table 10 ACGIH exposure limit and notations for lead and lead compounds

Substance		TWA	STEL/Ceiling	BEI	Notations	TLV Basis
Lead (elemental and inorganic)		0.05 mg/m^3	N/A	Lead in blood: 30 µg/dL [A] (no critical sampling time)	A3: Confirmed animal carcinogen with unknown relevance to humans	Central nervous system (CNS) and peripheral nervous system (PNS) impair; hematologic effects
Lead arsenate		0.15 mg/m^3	N/A		2A (probable human carcinogen); R (adverse reproductive effect)	Gastrointestinal (GI) damage; CNS impair; kidney damage, hematologic effect
Lead chromate	as Pb	0.05 mg/m^3	N/A		A2 (Suspected human carcinogen)	Male reproductive damage; teratogenic effect; vasoconstriction
	as Cr	0.012 mg/m^3				

[A]: Women of child bearing potential, whose blood-Pb exceeds 10 µg/dL, are at risk of delivering a child with a blood-Pb over the current Centers for Disease Control guideline of 10 µg/dL. If the blood-Pb of such children remains elevated, they may be at increased risk of cognitive deficits. The blood-Pb of these children should be closely monitored and appropriate steps should be taken to minimize the child's exposure to environmental lead.

Exposure limit by other jurisdictions

Exposure limits set up by US OSHA and NIOSH are summarized in **table 11** as below.

Table 11 OSHA and NIOSH exposure limits for lead and lead compounds

US OSHA	US NIOSH

50 µg/m^3 averaged over an 8-hour period is set up for lead ("Lead" means metallic lead, all inorganic lead, and a class of organic compounds called soaps; all other lead compounds are excluded from the definition). For work shifts more than 8 hours, maximum PEL equals 400 divided by hours worked in the day (µg/m^3).
 	
For other forms of lead, OSHA PELs are as follows:
    8hr TWA for general industry for tetraethyl lead: 0.075 mg/m^3
    8hr TWA for construction industry for tetraethyl lead: 0.1 mg/m^3
    8hr TWA for shipyard industry for tetraethyl lead: 0.1 mg/m^3
 	
Other OSHA requirements can be found in 29 CFR 1910.1025
	NIOSH REL for lead (8-hour TWA) is 0.05 mg/m^3, and air concentrations should be maintained so that worker blood lead remains less than 0.060 mg per 100 g of whole blood ("Lead" refers to metallic lead, lead oxides, and lead salts, which include organic salts such as lead soaps but excluding lead arsenate).
	
	An IDLH is 100 mg/m^3 is set up for airborne lead.

(II) Hazard Classification

Information on hazard classification by different agencies is summarized in **table 12** as below.

Table 12 Hazard classification of lead

Agency	Description	Reference
Canadian WHMIS hazard criteria	Lead meets the following criteria: D2A - Poisonous and infectious material - Other effects - Very toxic D2B - Poisonous and infectious material - Other effects - Toxic	[79]
European Union (EU) Classification and Labeling Information System	It meets the EU criteria for class(es): Toxic reproduction, Category 1; Toxic to Reproduction, Category 3; Harmful; Danger of cumulative effects. EU risk phrases for lead: May cause harm to the unborn child. Possible risk of impaired fertility. Also, harmful by inhalation and if swallowed. Danger of cumulative effects. EU safety phrases for lead: Avoid exposure - obtain special instruction before use. In case of accident or if you feel unwell, seek medical advice immediately (show label where possible). Safety phrases relate to the highest concentration division indicated, but may also be applicable to lower concentrations.	[79]

	EU comments **Concentration greater than or equal to 5%:** Toxic: May cause harm to the unborn child. Possible risk of impaired fertility. Also, harmful by inhalation and if swallowed. Danger of cumulative effects. **Concentration greater than or equal to 1% and less than 5%:** Toxic: May cause harm to the unborn child. Also, harmful by inhalation and if swallowed. Danger of cumulative effects. **Concentration greater than 0.5% and less than 1%:** Toxic: May cause harm to the unborn child. Danger of cumulative effects.	
US EPA	Designated as hazardous substances in accordance with Section 311(b)(2)(A) of the Clean Water Act	EPA 2005a 40 CFR 116.4
	National primary drinking water standards: MCLG ----0; MCL ----- Treatment technique; Action level: 0.015 mg/L	EPA 2002
	Designated to be hazardous air pollutant	EPA 2004b 42 USC 7412
	National primary and secondary ambient air quality standards: 1.5 µg/m3	EPA 2005b 40 CFR 50.12
	Reportable quantities of hazardous substances designated pursuant to Section 311 of the Clean Water Act: 10 pounds	EPA 2005c substances designated pursuant to 40 CFR 117.3

	Residential lead hazards standards:
	  Floors 40 µg/ft^2 Interior window sills: 250 µg/ft^2
	  Bare soil in children's play areas: 400 ppm Bare soil in rest of yard: 1,200 ppm average
		EPA 2005l TSCA Section 403

	EPA IRIS
	  Carcinogenicity classification: Group B2: Probable human carcinogen
	  Reference Concentration (RfC): Not available
	  Reference Dose (RfD): Not appicable
		IRIS 2005

US National Toxicology Program (NTP)	Carcinogenicity classification: Reasonably anticipated to be human carcinogens	NTP 2005

US FDA	Action level (µg/mL leaching solution)
	  Ceramic ware
	    Flatware (average of 6 units): 3.0 µg/mL
	    Small hollowware (other than cups and mugs) (any 1 of 6 units): 2.0 µg/mL
	    Large hollowware (other than pitchers) (any 1 of 6 units): 1.0 µg/mL
	    Cups and mugs (any 1 of 6 units) and pitchers (any 1 of 6 units): 0.5 µg/mL
	  Silver-plated hollowware
	    Product intended for use by adults (average of 6 units): 7 µg/mL
	    Product intended for use by infants and children (any 1 of 6 units): 0.5 µg/mL
	    Bottled drinking water 0.005 mg/L
		FDA 2004
		21 CFR 165.110

US OSHA	Lead meets criteria for hazardous material	29 CFR 1910.1200

Part 5 Control Measures

(I) Engineering Controls

Engineering methods to control hazardous conditions are preferred [79]. Engineering controls involve the installation and use of equipment, facilities, or modifications to work procedures to either protect workers from exposure or reduce exposure to industrial contaminants [81]. Engineering control measures of lead may include:

Isolation
Because of the high potential hazard associated with this substance, stringent control measures such as enclosure or isolation may be necessary. Setting up certain aspects of the range which are restricted to authorized personnel. For example, access to the maintenance area behind the range where the backstops are located is locked.

Avoid generating dusts
Prevent the release of dust into workplace air. Use the proper tools to open containers. Ripping open a container can cause an uneven tear, thus making spills more likely. Cover work surfaces with compatible, chemical resistant and/or disposable material for easier containment and clean-up of spills [80].

Ventilation
Use a ventilation system separate from other exhaust ventilation systems. Exhaust directly to the outside. Locate dust collectors outside or where permitted by regulation. Supply sufficient replacement air to make up for air removed by exhaust systems [80].

(II) Training

Employees who perform duties that involve the removal or disturbance of lead or lead contained materials (LCMs) or that are required to work in environments contaminated with lead dust shall be provided training annually, and the training should contain the following elements:

(I) The requirement of WorkSafeBC OHS Regulations concerning lead exposure.

(II) Potential health consequences following overexposure to lead.

(III) Initial syndromes of lead intoxication.

(IV) The specific operations that could result in an exposure to lead above the exposure limit, and the work practices and procedures that they are to follow to limit their exposure.

(V) Discussion of the importance of personal hygiene practices in reducing lead exposure.

(VI) Instruction about the use and care of appropriate protective equipment, including protective clothing and respiratory protection.

(VII) Other health & safety issues such as incompatible materials of lead, spill management, how to avoid damage to lead containers and so on.

(III) Personal Protective Equipments

If engineering controls and work practices are not effective in controlling exposure to this material, then wear suitable personal protection equipment including approved respiratory protection. Appropriate equipment should be made available for use in emergencies such as spills or fire [82].

Respirator

Canadian Standards Association has set up CSA Standard Z94.4-93 concerning the respiratory protection program to control lead exposure, which includes selection, fit testing, training, maintenance and inspection. US NIOSH also provides recommendations for controlling airborne lead as follows:

Up to 0.5 mg/m^3: Respirator with high-efficiency particulate filter(s); or supplied air respirator (SAR).

Up to 1.25 mg/m^3: SAR operated in a continuous-flow mode; or powered air- purifying respirator with high-efficiency particulate filter.

Up to 2.5 mg/m^3: Full-facepiece respirator with high-efficiency particulate filter(s); or SAR with a tight-fitting facepiece operated in a continuous-flow mode; or powered air-purifying respirator with tight-fitting facepiece and high-efficiency particulate filter; or full-facepiece self contained breathing apparatus (SCBA); or full-facepiece SAR.

Up to 50 mg/m^3: Positive pressure SAR.

Up to 100 mg/m^3: Positive pressure, full-facepiece SAR.

Emergency or planned entry into unknown concentrations or IDLH conditions (100 mg/m^3):
Positive pressure, full-facepiece SCBA; or positive pressure, full-facepiece SAR with an auxiliary positive pressure SCBA.
ESCAPE: Full-facepiece respirator with high-efficiency particulate filter(s); or escape-type SCBA.

Eye/face Protection

CCOHS recommends that chemical safety goggles or face shield may be necessary [82].

Skin Protection

CCOHS recommends chemical protective gloves, coveralls, boots, and/or other protective clothing to prevent skin contact [82].

References

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