

**KAY CO. GRAND JURY  
SUBMISSION OF QUESTION**

I, Jessica Pepper, formally request the Kay Co. Grand Jury to consider the following question for review.

**Did the Oklahoma Department of Environmental Quality (ODEQ), violate the law and/or the rights of the citizens of Blackwell, when it chose to remediate the contaminated soil of the Blackwell Zinc Smelter site and the soil of the surrounding community at 750ppm for lead, 75ppm for Cadmium and 50ppm for Arsenic according to its Record of Decision in 1996, well in excess of the EPA health standards for residential and recreational areas?**

In 1992 the Environmental Protection Agency began to investigate the contamination of the historic Blackwell Zinc Smelter which had closed in 1972. As a result of its investigation, it determined that the soil and ground water was contaminated and placed the site under the EPA's Superfund program.

Under a pilot program, the EPA allowed the ODEQ to handle the remediation of the site. In 1996, the ODEQ published the Record of Decision (ROD) according to the Comprehensive Environmental Response Compensation and Liability Act (CERCLA), an act of the federal government to regulate the Superfund program.

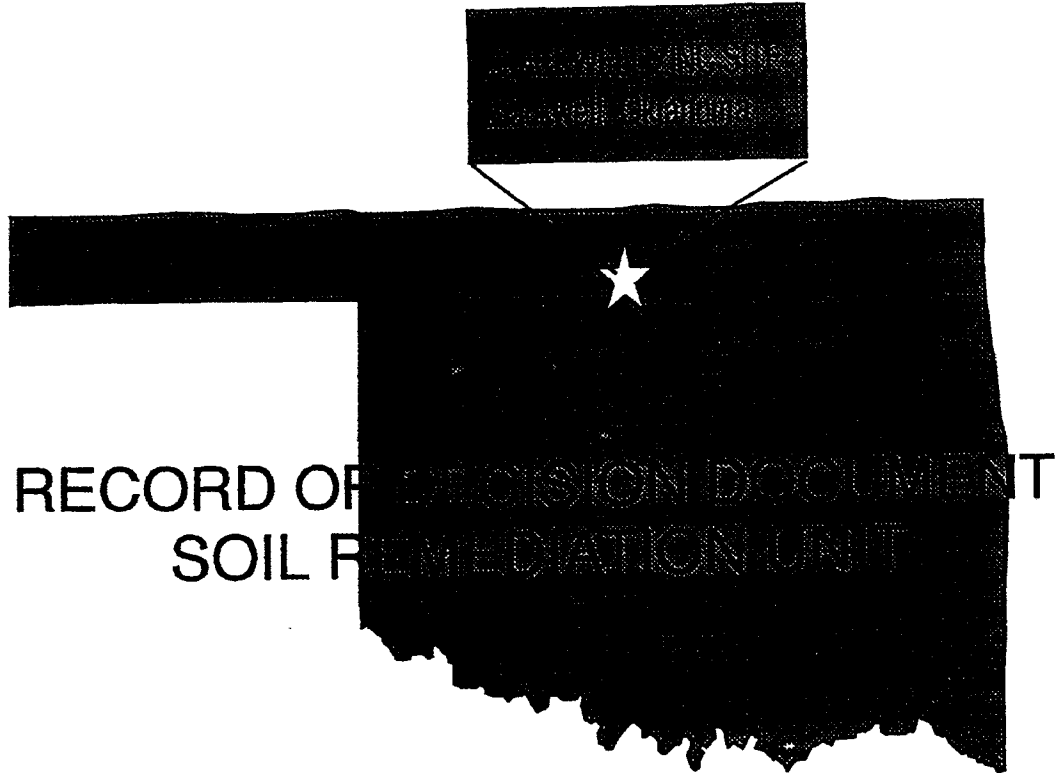
The Blackwell Zinc Smelter site did not have its own risk assessment, instead the ODEQ utilized the risk assessment from Bartlesville Ok, a similar site.

The ROD stated the remediation levels would be set at 750ppm in residential and recreational areas. The EPA set the standard for lead contaminated soil at 400ppm where children play. Clearly children play within the City of Blackwell and its parks. (See attached EPA and OSHA Standards)

The levels for Cadmium and Arsenic are also higher than what the EPA suggests to be safe levels for anyone, yet the ROD states that in residential and recreational areas for Cadmium it is at 75ppm for Blackwell. According to the National Institute of Occupational Safety and Health 7ppm is safe in an industrial environment. The ROD states that the 50ppm is standard for remediation of residential and recreational areas in Blackwell, yet 2ppm is reported safe by the EPA.

Submitted this day November 28, 2008.

J. Leppa  
Requesting Party



Prepared by

**OKLAHOMA**

**DEPARTMENT OF ENVIRONMENTAL QUALITY**

**APRIL 4, 1996**

## HIGHLIGHTS OF THE SELECTED REMEDY

- This Site is separated into three Remediation Units. This Record of Decision Document (ROD) is for the Soil Remediation Unit, which deals with human health concerns.
- Separate remediation levels are being established for two different land use categories. Category 1 levels apply to residential and recreational areas. Category 2 levels apply to commercial and industrial areas.

Remediation Levels (mg/kg)		
	Category 1	Category 2
Lead	750	2,000
Cadmium	75	200
Arsenic	50	200

- Removal of contaminated soils will be the primary remedy for Category 1 areas. Some tilling or other treatment will also be conducted on residential properties that have concentrations of metals near the remediation levels but which do not exceed them. Contaminated soil will be consolidated and then capped on the former smelter plant property.
- Deep tilling, removal, and/or containment will be conducted on Category 2 areas. Institutional Controls will be established to assure proper present and future land uses for any areas where Category 1 remediation levels are not used.
- The remedy allows for the continued development of the former smelter plant site for commercial or industrial uses.



<http://www.epa.gov/lead/pubs/leadhaz.htm>

Last updated on Thursday, November 6th, 2008.

## Lead in Paint, Dust, and Soil

You are here: [EPA Home](#) [Prevention, Pesticides & Toxic Substances](#) [Pollution Prevention & Toxic](#) [Lead in Paint, Dust, and Soil](#) [Hazard Standards](#)

### **Residential Lead Hazard Standards - TSCA Section 403**

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As part of EPA's ongoing efforts to protect children from lead poisoning, the Agency announces, new standards to identify dangerous levels of lead in paint, dust and soil. These new national standards are more protective than previous EPA guidance and will, for the first time, provide home owners, school and playground administrators, childcare providers and others with standards to protect children from hazards posed by lead, including children in federally-owned housing.

Under these new standards, federal agencies, including Housing and Urban Development, as well as state, local and tribal governments will have new uniform benchmarks on which to base remedial actions taken to safeguard children and the public from the dangers of lead. These standards will also apply to other Federal lead provisions, such as EPA's real estate disclosure requirements presently in place for people selling or renting a home or apartment. These hazard standards will also serve as general guidance for other EPA programs engaged in toxic waste cleanups. In addition, these standards will provide landlords, parents, and childcare providers with specific levels on which to make informed decisions regarding lead found in their homes, yards, or play areas.

Health problems from exposure to lead can include profound developmental and neurological impairment in children. Lead poisoning has been linked to mental retardation, poor academic performance and juvenile delinquency. Nearly one million children in America today have dangerously elevated levels of lead in their blood. Because of the potential dangers, any exposure to deteriorated lead-based paint presents a hazard.

Under the new standards, lead is considered a hazard when equal to or exceeding: 40 micrograms of lead in dust per square foot on floors; 250 micrograms of lead in dust per square foot on interior window sills and 400 parts per million (ppm) of lead in bare soil in children's play areas or 1200 ppm average for bare soil in the rest of the yard.

Identifying lead hazards through these standards will allow inspectors and risk assessors to assist property owners in deciding how to address problems which may include lead paint abatement, covering or removing soil or professional cleaning of lead dust.

This action appears in the [January 5, 2001 Federal Register \(PDF\)](#) (36 pp, 357 KB, [About PDF](#)).

[Contact the National Lead Information Center \(NLIC\)](#) to speak with an information specialist.

You will need the free Adobe Reader to view some of the files on this page.  
See [EPA's PDF page](#) to learn more.

### **Final Rules and Policy in Effect**

- 40 CFR Part 745, Lead; Identification of Dangerous Levels of Lead; Final Rule - 1/5/2001 (PDF) (36 pp, 357 KB)
  - Authorization Status of States and Tribes (PDF) (1 pg, 67 KB)
  - Interpretive Guidance for the Federal Program, TSCA Sections 402/403
  - Economic Analysis of Toxic Substances Control Act Section 403: Hazard Standards (PDF) (207 pp, 1 MB)
- Risk Analysis to Support Standards for Lead in Paint, Dust, and Soil, June 1998 (EPA 747-R-97-006)
- Risk Analysis to Support Standards for Lead in Paint, Dust, and Soil: Supplemental Report, December 2000 (EPA 747-R-00-004)
  - Response to Comments (PDF) (188 pp, 373 KB)
- Fact Sheet: Identifying Lead hazards in Residential Properties - April 2001 (PDF) (2 pp, 221 KB)

### **Proposed Rules and Other Notices**

- 40 CFR Part 745, Lead; Identification of Dangerous Levels of Lead; Proposed Rule - 6/3/98



<http://www.epa.gov/ttn/atw/hlthef/arsenic.html>

Last updated on Tuesday, November 6th, 2007.

## Technology Transfer Network Air Toxics Web Site

You are here: [EPA Home](#) [Air & Radiation](#) [TTN Web - Technology Transfer Network](#) [Air Toxics Web site](#) [Arsenic Compounds](#)

# Arsenic Compounds

## ARSENIC COMPOUNDS(A) 107-02-8

### Hazard Summary-Created in April 1992; Revised in January 2000

Arsenic, a naturally occurring element, is found throughout the environment; for most people, food is the major source of exposure. Acute (short-term) high-level inhalation exposure to arsenic dust or fumes has resulted in gastrointestinal effects (nausea, diarrhea, abdominal pain); central and peripheral nervous system disorders have occurred in workers acutely exposed to inorganic arsenic. Chronic (long-term) inhalation exposure to inorganic arsenic in humans is associated with irritation of the skin and mucous membranes. Chronic oral exposure has resulted in gastrointestinal effects, anemia, peripheral neuropathy, skin lesions, hyperpigmentation, and liver or kidney damage in humans. Inorganic arsenic exposure in humans, by the inhalation route, has been shown to be strongly associated with lung cancer, while ingestion of inorganic arsenic in humans has been linked to a form of skin cancer and also to bladder, liver, and lung cancer. EPA has classified inorganic arsenic as a Group A, human carcinogen.

Arsine is a gas consisting of arsenic and hydrogen. It is extremely toxic to humans, with headaches, vomiting, and abdominal pains occurring within a few hours of exposure. EPA has not classified arsine for carcinogenicity.

Please Note: The main sources of information for this fact sheet are EPA's Integrated Risk Information System (IRIS), which contains information on inhalation chronic toxicity and the RfC for arsine, oral chronic toxicity and the RfD for inorganic arsenic, and the carcinogenic effects of inorganic arsenic including the unit cancer risk for inhalation exposure, and the Agency for Toxic Substances and Disease Registry's (ATSDR's) Toxicological Profile for Arsenic.

### Uses

- The major use for inorganic arsenic is in wood preservation; arsine is used in the microelectronics industry and in semiconductor manufacture. (2)
- Until the 1940s, inorganic arsenic solutions were widely used in the treatment of various diseases, such as syphilis and psoriasis. Inorganic arsenic is still used as an antiparasitic agent in veterinary medicine and in homeopathic and folk remedies in the United States and other countries. (2)

## Sources and Potential Exposure

- Inorganic arsenic is found throughout the environment; it is released into the air by volcanoes, the weathering of arsenic-containing minerals and ores, and by commercial or industrial processes. (1,2)
- For most people, food is the largest source of arsenic exposure (about 25 to 50 micrograms per day [ $\mu\text{g}/\text{d}$ ]), with lower amounts coming from drinking water and air. Among foods, some of the highest levels are found in fish and shellfish; however, this arsenic exists primarily as organic compounds, which are essentially nontoxic. (1)
- Elevated levels of inorganic arsenic may be present in soil, either from natural mineral deposits or contamination from human activities, which may lead to dermal or ingestion exposure. (1)
- Workers in metal smelters and nearby residents may be exposed to above-average inorganic arsenic levels from arsenic released into the air. (1)
- Other sources of inorganic arsenic exposure include burning plywood treated with an arsenic wood preservative or dermal contact with wood treated with arsenic. (2)
- Most arsenic poisoning incidents in industry have involved the production of arsine, a short-lived, extremely toxic gas. (3)

## Assessing Personal Exposure

- Measurement of inorganic arsenic in the urine is the best way to determine recent exposure (within the last 1 to 2 days), while measuring inorganic arsenic in hair or fingernails may be used to detect high-level exposures that occurred over the past 6-12 months. (1)

## Health Hazard Information

### **Acute Effects:**

#### **Inorganic Arsenic**

- Acute inhalation exposure of workers to high levels of arsenic dusts or fumes has resulted in gastrointestinal effects (nausea, diarrhea, abdominal pain), while acute exposure of workers to inorganic arsenic has also resulted in central and peripheral nervous system disorders. (1)
- Acute oral exposure to inorganic arsenic, at doses of approximately 600 micrograms per kilogram body weight per day ( $\mu\text{g}/\text{kg}/\text{d}$ ) or higher in humans, has resulted in death. Oral exposure to lower levels of inorganic arsenic has resulted in effects on the gastrointestinal tract (nausea, vomiting), central nervous system (CNS) (headaches, weakness, delirium), cardiovascular system (hypotension, shock), liver, kidney, and blood (anemia, leukopenia). (1,2)
- Acute animal tests in rats and mice have shown inorganic arsenic to have moderate to high acute toxicity. (5)

#### **Arsine**

- Acute inhalation exposure to arsine by humans has resulted in death; it has been reported that a half-hour exposure to 25 to 50 parts per million (ppm) can be lethal. (4)
- The major effects from acute arsine exposure in humans include headaches, vomiting, abdominal pains, hemolytic anemia, hemoglobinuria, and jaundice; these effects can lead to kidney failure. (4,8)
- Arsine has been shown to have extreme acute toxicity from acute animal tests. (5)

### **Chronic Effects (Noncancer):**



### **Inorganic arsenic**

- Chronic inhalation exposure to inorganic arsenic in humans is associated with irritation of the skin and mucous membranes (dermatitis, conjunctivitis, pharyngitis, and rhinitis). (1,2)
- Chronic oral exposure to inorganic arsenic in humans has resulted in gastrointestinal effects, anemia, peripheral neuropathy, skin lesions, hyperpigmentation, gangrene of the extremities, vascular lesions, and liver or kidney damage. (1,2)
- No chronic inhalation exposure studies have been performed in animals for any inorganic arsenic compound. (1)
- Some studies have suggested that inorganic arsenic is an essential dietary nutrient in goats, chicks, and rats. However, no comparable data are available for humans. EPA has concluded that essentiality, although not rigorously established, is plausible. (1,6)
- EPA has not established a Reference Concentration (RfC) for inorganic arsenic. (6)
- The California Environmental Protection Agency (CalEPA) has established a chronic inhalation reference level of 0.00003 milligrams per cubic meter ( $\text{mg}/\text{m}^3$ ) based on developmental effects in mice. The CalEPA reference exposure level is a concentration at or below which adverse health effects are not likely to occur. It is not a direct estimator of risk, but rather a reference point to gauge the potential effects. At lifetime exposures increasingly greater than the reference exposure level, the potential for adverse health effects increases. (7)
- The Reference Dose (RfD) for inorganic arsenic is 0.0003 milligrams per kilogram body weight per day ( $\text{mg}/\text{kg}/\text{d}$ ) based on hyperpigmentation, keratosis, and possible vascular complications in humans. The RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without appreciable risk of deleterious noncancer effects during a lifetime. (6)
- EPA has medium confidence in the study on which the RfD for inorganic arsenic was based because, although an extremely large number of people were included in the assessment (>40,000), the doses were not well characterized and other contaminants were present. The supporting human toxicity database, while extensive, is somewhat flawed and, consequently, EPA has assigned medium confidence to the RfD. (6)

### **Arsine**

- No information is available on the chronic effects of arsine in humans.
- The RfC for arsine is 0.00005  $\text{mg}/\text{m}^3$  based on increased hemolysis, abnormal red blood cell morphology, and increased spleen weight in rats, mice, and hamsters. (4)
- EPA has medium confidence in the RfC based on: (1) high confidence in the studies on which the RfC for arsine was based because the sample sizes were adequate, statistical significance was reported, concentration dose-response relationships were documented, three species were investigated, and both a no-observed-adverse-effect level (NOAEL) and a lowest-observed-adverse-effect level (LOAEL) were identified, and (2) medium confidence in the database because while there were three inhalation animal studies and a developmental/reproductive study, there were no data available on human exposure. (4)

### **Reproductive/Developmental Effects:**

#### **Inorganic arsenic**

- Several studies have suggested that women who work in, or live near, metal smelters may have higher than normal spontaneous abortion rates, and their children may exhibit lower than normal birthweights. However, these studies are limited because they were designed to evaluate the effects of smelter pollutants in general, and are not specific for inorganic arsenic. (1)
- Ingested inorganic arsenic can cross the placenta in humans, exposing the fetus to the chemical. (2)
- Oral animal studies have reported inorganic arsenic at very high doses to be fetotoxic and to cause birth defects. (1)

#### **Arsine**

- Human studies have indicated higher than expected spontaneous abortion rates in women in the microelectronics industry who were exposed to arsine. However, these studies have several limitations, including small sample size and exposure to other chemicals in addition to arsine. (4)

#### **Cancer Risk:**

##### **Inorganic arsenic**

- Human, inhalation studies have reported inorganic arsenic exposure to be strongly associated with lung cancer. (1,2,6)
- Ingestion of inorganic arsenic in humans has been associated with an increased risk of nonmelanoma skin cancer and also to an increased risk of bladder, liver, and lung cancer. (1,6)
- Animal studies have not associated inorganic arsenic exposure via the oral route with cancer, and no cancer inhalation studies have been performed in animals for inorganic arsenic. (1)
- EPA has classified inorganic arsenic as a Group A, human carcinogen. (6)
- EPA used a mathematical model, using data from an occupational study of arsenic-exposed copper smelter workers, to estimate the probability of a person developing cancer from continuously breathing air containing a specified concentration of inorganic arsenic. EPA calculated an inhalation unit risk estimate of  $4.3 \times 10^{-3}(\mu\text{g}/\text{m}^3)^{-1}$ . EPA estimates that, if an individual were to continuously breathe air containing inorganic arsenic at an average of  $0.0002 \mu\text{g}/\text{m}^3$  ( $2 \times 10^{-7} \text{mg}/\text{m}^3$ ) over his or her entire lifetime, that person would theoretically have no more than a one-in-a-million increased chance of developing cancer as a direct result of breathing air containing this chemical. Similarly, EPA estimates that continuously breathing air containing  $0.002 \mu\text{g}/\text{m}^3$  ( $2 \times 10^{-6} \text{mg}/\text{m}^3$ ) would result in not greater than a one-in-a-hundred thousand increased chance of developing cancer, and air containing  $0.02 \mu\text{g}/\text{m}^3$  ( $2 \times 10^{-5} \text{mg}/\text{m}^3$ ) would result in not greater than a one-in-ten thousand increased chance of developing cancer. For a detailed discussion of confidence in the potency estimates, please see IRIS. (6)
- EPA has calculated an oral cancer slope factor of  $1.5 (\text{mg}/\text{kg}/\text{d})^{-1}$  for inorganic arsenic. (6)

#### **Arsine**

- No cancer inhalation studies in humans or animals are available for arsine. (1)
- EPA has not classified arsine for carcinogenicity. (4)

#### **Physical Properties**

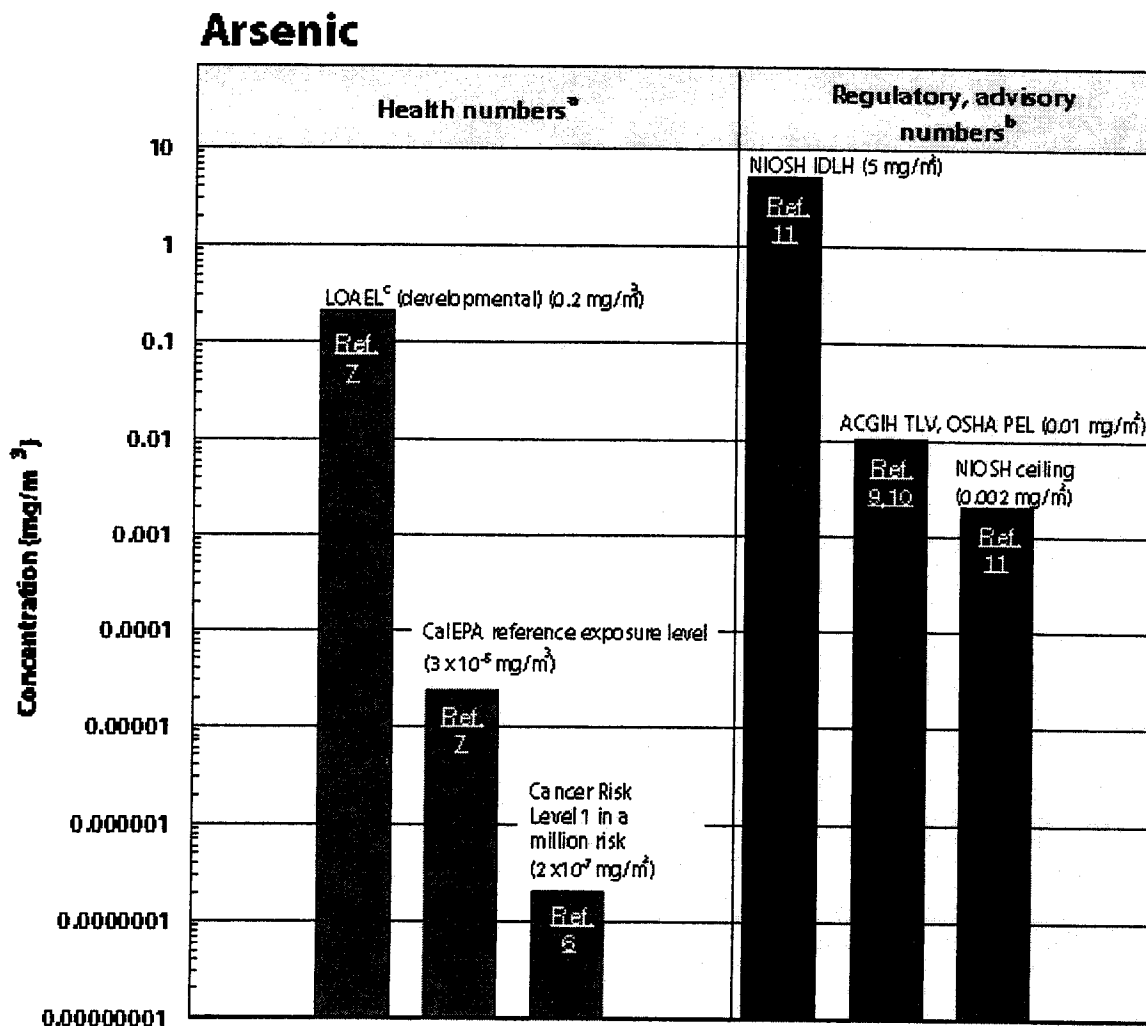
- Inorganic arsenic is a naturally occurring element in the earth's crust.(1)
- Pure inorganic arsenic is a gray-colored metal, but inorganic arsenic is usually found combined with other elements such as oxygen, chlorine, and sulfur. (1)

- The chemical symbol for inorganic arsenic is As, and it has an atomic weight of 74.92 g/mol. (3)
- The chemical formula for arsine is AsH<sub>3</sub>, and it has a molecular weight of 77.95 g/mol. (8)
- Arsine is a colorless gas with a disagreeable garlic odor. (8)
- Arsenic combined with elements such as oxygen, chlorine, and sulfur forms inorganic arsenic; inorganic arsenic compounds include arsenic pentoxide, arsenic trioxide, and arsenic acid. Arsenic combined with carbon and hydrogen forms organic arsenic; organic arsenic compounds include arsanilic acid, arsenobetaine, and dimethylarsinic acid. (1)

**Conversion Factors (only for the gaseous form):**

To convert concentrations in air (at 25°C) from ppm to mg/m<sup>3</sup>:  $mg/m^3 = (ppm) \times (molecular\ weight\ of\ the\ compound)/(24.45)$ . For inorganic arsenic: 1 ppm = 3.06 mg/m<sup>3</sup>. For arsine: 1 ppm = 3.19 mg/m<sup>3</sup>. To convert concentrations in air from µg/m<sup>3</sup> to mg/m<sup>3</sup>:  $mg/m^3 = (\mu g/m^3) \times (1\ mg/1,000\ \mu g)$ .

**Health Data from Inhalation Exposure (Inorganic Arsenic)**



**ACGIH TLV**--American Conference of Governmental and Industrial Hygienists' threshold limit

value expressed as a time-weighted average; the concentration of a substance to which most workers can be exposed without adverse effects.

**NIOSH IDLH**--National Institute of Occupational Safety and Health's immediately dangerous to life or health concentration; NIOSH recommended exposure limit to ensure that a worker can escape from an exposure condition that is likely to cause death or immediate or delayed permanent adverse health effects or prevent escape from the environment.

**NIOSH REL ceiling value**--NIOSH's recommended exposure limit ceiling; the concentration that should not be exceeded at any time.

**OSHA PEL**--Occupational Safety and Health Administration's permissible exposure limit expressed as a time-weighted average; the concentration of a substance to which most workers can be exposed without adverse effect averaged over a normal 8-h workday or a 40-h workweek.

The health and regulatory values cited in this factsheet were obtained in December 1999.

<sup>a</sup> Health numbers are toxicological numbers from animal testing or risk assessment values developed by EPA.

<sup>b</sup>Regulatory numbers are values that have been incorporated in Government regulations, while advisory numbers are nonregulatory values provided by the Government or other groups as advice. OSHA numbers are regulatory, whereas NIOSH and ACGIH numbers are advisory.

<sup>c</sup>The LOAEL is from the critical study used as the basis for the CalEPA chronic reference exposure level.

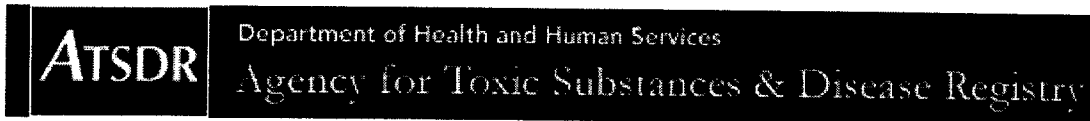
## References

1. Agency for Toxic Substances and Disease Registry (ATSDR). *Toxicological Profile for Arsenic* (Draft). U.S. Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA. 1998.
2. Agency for Toxic Substances and Disease Registry (ATSDR). *Case Studies in Environmental Medicine. Arsenic Toxicity*. U.S. Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA. 1990.
3. U.S. Environmental Protection Agency. *Health Assessment Document for Inorganic Arsenic*. EPA/540/1-86/020. Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Office of Research and Development, Washington, DC. 1984.
4. U.S. Environmental Protection Agency. *Integrated Risk Information System (IRIS) on Arsenic*. National Center for Environmental Assessment, Office of Research and Development, Washington, DC. 1999.
5. U.S. Department of Health and Human Services. Registry of Toxic Effects of Chemical Substances (RTECS, online database). National Toxicology Information Program, National Library of Medicine, Bethesda, MD. 1993.
6. U.S. Environmental Protection Agency. *Integrated Risk Information System (IRIS) on Arsenic*. National Center for Environmental Assessment, Office of Research and Development, Washington, DC. 1999.
7. California Environmental Protection Agency (CalEPA). *Technical Support Document for the Determination of Noncancer Chronic Reference Exposure Levels. Draft for Public Comment*. Office of Environmental Health Hazard Assessment, Berkeley, CA. 1997.
8. M. Windolz. *The Merck Index, An Encyclopedia of Chemicals, Drugs, and Biologicals*. 10th ed. Merck and Co., Rahway, NJ. 1983.
9. American Conference of Governmental Industrial Hygienists (ACGIH). *1999 TLVs and BEIs. Threshold Limit Values for Chemical Substances and Physical Agents. Biological Exposure Indices*. Cincinnati, OH. 1999.
10. Occupational Safety and Health Administration (OSHA). Occupational Safety and Health Standards, Toxic and Hazardous Substances. *Code of Federal Regulations*. 29 CFR 1910.1000. 1998.
11. National Institute for Occupational Safety and Health (NIOSH). *Pocket Guide to Chemical Hazards*. U.S. Department of Health and Human Services, Public Health

Service, Centers for Disease Control and Prevention. Cincinnati, OH. 1997.

A. \* This fact sheet addresses the toxicity of the inorganic arsenic compounds as well as the toxicity of the gaseous arsenic trihydride: arsine.

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ATSDR en Español

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**Case Studies in Environmental Medicine (CSEM)**

# Cadmium Toxicity

## What Are the U.S. Standards for Cadmium Exposure?

<b>Learning Objective</b>	Upon completion of this section, you will be able to: <ul style="list-style-type: none"> <li>● identify U.S. guidelines and regulations</li> </ul>
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<b>Introduction</b>	With increasing evidence of cadmium's toxicity, regulatory agencies have sought to regulate its exposure. This course discusses the toxic and health effects of cadmium; there are established standards for occupational, health, and environmental exposure.
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<b>Workplace Standards</b>	OSHA has established workplace levels to protect workers exposed to cadmium.  The OSHA limits are: <ul style="list-style-type: none"> <li>● Permissible Exposure Limit- TWA (PEL)</li> </ul> The National Institute of Occupational Safety and Health (NIOSH) has established the following limits: <ul style="list-style-type: none"> <li>● Immediately Dangerous to Life and Health (IDLH) (NIOSH 2006; NTP 2004).</li> </ul>
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**Case Studies (CSEM)**

- CSEM Home
- Continuing Education
- Online Registration

**ATSDR Resources**

- Case Studies (CSEM)
- Exposure Pathways
- GATHER (GIS)
- Health Assessments
- Health Statements
- Interaction Profiles
- Interactive Learning
- Managing Incidents
- Medical Guidelines
- Minimal Risk Levels
- Priority List
- ToxFAQs™
- ToxFAQs™ CABS
- Toxicological Profiles
- Toxicology Curriculum

**External Resources**

- CDC
- eLCOSH
- EPA
- Healthfinder®
- Medline Plus
- NCEH
- NIEHS
- NIOSH
- OSHA

<p><b>Health Standards</b></p>	<p>Many health agencies have set exposure star public from excess cadmium exposure from v</p> <p><b>FDA</b></p> <ul style="list-style-type: none"> <li>● Maximum limit of cadmium in bottled v</li> </ul> <p><b>ATSDR</b></p> <ul style="list-style-type: none"> <li>● Chronic durational oral minimal risk lev cadmium based on its renal effects.</li> <li>● This MRL standard states how much ca without risk of adverse health effects (</li> </ul> <p><b>EPA</b></p> <ul style="list-style-type: none"> <li>● Food – Reference dose is <math>1 \times 10^{-3}</math> mg/l</li> <li>● Water - Reference dose for human exp</li> <li>● Reference dose (Rfd) is an estimate of population (including sensitive subgrou appreciable risk of deleterious effects d</li> </ul> <p><b>World Health Organization (WHO)</b></p> <ul style="list-style-type: none"> <li>● Tolerable weekly intake for cadmium at</li> </ul>
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<p><b>Carcinogenicity</b></p>	<p>Positions on carcinogenicity of cadmium by U organizations.</p> <ul style="list-style-type: none"> <li>● EPA classifies cadmium as a probable h</li> <li>● International Agency for Research on C known human carcinogen.</li> <li>● American Conference of Industrial Hygi suspected human carcinogen.</li> <li>● National Toxicology Program (NTP) clas carcinogen (NTP 2004).</li> </ul>
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<p><b>Environmental Standards</b></p>	<p><b>EPA</b></p> <ul style="list-style-type: none"> <li>● Drinking water - maximum contaminant 0.005 mg/L. (ATSDR, 1999)</li> <li>● Air - Cadmium is on the EPA National E Pollutants (NESHAP) list of 189 hazardous one of 33 hazardous air pollutants that health in urban areas (ATSDR 1999).</li> <li>● Soil - EPA biosolids rule states that the can be applied to land is 85 mg/kg fill r</li> </ul>
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<p><b>Key Points</b></p>	<ul style="list-style-type: none"> <li>● Because much is known about the human large database from which to set standards</li> <li>● With increasing evidence of its toxicity, have sought to regulate cadmium exposure</li> </ul>
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<p><b>Progress Check</b></p>	<p>6. Guidelines issued by U.S. agencies are designed to of the following?</p> <ul style="list-style-type: none"> <li><input type="radio"/> A. The ATSDR MRL, which states how much cadmium without risk of adverse health effects, is 0.0002 mg/kg effects.</li> <li><input type="radio"/> B. NIOSH has set an IDLH of 9 mg/m<sup>3</sup>.</li> <li><input type="radio"/> C. The EPA reference dose for daily exposure to the without appreciable risk of deleterious effects during ; water.</li> <li><input type="radio"/> D. The OSHA PEL for people occupationally exposed</li> <li><input type="radio"/> E. All of the above.</li> </ul> <p>Answer:</p>
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