Lead and compounds (inorganic); CASRN 7439-92-1

Human health assessment information on a chemical substance is included in the IRIS database only after a comprehensive review of toxicity data, as outlined in the IRIS assessment development process. Sections I (Health Hazard Assessments for Noncarcinogenic Effects) and II (Carcinogenicity Assessment for Lifetime Exposure) present the conclusions that were reached during the assessment development process. Supporting information and explanations of the methods used to derive the values given in IRIS are provided in the guidance documents located on the IRIS website.

STATUS OF DATA FOR Lead and compounds (inorganic)

File First On-Line 03/01/1988

Category (section)	Assessment Available?	Last Revised
Oral RfD (I.A.)	qualitative discussion	07/08/2004
Inhalation RfC (I.B.)	not evaluated	
Carcinogenicity Assessment (II.)	yes	09/26/1988

I. Chronic Health Hazard Assessments for Noncarcinogenic Effects

I.A. Reference Dose for Chronic Oral Exposure (RfD)

Substance Name — Lead and compounds (inorganic)
CASRN — 7439-92-1
Section I.A. Last Revised — 07/08/2004

In general, the oral Reference Dose (RfD) is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. The RfD is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis and is expressed in units of mg/kg-day. Please refer to the guidance documents at http://www.epa.gov/iris/backgrd.html for an elaboration of these concepts. Since RfDs can be derived for the noncarcinogenic health effects of substances that are also carcinogens, it is

essential to refer to other sources of information concerning the carcinogenicity of this chemical substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.A.1. Oral RfD Summary

Health effects associated with exposure to inorganic lead and compounds include, but are not limited to, neurotoxicity, developmental delays, hypertension, impaired hearing acuity, impaired hemoglobin synthesis, and male reproductive impairment. Importantly, many of lead's health effects may occur without overt signs of toxicity. Lead has particularly significant effects in children, well before the usual term of chronic exposure can take place. Children under 6 years old have a high risk of exposure because of their more frequent hand-to-mouth behavior (Centers for Disease Control and Prevention (CDC), 1991: http://www.cdc.gov/nceh/lead/publications/books/plpyc/contents.htm).

EPA considered providing an RfD for inorganic lead in 1985, and concluded that it was inappropriate to develop an RfD, as documented online in the following statement in 1988:

A great deal of information on the health effects of lead has been obtained through decades of medical observation and scientific research. This information has been assessed in the development of air and water quality criteria by the Agency's Office of Health and Environmental Assessment (OHEA) in support of regulatory decision-making by the Office of Air Quality Planning and Standards (OAQPS) and by the Office of Drinking Water (ODW). By comparison to most other environmental toxicants, the degree of uncertainty about the health effects of lead is quite low. It appears that some of these effects, particularly changes in the levels of certain blood enzymes and in aspects of children's neurobehavioral development, may occur at blood lead levels so low as to be essentially without a threshold. The Agency's RfD Work Group discussed inorganic lead (and lead compounds) at two meetings (07/08/1985 and 07/22/1985) and considered it inappropriate to develop an RfD for inorganic lead.

EPA is not providing a review of current literature concerning the health effects of lead at this time (June, 2004), given the ongoing effort of the CDC to re-evaluate the blood lead level of concern (CDC, 2004: http://www.cdc.gov/nceh/lead/spotLights/changeBLL.htm). A screening-level review of the more recent toxicology literature pertinent to noncancer effects associated with oral exposure to Lead and compounds (inorganic) was conducted by an EPA contractor in September 2002, and identified one or more significant new studies since the 1985 assessment. IRIS users may request the references for those studies from the IRIS Hotline at hotline.iris@epa.gov or (202)566-1676.

Current knowledge of lead pharmacokinetics indicates that risk values derived by standard procedures would not truly indicate the potential risk, because of the difficulty in accounting for pre-existing body burdens of lead. Lead bioaccumulates in the body, primarily in the skeleton. Lead body burdens vary significantly with age, health status, nutritional state, maternal body burden during gestation and lactation, etc. For this reason, and because of the continued apparent lack of threshold (CDC, 2004: http://www.cdc.gov/nceh/lead/spotLights/changeBLL.htm), it is still inappropriate to develop reference values for lead.

The purpose of this 2004 update is to assist IRIS users in finding relevant risk information and risk assessment methods that have been developed within EPA for media-specific applications. Most of these methods focus on blood lead levels, through the development of several media-specific lead exposure levels for risk management and regulatory purposes. Further information on these lead risk assessment methods and tools is described in Section 1.A.4.

I.A.2. Principal and Supporting Studies (Oral RfD)

Not applicable.

I.A.3. Uncertainty and Modifying Factors (Oral RfD)

Not applicable.

I.A.4. Additional Studies/Comments (Oral RfD)

As noted above, EPA is not providing a review of current literature at this time. In addition to information developed by CDC and the Agency for Toxic Substances and Disease Registry (ATSDR), described below, EPA has developed a number of lead exposure limits in support of regulatory decision-making, and has developed methods to assess risk from lead in situations not covered by these limits, also described below.

The CDC identified 10 µg/dL as the blood lead level of concern in children in their 1991 report "Preventing Lead Poisoning in Young Children" (http://www.cdc.gov/nceh/lead/publications/books/plpyc/contents.htm), and provided risk management options for categories of blood lead levels higher than 10 µg/dL. In view of the fact that health effects were and continue to be identified below the level of concern, the CDC convened an Advisory Committee on Childhood Lead Poisoning Prevention to consider whether the level of concern should be changed. The Advisory Committee's progress reports can be found at http://www.cdc.gov/nceh/lead/ACCLPP/acclppmain.htm. At this time, CDC has not changed the blood lead level of concern for three reasons (March 23, 2004, http://www.cdc.gov/nceh/lead/spotLights/changeBLL.htm:

- "No effective clinical interventions are known to lower blood lead levels for children with levels less than 10 µg/dL or to reduce the risks for adverse developmental effects."
- "Children cannot be accurately classified as having blood lead levels above or below 10 µg/dL because of the inaccuracy inherent in laboratory testing."
- "Finally, there is no evidence of a threshold below which adverse effects are not experienced. Thus, any decision to establish a new level of concern would be arbitrary and provide uncertain benefits."

In addition to the ongoing CDC effort, the ATSDR is mandated by Congress under CERCLA (1980) and SARA (1986) to develop toxicological profiles for all hazardous waste sites on the National Priorities List (NPL) (PB/99/166704, NTIS PB-99-66704; http://www.atsdr.cdc.gov/toxprofiles/tp13.html). Lead is the most common toxic metal at these sites. The ATSDR Toxicological Profile for Lead was last updated in 1999.

EPA has developed a number of lead exposure levels in support of regulatory decision-making, which limit the amount of lead that can be present in various environmental media. Under the National Primary Drinking Water Regulations, EPA established a Maximum Contaminant Level (MCL) for lead in drinking water due to any source. Under the Toxic Substances Control Act (TSCA) as amended by the Residential Lead-Based Paint Hazard Reduction Act, EPA established standards for lead in paint, house dust, and outdoor soil. The following links provide more information:

Action level for lead in drinking water: http://www.epa.gov/safewater/mcl.html#mcls

Regulation for lead in paint housedust, and outdoor soil:
http://www.epa.gov/oppt/lead/pubs/leadhaz.htm

National ambient air quality standard: http://www.epa.gov/ttn/naaqs/

Note that additional relevant information will be found by following links provided at these websites, such as dose-response relationships relating blood lead levels to changes in IQ (children) and hypertension (men).

Due to the likelihood of simultaneous exposure to various sources of lead, EPA recommends a case-by-case evaluation of the relative contributions of relevant lead exposures in a particular setting. The Superfund Program has guidelines for identifying lead-contaminated soil (http://www.epa.gov/superfund/programs/lead/products.htm), and facilitates site-specific risk assessments at contaminated waste sites on the National Priority List, where the major sources of soil contamination are activities such as disposal of mining and manufacturing wastes contaminated with lead and deposition of lead from air, rather than presence of lead-based paint in residences (see: http://www.epa.gov/superfund/programs/lead/trwhome.htm). The Superfund

Program uses the Integrated Exposure Uptake Biokinetic (IEUBK) Model for Lead in Children. The IEUBK Model provides estimates of the risk of elevated blood lead associated with particular combinations of relevant sources of lead exposure. This interactive model is described in more detail and provided for downloading at http://www.epa.gov/superfund/programs/lead/products.htm. Note that the IEUBK model was used in direct support of the lead-based paint regulation cited above.

The Superfund Program has also developed an Adult Lead Model, for use when lead exposures to adults, especially pregnant women, are of greater concern. This model is described and provided for downloading at http://www.epa.gov/superfund/programs/lead/adult.htm.

EPA maintains a lead resources page (http://www.epa.gov/opptintr/lead/pubs/resources.htm). Links are provided to many other EPA offices, government agencies, and other organizations involved in efforts to reduce exposure to lead in the environment.

I.A.5. Confidence in Risk Values

Not applicable.

I.A.6. EPA Documentation and Review of the Oral RfD

Agency Completion Date - 05/26/2004

I.A.7. EPA Contacts (Oral RfD)

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202)566-1676 (phone), (202)566-1749 (fax), or hotline.iris@epa.gov (email address).

I.B. Reference Concentration for Chronic Inhalation Exposure (RfC)

Substance Name — Lead and compounds (inorganic)
CASRN - 7439-92-1

In general, the Reference Concentration (RfC) is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily inhalation exposure of the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. The RfC considers toxic effects for both the respiratory system (portal-of-entry) and for

effects peripheral to the respiratory system (extrarespiratory effects). The inhalation RfC (generally expressed in units of mg/m3) is analogous to the oral RfD and is likewise based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis.

Inhalation RfCs are derived according to the *Interim Methods for Development of Inhalation Reference Doses* (EPA/600/8-88/066F, 1989) and subsequently, according to *Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry* (EPA/600/8-90/066F, 1994). Since RfCs can also be derived for the noncarcinogenic health effects of substances that are carcinogens, it is essential to refer to other sources of information concerning the carcinogenicity of this chemical substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.B.1. Inhalation RfC Summary

No RfC is available. See Section I.A. for additional information.

I.B.2. Principal and Supporting Studies (Inhalation RfC)

Not applicable.

I.B.3. Uncertainty and Modifying Factors (Inhalation RfC)

Not applicable.

I.B.4. Additional Studies/Comments (Inhalation RfC)

Not applicable.

I.B.5. Confidence in the Inhalation RfC

Not applicable.

I.B.6. EPA Documentation and Review of the Inhalation RfC

Agency Completion Date -- Not applicable.

I.B.7. EPA Contacts (Inhalation RfC)

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202)566-1676 (phone), (202)566-1749 (fax), or hotline.iris@epa.gov (email address).

II. Carcinogenicity Assessment for Lifetime Exposure

Substance Name — Lead and compounds (inorganic)
CASRN — 7439-92-1
Section II Last Revised — 09/26/1988

Section II provides information on three aspects of the carcinogenic assessment for the substance in question; the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative estimate in terms of either risk per µg/L drinking water or risk per µg/cu.m air breathed. The third form in which risk is presented is a concentration of the chemical in drinking water or air associated with cancer risks of 1 in 10,000, 1 in 100,000, or 1 in 1,000,000. The rationale and methods used to develop the carcinogenicity information in IRIS are described in the Risk Assessment Guidelines of 1986 (EPA/600/8-87/045) and in the IRIS Background Document. IRIS summaries developed since the publication of EPA's more recent Proposed Guidelines for Carcinogen Risk Assessment also utilize those Guidelines where indicated (Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

II.A. Evidence for Human Carcinogenicity

Note that the National Toxicology Program's Report on Carcinogens Review Committee has recommended that lead and lead compounds be considered "reasonably anticipated to be human carcinogens" (http://ntp-server.niehs.nih.gov/NewHomeRoc/roc11Bkgrnd2003.html; July 2003). Also, the International Agency for Research on Cancer (IARC) has undertaken a reevaluation of lead's carcinogenicity (http://monographs.iarc.fr/ENG/Monographs/vol87/index.php).

II.A.1. Weight-of-Evidence Characterization

Classification — B2; probable human carcinogen

Basis — Sufficient animal evidence. Ten rat bioassays and one mouse assay have shown statistically significant increases in renal tumors with dietary and subcutaneous exposure to several soluble lead salts. Animal assays provide reproducible results in several laboratories, in multiple rat strains with some evidence of multiple tumor sites. Short term studies show that lead affects gene expression. Human evidence is inadequate.

II.A.2. Human Carcinogenicity Data

Inadequate. There are four epidemiologic studies of occupational cohorts exposed to lead and lead compounds. Two studies (Dingwall-Fordyce and Lane, 1963; Nelson et al., 1982) did not find any association between exposure and cancer mortality. Selevan et al. (1985), in their retrospective cohort mortality study of primary lead smelter workers, found a slight decrease in the total cancer mortality (SMR=95). Apparent excesses were observed for respiratory cancer (SMR=111, obs=41, $p>0.05$) and kidney cancer (SMR=204, obs=6, $p>0.05$). Cooper and Gaffey (1975) and Cooper (1985 update) performed a cohort mortality study of battery plant workers and lead smelter workers. They found statistically significant excesses for total cancer mortality (SMR=113, obs=344), stomach cancer (SMR=168, obs=34), and lung cancer (SMR=124, obs=109) in the battery plant workers. Although similar excesses were observed in the smelter workers, they were not statistically significant. Cooper and Gaffey (1975) felt it was possible that individual subjects were monitored primarily on the basis of obvious signs of lead exposure, while others who showed no symptoms of lead poisoning were not monitored.

All of the available studies lacked quantitative exposure information, as well as information on the possible contribution from smoking. All studies also included exposures to other metals such as arsenic, cadmium, and zinc for which no adjustment was done. The cancer excesses observed in the lung and stomach were relatively small (<200). There was no consistency of site among the various studies, and no study showed any dose-response relationship. Thus, the available human evidence is considered to be inadequate to refute or demonstrate any potential carcinogenicity for humans from lead exposure.

II.A.3. Animal Carcinogenicity Data

Sufficient. The carcinogenic potential of lead salts (primarily phosphates and acetates) administered via the oral route or by injection has been demonstrated in rats and mice by more than 10 investigators. The most characteristic cancer response is bilateral renal carcinoma. Rats given lead acetate or subacetate orally have developed gliomas, and lead subacetate also produced lung adenomas in mice after i.p. administration. Most of these investigations found a carcinogenic response only at the highest dose. The lead compounds tested in animals are almost all soluble salts. Metallic lead, lead oxide and lead tetraalkyls have not been tested adequately. Studies of inhalation exposure have not been located in the literature.

Azar et al. (1973) administered 10, 50, 100, and 500 ppm lead as lead acetate in dietary concentrations to 50 rats/sex/group for 2 years. Control rats (100/sex) received the basal laboratory diet. In a second 2-year feeding study, 20 rats/group were given diets containing 0, 1000, and 2000 ppm lead as lead acetate. No renal tumors were reported in the control groups or in treated animals of either sex receiving 10 to100 ppm. Male rats fed 500, 1000, and 2000 ppm lead acetate had an increased renal tumor incidence of 5/50, 10/20, and 16/20, while 7/20 females in the 2000 ppm group developed renal tumors.

The Azar et al. (1973) study is limited by the lack of experimental detail. The possibility of environmental contamination from lead in the air or drinking water was not mentioned. The strain of rats used was not specified in the study, but the Health Effects Assessment for Lead (U.S. EPA, 1984) indicates the rats were Wistar strain. The weight gain at 1000 and 2000 ppm was reported to be depressed, but details were not given.

Kasprzak et al. (1985), in investigating the interaction of dietary calcium on lead carcinogenicity, fed 1% lead subacetate (8500 ppm Pb) to male Sprague-Dawley rats in the diet for 79 weeks. Of the rats surviving (29/30) in this treatment group beyond 58 weeks, 44.8% had renal tumors. Four rats had adenocarcinomas; the remaining nine had adenomas. Bilateral tumors were noted. No renal tumors were noted among the controls.

As part of a study to determine interactions between sodium nitrite, ethyl urea and lead, male Sprague-Dawley rats were given lead acetate in their drinking water for 76 weeks (Koller et al., 1986). The concentration of lead was 2600 ppm. No kidney tumors were detected among the 10 control rats. Thirteen of 16 (81%) lead-treated rats had renal tubular carcinoma; three tumors were detected at 72 weeks and the remainder detected at the termination of the study.

Van Esch and Kroes (1969) fed basic lead acetate at 0, 0.1%, and 1.0% in the diet to 25 Swiss mice/sex/group for 2 years. No renal tumors developed in the control group, but 6/25 male mice of 0.1% basic lead acetate group had renal tumors (adenomas and carcinomas combined). In the 1.0% group, one female had a renal tumor. The authors thought that the low incidence in the 1.0% group was due to early mortality.

Hamsters given lead subacetate at 0.5% and 1% in the diet had no significant renal tumor response (Van Esch and Kroes, 1969).

II.A.4. Supporting Data for Carcinogenicity

Lead acetate induces cell transformation in Syrian hamster embryo cells (DiPaolo et al., 1978) and also enhances the incidence of simian adenovirus induction. Lead oxide showed similar enhanced adenovirus induction (Casto et al., 1979).

Under certain conditions lead compounds are capable of inducing chromosomal aberrations in vivo and in tissue cultures. Grandjean et al. (1983) showed a relationship between sister chromatid exchange and lead exposure in exposed workers. Lead has been shown, in a number of DNA structure and function assays, to affect the molecular processes associated with the regulation of gene expression (U.S. EPA, 1986b).

II.B. Quantitative Estimate of Carcinogenic Risk from Oral Exposure

Not available.

Quantifying lead's cancer risk involves many uncertainties, some of which may be unique to lead. Age, health, nutritional state, body burden, and exposure duration influence the absorption, release, and excretion of lead. In addition, current knowledge of lead pharmacokinetics indicates that an estimate derived by standard procedures would not truly describe the potential risk. Thus, the Carcinogen Assessment Group recommends that a numerical estimate not be used.

II.C. Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure

Not available.

II.D. EPA Documentation, Review, and Contacts (Carcinogenicity Assessment)

II.D.1. EPA Documentation

Source Document — U.S. EPA, 1984, 1986, 1989

U.S. EPA, 1989 has received OHEA and SAB review.

The 1986 Air Quality Criteria Document for Lead has received Agency and External Review.

II.D.2. EPA Review (Carcinogenicity Assessment)

Verification Date — 05/04/1988

Screening-Level Literature Review Findings — A screening-level review of the more recent toxicology literature pertinent to the cancer assessment for Lead and compounds (inorganic), conducted by an EPA contractor in September 2002, did not identify any critical new studies.

IRIS users who know of important new studies may provide that information to the IRIS Hotline at hotline.iris@epa.gov or (202)566-1676.

II.D.3. EPA Contacts (Carcinogenicity Assessment

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202)566-1676 (phone), (202)566-1749 (fax), or hotline.iris@epa.gov (email address).

III. [reserved]
IV. [reserved]
V. [reserved]

VI. Bibliography

Substance Name — Lead and compounds (inorganic)
CASRN — 7439-92-1

VI.A. Oral RfD References

None.

VI.B. Inhalation RfC References

None.

VI.C. Carcinogenicity Assessment References

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Van Esch, G.J. and R. Kroes. 1969. The induction of renal tumors by feeding of basic lead acetate to mice and hamsters. Br. J. Cancer. 23: 265-271.

VII. Revision History

Substance Name — Lead and compounds (inorganic)
CASRN — 7439-92-1
File First On-Line 03/01/1988

Date	Section	Description
09/26/1988	II.	Carcinogen summary on-line
12/03/2002	I.A., II.D.2.	Screening-Level Literature Review Findings message has been added.
07/08/2004	I.A., I.B., II.	Previous reference value discussions replaced with links to EPA websites containing regulatory information and other websites with risk assessment information; cancer discussion retained, added links to agencies conducting reviews of lead carcinogenicity.

VIII. Synonyms

Substance Name — Lead and compounds (inorganic)
CASRN — 7439-92-1
Last Revised — 07/08/2004

- 7439-92-1
- Lead
- Lead and compounds
- plumbum