

HEAVY METALS

CDC's National Exposure Report includes data on the body burdens of a number of so-called "heavy" metals. Heavy metals include those metals from periodic table group IIA to VIA, which are characterized by a specific gravity (a measure of density) at least five times that of water. The heavy metals most often linked to human poisoning are lead, mercury, arsenic, and cadmium. Other heavy metals, including copper, zinc, and chromium, are required by the body in small amounts, but can be toxic in larger doses.

For most of the metals included in the study, urine specimens were collected and analyzed for persons 6 years of age and older. However, because of known health effects of lead, cadmium, and mercury, additional populations are included in the study. Young children are highly susceptible to the toxic effects of exposure to these metals, particularly serious psychological and neurological effects. Mercury levels in women of childbearing age are included because they provide an indication of potential fetal exposure levels. The table below summarizes the specific population groups and analyses conducted for lead, cadmium, and mercury.

Heavy Metal	Sample Type	Population/Age
Lead	Blood	≥ 1 year old
	Urine	≥ 6 years old
Cadmium	Blood	≥ 1 year old
	Urine	≥ 6 years old
Mercury	Blood	Children, 1-5 years old Females, 16-49 years old
All other metals	Urine	≥ 6 years old

Sources of Heavy Metals and Routes of Exposure:

Heavy metals have been widely used in industry since the 1800s and as a result are common environmental contaminants. Sources of heavy metals include air emissions from coal-burning plants, smelters, and other industrial facilities (e.g., cadmium and arsenic); waste incinerators (mercury and cadmium); process wastes from mining and industry; pesticides and wood preservatives (e.g., arsenic and chromium); fertilizers (e.g., cadmium is found in phosphate fertilizers); and lead in household plumbing and old house paints. While certain types of industrial facilities are required by the EPA to report their releases of some heavy metals to the Toxics Release Inventory (TRI), other major sources—including power plants and waste incinerators—are not. Consequently, the TRI may significantly under-report actual environmental releases of some metals.

Heavy metals can also enter the environment through natural processes. For example, in some parts of the U.S., naturally occurring geologic deposits of arsenic dissolve into groundwater, potentially resulting in unsafe levels in drinking water supplies. Once released to the environment, metals can remain for decades or centuries, increasing the likelihood of human exposure.

Humans are exposed to heavy metals through inhalation of air pollutants, consumption of contaminated drinking water, exposure to contaminated soils or industrial waste, or consumption of contaminated food. Food sources such as vegetables, grains, fruits, fish, and shellfish can become contaminated by accumulating metals from surrounding soil and water.

Health Effects of Heavy Metals:

Heavy metals cause serious health effects, including reduced growth and development, cancer, organ damage, nervous system damage, and in extreme cases, death. Exposure to some metals, such as mercury and lead, may also cause development of autoimmunity, in which a person's immune system attacks its own cells. This can lead to joint diseases, such as rheumatoid arthritis, and diseases of the kidneys, circulatory system, and nervous system. Metals are particularly toxic to the sensitive rapidly developing systems of the fetus, infants, and young children. Some metals, such as lead and mercury, easily cross the placenta and can damage the fetal brain. Childhood exposure to some metals can result in learning difficulties, memory impairment, damage to the nervous system, and behavioral problems such as aggressiveness and hyperactivity. At higher doses, heavy metals can cause irreversible brain damage. Children may receive higher doses of metals from food than adults, since they consume more food for their body weight than adults. Also, children absorb metals more readily through their intestinal tract than adults.

Sources of Information

Klaassen CD, ed. 1996. Casarett and Doull's Toxicology: The Basic Science of Poisons. New York: McGraw-Hill.

World Resources Institute (WRI). *Heavy Metals and Health*. Accessed online at <http://www.wri.org/wri/wr-98-99/metals2.htm>.

OSHA. *Heavy Metals*. Occupational Safety and Health Administration. Accessed online at <http://www.osha-slc.gov/SLTC/metalsheavy/index.html>.

Chemical Name : LEAD

Summary: Lead is a heavy, soft gray metal with a wide variety of industrial and consumer uses. The EPA classifies lead as a probable human carcinogen.

Lead poisoning is one of the most common environmental health problems in the U.S., with particularly severe effects on young children. Prenatal or childhood exposure to lead can result in long-term damage to the brain and nervous system and behavioral problems lasting into adulthood.

Uses and Sources: Lead has a wide variety of uses due to its properties of high density, softness, low melting point, resistance to corrosion, and ability to stop gamma and x-rays. Lead is commonly used in storage batteries for automobiles and industry; electrical and electronic equipment; machine bearings; cable coverings; and in pipes, traps, solder, and sheets for building construction. In the U.S., lead and zinc processing facilities account for a large portion of lead releases to the environment. These operations and various industrial sources have contributed to widespread environmental contamination by lead. According to EPA's Toxics Release Inventory, reporting industries released more than 370 million pounds of lead and lead compounds into the environment in 2000.

Major Consumer Products: Lead is used for many consumer products, including lead-acid batteries, metal products, ammunition, and ceramic glazes. Use of lead in gasoline was phased out in the U.S. and is now banned for transportation purposes. Lead is also used in some paints, although the amount has been reduced because of health concerns.

Routes of Human Exposure: The risk of lead poisoning is highest for children who live in housing containing lead-based paint. Many older homes were painted with leaded paint that can release lead dust and chips as it ages. (The lead content of paint was not regulated until 1977.) This lead dust accumulates on window ledges, floors, and other surfaces, where children come into contact with it while crawling and playing. Through normal hand-to-mouth activity, children can then ingest this lead-contaminated dust. Some children with pica, a behavior of eating non-food items, can also be exposed to lead by eating contaminated paint chips.

Exposure to lead can also result from breathing contaminated air, contacting lead-contaminated soils, or drinking contaminated water. Lead gasoline additives, smelters, and battery plants are the most significant contributors to lead emissions into the atmosphere. Tap water contamination with lead occurs when water (especially acidic water) passes through older pipes containing lead, lead solder, or brass fixtures that contain lead.

Health Effects: The health effects of lead exposure have been extensively studied. When lead enters the body, it travels through the blood to the soft tissues, such as brain, liver, and kidneys. In adults, most lead taken into the body is excreted, with a small amount stored in bones and teeth, where it can accumulate with repeated exposure. Chronic exposure to lead can cause a variety of adverse health effects in adults, including brain and kidney damage, poor reaction time, joint weakness, anemia (a disorder of the blood),

memory impairment, and possibly increased blood pressure. These effects can persist long after lead exposure ends. Lead can be released from the bones into blood under certain circumstances, such as during pregnancy and breastfeeding. These releases can potentially expose the developing fetus or nursing infant. Lead is classified by EPA as a probable human carcinogen, based on evidence from rodent studies. Large doses of lead have caused tumors in rats and mice. There is not yet enough information to determine whether lead causes cancer in humans.

Children are highly sensitive to the effects of lead largely because lead affects the child's developing nervous system. Children absorb and retain more lead than adults (in proportion to their body weight), and most of the lead that enters a child's body remains stored in the body. Because lead accumulates in the body faster than it is excreted, repeated exposure to small amounts of lead can result in poisoning. Prenatal exposure has been linked to a variety of effects in the infant, including premature birth, low birth weight, decreased mental ability, learning difficulties, and slow growth. Childhood exposure to lead has been associated with anemia, learning disabilities, attention deficits, lower intelligence scores, slowed or stunted growth, and effects on kidney function—effects that can persist as the child grows older. Lead exposure has also been associated with behavioral effects such as impulsiveness and aggression.

Detecting Exposure: Lead is one of the few chemicals for which health effects can be directly linked to specific measured levels in the blood. Measuring the amount of lead in the blood has become a common way of determining the amount of lead in the body and for screening children for lead poisoning. The CDC considers children to have an elevated level of lead if the amount in the blood is 10 micrograms per deciliter ($\mu\text{g}/\text{dL}$) or higher. However recent information suggests that effects on the nervous system and developmental effects in children can occur at blood lead levels as low as $2.5 \mu\text{g}/\text{dL}$. CDC's first National Exposure Report (2001) reported geometric mean blood lead levels of $2 \mu\text{g}/\text{dL}$ in children ages 1-5 years, and $1.6 \mu\text{g}/\text{dL}$ for all age groups. The geometric mean of urine lead concentration across all age groups was $0.80 \mu\text{g}/\text{L}$.

Regulations for Protection of Human Health:

Air – EPA regulates lead as a “criteria air pollutant” under the Clean Air Act, and has designated lead as a hazardous air pollutant. Lead emissions are controlled for various industrial operations and in motor vehicle emissions. EPA limits concentrations of lead in the air to 1.5 micrograms of lead per cubic meter of air ($1.5 \mu\text{g}/\text{m}^3$).

Drinking Water – EPA has established an “action level” of $0.015 \text{ mg}/\text{L}$ for lead in drinking water. Action levels are not enforceable standards; rather, they are intended to serve as guidance for public health decision-making. Water utilities are required to replace lead service lines when more than 10% of homes exceed the action level (if corrosion cannot be controlled).

Dust and Soil – To help protect children from lead poisoning, EPA has established new standards to identify dangerous levels of lead in paint, dust, and contaminated soil. These

standards provide specific targets that can be used in cleaning up lead found in homes, yards, businesses, schools, and play areas. The standards are: 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 of lead in bare soil in children's play areas.

Lead-Based Paints – To prevent exposure to lead-based paints, the Consumer Product Safety Commission limits the amount of lead in most paints to less than 0.06% lead.

Food – FDA has set action levels of 1.5 ppm lead in crustacea and 1.7 ppm in clams, oysters, and mussels.

Occupational Exposure – OSHA has established a PEL of 0.050 mg/m³ for lead in workplace air.

Sources of Information:

ATSDR. *Toxicological Profile for Lead (Update)*. Agency for Toxic Substances and Disease Registry, Atlanta, GA. 1999. Accessed online at <http://www.atsdr.cdc.gov/toxprofiles/tp13.html>.

ATSDR *Case Studies in Environmental Medicine. Lead Toxicity*. U.S. Department of Health and Human Services, Atlanta, GA. 2000. Accessed online at <http://www.atsdr.cdc.gov/HEC/CSEM/lead/index.html>.

EPA. 2000 Toxics Release Inventory (TRI) Public Data Release Report. Office of Environmental Information, Washington, DC. EPA-260-R-02-003. Accessed online at <http://www.epa.gov/tri/tridata/tri00/pdr/index.htm>.

EPA. National Primary Drinking Water Regulations, Consumer Factsheet on Lead. Office of Groundwater and Drinking Water, Washington, DC. 1998. Accessed online at www.epa.gov/OGWDW/dwh/c-ioc/lead.html.

EPA Integrated Risk Information System. Lead and compounds (inorganic). National Center for Environmental Assessment, Washington, DC. Accessed online at <http://www.epa.gov/iris/index.html>.

National Library of Medicine (NLM). TOXNET Database, Hazardous Substances Databank (HSDB). National Institutes of Health, Bethesda, MD. Accessed online at <http://toxnet.nlm.nih.gov/>.

Physicians for Social Responsibility. "Drinking water fact sheet #8, Lead: What health care providers should know."

Chemical Name: MERCURY

Summary: Mercury is a silvery, heavy, odorless liquid best known for its use in fever thermometers. This persistent heavy metal occurs in elemental form and in various organic forms, most notably, methylmercury. Environmental contamination with mercury is widespread as a result of industrial activity, particularly fossil fuel combustion. Highly toxic methylmercury accumulates in many edible fish species, which are a major source of mercury exposure in the U.S. Exposures by pregnant women are of particular concern, because methylmercury can cross the placenta and enter the fetal brain. Children exposed to even low levels of mercury before birth can experience serious neurological and development deficits.

Uses and Sources: Mercury is used to make a variety of chemicals, industrial equipment, and consumer products. Most of the mercury associated with human activity is released to the air from fossil fuel power plants. Mercury is also released into the environment by mining, smelting, and waste incineration. According to EPA's Toxics Release Inventory, reporting industries released more than 4 million pounds of mercury and mercury compounds into the environment in 2000.

Major Consumer Products: In addition to thermometers, mercury is found in barometers, batteries, vapor lamps, mirror coatings, dental amalgams, and various medical devices.

Routes of Human Exposure: Metallic and inorganic mercury are the most common forms found in the environment. Mercury released to the atmosphere can be deposited onto soil or into waterways, where biological processes can change inorganic mercury to organic forms. The most common organic form of mercury is methylmercury. Methylmercury is of particular concern because it is more toxic than other forms of mercury. It tends to accumulate in plants, animals, fish, and in the human body. People who eat certain types of fish, such as tuna and swordfish, may be exposed to higher levels of methylmercury. In addition to fish consumption, exposure to methylmercury can result from drinking contaminated water, breathing contaminated air, or through skin contact with contaminated soil or water. In the body, methylmercury is rapidly absorbed and moves into the brain. Mercury in the mother's body can be passed to the fetus, where it moves into the fetal brain. Once mercury enters the human body, it can remain for months.

Health Effects: The developing fetus and young children are most sensitive to the effects of mercury exposure, even at low levels. The developing nervous system and brain are highly sensitive to the effects of mercury in any form, but particularly to methylmercury and mercury vapors. Maternal exposure to organic mercury may lead to brain damage, mental retardation, poor coordination, speech difficulties, and other serious effects on the fetus and newborn. Children born to women exposed to methylmercury have shown a variety of abnormalities including delays in development and learning ability. Recent epidemiological studies found that children exposed to low levels of mercury before birth experienced subtle effects on motor skills, learning capacity, memory, and other

symptoms of neurological damage. The National Research Council (NRC) has estimated that neurobehavioral effects in the fetus could occur at methylmercury levels as low as 58 ppb in cord blood. The NRC cautions that children of women who consume large amounts of fish and seafood during pregnancy are at particular risk. Methylmercury has also shown potential to affect the cardiovascular system. In adults, chronic exposure to either organic or inorganic mercury can permanently damage the brain and kidneys. Exposure to metallic or organic mercury may affect the brain, resulting in effects such as personality changes, tremors, vision problems, poor muscle coordination, and memory loss.

Animal studies have shown that long-term exposure to inorganic mercury salts can cause kidney damage; effects on the nervous system and immune system; effects on blood pressure and heart rate; and effects on the stomach. Long-term animal studies have also found that exposure to organic mercury at high levels can cause nervous system damage; damage to the kidneys, stomach, and large intestine; changes in blood pressure and heart rate; adverse effects on the developing fetus, sperm, and male reproductive organs; and increases in the number of spontaneous abortions and stillbirths.

EPA has classified methylmercury and mercuric chloride as possible human carcinogens.

Detecting Exposure: Blood and urine are commonly used to measure recent exposure to mercury. Hair is a biomarker of long-term exposure to methylmercury. CDC's first National Exposure Report (2001) reported geometric means of mercury in blood of 0.3 µg/L for children ages 1- 5 years and 1.2 µg/L for women ages 16-49 years. However, 10% of the women tested by CDC were found to have mercury levels that exceeded the level defined by a National Research Council expert committee to be optimally protective for the developing fetus.

Regulations for Protection of Human Health:

Air – Mercury is regulated by the EPA as a Hazardous Air Pollutant under the Clean Air Act. EPA has indicated that it intends to begin regulating mercury emissions from power plants, although these regulations have not yet been developed.

Drinking Water – EPA has established an MCL of 0.002 mg/L for inorganic mercury in drinking water. The FDA allows no more than 2 mg/L of mercury in bottled water.

Food – In January 2001, EPA and FDA issued advisories cautioning that both commercially available and non-commercial fish may be contaminated with mercury. The FDA advised that pregnant women, nursing mothers, women of childbearing age, and young children should not to eat shark, swordfish, king mackerel, and tilefish because they may contain potentially high levels of methylmercury. EPA recommended that women who are pregnant or may become pregnant, nursing mothers and young children limit consumption of non-commercial fish to one meal per week.

The FDA has set a maximum allowable level of 1 ppm methylmercury for all fish.

At least 40 states have issued fish advisories warning pregnant women or women of reproductive age to limit or avoid consuming freshwater fish that may be contaminated with mercury. Each state, Native American tribe, or U.S. Territory establishes its own criteria for issuing fish and wildlife advisories.

Occupational Exposure – OSHA has established a PEL of 0.1 mg/m³ for mercury in workplace air.

Sources of Information:

ATSDR. *Toxicological Profile for Mercury*. Agency for Toxic Substances and Disease Registry, Atlanta, GA. 1999. Accessed online at <http://www.atsdr.cdc.gov/toxprofiles/tp46.html>.

EPA. Mercury Study Report to Congress. Volume V: Health Effects of Mercury and Mercury Compounds. Office of Air Quality Planning and Standards; Office of Research and Development. December 1997. Accessed online at <http://www.epa.gov/ttn/oarpg/t3/reports/merover.pdf>

EPA. 2000 Toxics Release Inventory (TRI) Public Data Release Report. Office of Environmental Information, Washington, DC. EPA-260-R-02-003. Accessed online at <http://www.epa.gov/tri/tridata/tri00/pdr/index.htm>.

National Research Council. *Toxicological Effects of Methylmercury*. National Academy Press, Washington, DC. 2000.

Physicians for Social Responsibility (PSR). “In Harm’s Way: Toxic Threats to Child Development”. Greater Boston PSR. 2000.

Chemical Name : CADMIUM

Summary: Cadmium is a heavy metal that occurs naturally in the environment. It also has many industrial uses, which can result in its release to air, water, and land. Cadmium accumulates in the body and can cause serious health effects such as lung disease, kidney damage, and lung cancer. Exposure to cadmium, even at low levels, may also have serious effects on the fetus, infants, and young children. This heavy metal is classified as a known human carcinogen, but few human studies have been conducted to assess other effects of cadmium at low levels of exposure.

Uses and Sources: Cadmium is a naturally occurring element, used for a variety of purposes including silver solder; metal plating; pigments in plastics, ceramics and glass; nickel-cadmium batteries; electronic devices; PVC stabilizers; coatings on steel and non-ferrous metals; components of specialized alloys; and as a catalyst. The largest sources of cadmium to the environment are fossil fuel emissions and incineration of municipal wastes. Cadmium is also released in smelter emissions. According to EPA's Toxics Release Inventory, reporting industries released more than 9 million pounds of cadmium and related compounds into the environment in 2000.

Major Consumer Products: Cadmium is used in nickel-cadmium (Ni-CD) batteries, pigments, and plastics.

Routes of Human Exposure: Because of cadmium's strong tendency to accumulate in plants and other living organisms, consumption of contaminated food is the primary non-occupational route of exposure to cadmium. People can also be exposed to cadmium by drinking contaminated water, by breathing contaminated air (including cigarette smoke), or by swallowing small quantities of contaminated soil or dust. Cigarette smokers can have approximately twice the cadmium in their bodies as non-smokers. Cadmium released into the air by industry, incinerators, smelters, and fossil fuel emissions is deposited onto soils or surface water where it tends to accumulate in plants (including food crops), fish, and shellfish. Cadmium is also a contaminant in sewage sludge from industrial sources. Crops grown on soils treated with sewage sludge can take up cadmium, increasing the potential for human exposure.

Health Effects: Once cadmium enters the body, it accumulates in soft tissues, primarily the liver and kidneys. Exposure to cadmium over time can result in accumulation of the metal in the body, which can have significant health significance for children. Human studies have shown that chronic exposure to cadmium can lead to serious health effects including lung cancer, emphysema and other lung diseases, and kidney damage. Limited evidence suggests that maternal exposure to cadmium can cause lower birth weight in infants. Psychological and neurological effects have been observed in children exposed to low levels of cadmium. Acute exposure to cadmium is rare, but can cause severe irritation of the stomach (by ingestion) and the lungs (by inhalation).

The U.S. National Toxicology Program now classifies cadmium and cadmium compounds as known human carcinogens based on evidence of lung and prostate cancer in humans.

Animal studies have shown that cadmium affects development, including low fetal weight, skeletal malformations, changes in fetal metabolism, and behavioral and neurological effects. Decreased reproduction and testicular damage have also been observed in animals exposed to cadmium. Other adverse effects shown in chronic animal studies include effects on the kidneys, liver, lungs, bone, immune system, blood and nervous system. There is little evidence for many of these effects in humans. It is important to note, however, that few human studies have assessed the effects of cadmium at low levels of exposure.

Detecting Exposure: Cadmium exposure can be measured by its presence in blood or urine. The amount of cadmium in blood is a good indicator of recent exposure, whereas the amount of cadmium in urine is more representative of total body burden. CDC's first National Exposure Report (2001) found that blood levels of cadmium were below the analytical detection limit for many participants. In urine samples, CDC reported a geometric mean urinary cadmium level of 0.32 µg/L in persons 6 years of age and older.

Regulations for Protection of Human Health:

Air – Cadmium compounds are regulated by EPA as Hazardous Air Pollutants under the Clean Air Act.

Drinking Water – EPA has established an MCL of 0.005 mg/L for cadmium in drinking water.

Food – FDA has set action levels of 3 ppm cadmium in crustacea and 4 ppm in clams, oysters, and mussels. FDA also limits cadmium in food colors and ceramic dishware.

Occupational Exposure – OSHA has established a PEL-TWA of 0.005 mg/m³ (5 µg/m³) for cadmium in workplace air.

Sources of Information:

ATSDR. *Toxicological Profile for Cadmium (Update)*. Agency for Toxic Substances and Disease Registry, Atlanta, GA. 1999. Accessed online at <http://www.atsdr.cdc.gov/toxprofiles/tp5.html>.

California Environmental Protection Agency (Cal/EPA). "Evidence on Developmental and Reproductive Toxicity of Cadmium." October 1996. Accessed online at <http://www.oehha.org/prop65/pdf/CD-HID.pdf>.

EPA. 2000 Toxics Release Inventory (TRI) Public Data Release Report. Office of Environmental Information, Washington, DC. EPA-260-R-02-003. Accessed online at <http://www.epa.gov/tri/tridata/tri00/pdr/index.htm>.

EPA. Technology Transfer Network Air Toxics Website, Health Effects Notebook: Cadmium and Compounds. Washington, DC. December 1994. Accessed online at <http://www.epa.gov/ttn/atw/hapindex.html>.

National Toxicology Program (NTP). Ninth Report on Carcinogens: Cadmium and Cadmium Compounds. U.S. Department of Health and Human Services, Research Triangle Park, NC. (2001).

Chemical Name: ANTIMONY

Summary: Antimony is a metal that is released into the environment during mining operations and from industrial discharges. Exposure to antimony is associated with damage to the heart, lungs, and other organs. There is limited evidence that chronic exposure can cause developmental and reproductive effects.

Uses and Sources: Antimony is used in alloys in the metals industry and to produce fireproofing chemicals, ceramics, ammunition, glassware, pigments, and other items. The primary source of antimony releases to the environment is mining and processing of gold ores. According to EPA's Toxic Release Inventory, reporting industries released almost 30 million pounds of antimony and related compounds into the environment in 2000.

Major Consumer Products: Antimony is used in a variety of products, including storage batteries, pewter, textiles, plastics, paints, ceramics, and enamels.

Routes of Human Exposure: People can be exposed to antimony by breathing contaminated air, drinking contaminated water, or by eating foods that contain this metal. Exposure can also occur through skin contact with contaminated soil or water. Industrial workers can be exposed to antimony by breathing dust or by skin contact.

Health Effects: Chronic exposure to antimony can cause lung and heart disease, increased blood pressure, stomach pain, diarrhea, vomiting, and stomach ulcers. There is limited evidence that antimony can cause developmental and reproductive effects. Animal studies have shown that long-term exposure to antimony can cause heart problems, effects on fertility, and lung cancer. EPA has not determined the potential for antimony to cause cancer in humans. One form of antimony (antimony trioxide) has been identified as a possible human carcinogen by the International Agency for Research on Cancer (IARC).

Detecting Exposure: Antimony is absorbed slowly in the body and can concentrate in liver, lungs, plasma, or red blood cells. It can be measured in the urine, feces, and blood for several days after exposure. CDC's first National Exposure Report (2001) reported a geometric mean urinary antimony level of 0.10 µg/L in persons 6 years of age and older.

Regulations for Protection of Human Health:

Air – Antimony compounds are regulated by EPA as Hazardous Air Pollutants under the Clean Air Act.

Drinking Water - EPA has established an MCL of 0.006 mg/L for antimony in drinking water.

Occupational Exposure – OSHA has established a PEL of 0.5 mg/m³ for antimony in workplace air.

Sources of Information:

ATSDR. *Toxicological Profile for Antimony and Compounds*. Agency for Toxic Substances and Disease Registry, Atlanta, GA. 1992. Accessed online at <http://www.atsdr.cdc.gov/toxprofiles/tp23.html>.

EPA. Integrated Risk Information System (IRIS): Antimony. Office of Research and Development, Washington, DC. Accessed online at <http://www.epa.gov/iris/index.html>.

EPA. 2000 Toxics Release Inventory (TRI) Public Data Release Report. Office of Environmental Information, Washington, DC. EPA-260-R-02-003. Accessed online at <http://www.epa.gov/tri/tridata/tri00/pdr/index.htm>.

Klaassen CD, ed. 1996. Casarett and Doull's Toxicology: The Basic Science of Poisons. New York: McGraw-Hill.

Chemical Name: BARIUM

Summary: Barium is a metal commonly found in soils, foods, surface water, and in public drinking water supplies. Barium is also released into the environment from industrial facilities. Exposure to barium can cause a variety of health effects, including damage to the liver, kidney, heart, and spleen. Exposure to barium can also cause difficulty breathing, muscle weakness, and effects on the nervous system.

Uses and Sources: Barium occurs naturally and is commonly found in soils, surface water, and public drinking water supplies. Barium compounds are used in a wide variety of materials including stabilizers for plastics, drilling tools for the oil and gas industry, case hardening steels, paints, bricks, tiles, lubricating oils, and jet fuels. Some barium compounds are used to make insect and rat poisons. Barium sulfate is used by doctors to perform medical tests and taking certain x-rays of the gastrointestinal tract. In the environment, barium typically combines with other elements. It can be taken up into edible plants from the soil. Barium is also discharged into surface water and air by industrial sources. According to EPA's Toxics Release Inventory, reporting industries released over 310 million pounds of barium and barium compounds into the environment in 2000.

Major Consumer Products: Used as contrast material in medical x-ray diagnostics.

Routes of Human Exposure: Barium is released into the environment by natural processes, industrial and manufacturing operations, and combustion of materials such as plastics, paints, oils, and jet fuels. The general population can be exposed by consuming contaminated drinking water or food or by inhaling barium in ambient air.

Health Effects: The health effects of barium depend, in part, on which form of the chemical enters the body. In general, ingestion of barium can cause difficulty breathing, increased blood pressure, changes in heart rhythm, stomach irritation, muscle weakness, changes in nerve reflexes, and damage to the liver, kidney, heart, and spleen. No reproductive or developmental effects have been linked to barium exposure, and EPA has concluded that barium is unlikely to cause cancer in humans by the ingestion route. There is inadequate data to evaluate the potential carcinogenicity of barium by inhalation.

Detecting Exposure: Barium leaves the body primarily in the feces, but can also be found in urine. Most barium leaves the body within a few days, and almost all is gone within 1-2 weeks. Barium remaining in the body is found in the bones (91%), with the remainder stored in soft tissues such as brain, heart, kidneys, and lungs. CDC's first National Exposure Report (2001) reported a geometric mean urinary barium level of 1.6 µg/L in persons 6 years of age and older.

Regulations for Protection of Human Health:

Air – Barium is not regulated in ambient air.

Drinking Water – EPA has established an MCL of 2 mg/L for barium in drinking water.

Occupational Exposure – OSHA has established a PEL of 0.5 mg/m³ for soluble barium compounds and 15 mg/m³ for barium sulfate dust in workplace air.

Sources of Information:

ATSDR. *Toxicological Profile for Barium*. Agency for Toxic Substances and Disease Registry, Atlanta, GA. 1992. Accessed online at <http://www.atsdr.cdc.gov/toxprofiles/tp24.html>.

EPA. 2000 Toxics Release Inventory (TRI) Public Data Release Report. Office of Environmental Information, Washington, DC. EPA-260-R-02-003. Accessed online at <http://www.epa.gov/tri/tridata/tri00/pdr/index.htm>.

EPA. Toxicological Review of Barium and Compounds. March 1998. Accessed online at <http://www.epa.gov/iris/toxreviews/0010-tr.pdf>.

Chemical Name : BERYLLIUM

Summary: Beryllium is a hard, grayish metal that occurs in certain rocks, coal and oil, soil, and volcanic dust. It enters the environment through natural processes as well as through mining operations and industrial releases. Inhaled beryllium can remain deposited in the lungs for long periods and can cause lung disease, lung cancer, and effects on the heart. Beryllium can also affect immune system function.

Uses and Sources: Most beryllium ore is used in alloys to make electrical and electronic parts, construction materials, and molds for plastics. Pure beryllium metal is used in nuclear weapons and reactors, aircraft and space vehicles, instruments, and x-ray machines. Beryllium occurs naturally, and can enter water sources from dissolved rocks or soil. Beryllium is also discharged into surface water and air from mining and ore processing facilities and industrial sources. Most beryllium in the atmosphere is from combustion of coal and oil. According to EPA's Toxics Release Inventory, reporting industries released over 940,000 pounds of beryllium and beryllium compounds into the environment in 2000.

Major Consumer Products: No major consumer products.

Routes of Human Exposure: People can be exposed to beryllium by breathing contaminated air, consuming food, or drinking water that contains beryllium. Cities have higher levels of beryllium in air, because it is released from burning coal and fuel oil. Beryllium can be found in drinking water supplies, but is not a common contaminant. It also occurs naturally in some foods. Workers involved with beryllium mining, processing, or other use can be exposed to higher-than-normal levels.

Health Effects: Inhalation exposure to beryllium has been associated with lung cancer in animals, and beryllium is likely to cause cancer in humans. Some forms of beryllium, when inhaled, can cause lung disease and effects on the heart. Animal studies have found gastrointestinal ulcers and effects on the kidneys. Exposure to beryllium can also affect immune system function. There is insufficient information to determine whether beryllium causes reproductive or developmental effects in humans.

Detecting Exposure: Most beryllium that is swallowed leaves the body within a few days through the feces without entering the bloodstream. Inhaled beryllium can be deposited in the lungs and can remain in the body for months to years. Beryllium can be measured in the urine and blood, but these levels may not accurately reflect the amount of exposure. In CDC's first National Exposure Report (2001), beryllium was not detected in urine of study participants (persons ages 6 and older).

Regulations for Protection of Human Health:

Air – Beryllium compounds are regulated by EPA as Hazardous Air Pollutants under the Clean Air Act.

Drinking Water – EPA has established an MCL of 0.004 mg/L for beryllium in drinking water.

Occupational Exposure – OSHA has established a PEL-TWA of 2 µg/m³ for beryllium and beryllium compounds in workplace air.

Sources of Information:

ATSDR. *Toxicological Profile for Beryllium (Update)*. Agency for Toxic Substances and Disease Registry, Atlanta, GA. September 2000. Accessed online at <http://www.atsdr.cdc.gov/toxprofiles/tp4.html>.

EPA. 2000 Toxics Release Inventory (TRI) Public Data Release Report. Office of Environmental Information, Washington, DC. EPA-260-R-02-003. Accessed online at <http://www.epa.gov/tri/tridata/tri00/pdr/index.htm>.

Chemical Name: CESIUM

Summary: Cesium is a soft, malleable, silver-white metal that naturally occurs in rocks, soil, and clay. For most people, ingestion of contaminated food is the most significant source of exposure to cesium. Naturally occurring cesium is not radioactive and has low toxicity to humans. However, radioactive forms of cesium, such as produced by nuclear power plants and nuclear weapon explosions, can cause serious health effects. In humans, radioactive cesium has been shown to cause reduced fertility. Animal studies have indicated potential for neurological, developmental, reproductive, genotoxic, and cancer-causing effects.

Uses and Sources: Because cesium is not mined in the U.S., domestic use is dependent on imports. Only small quantities of cesium are used in the U.S., primarily in research and development and in electronic, photoelectric, and medical applications. Most cesium in the environment is non-radioactive, and comes from natural erosion and weathering of rocks and minerals. Radioactive cesium is released into the environment during operation of nuclear power plants, explosions of nuclear weapons (especially atmospheric testing carried out in the past), and nuclear accidents. Cesium is also released in the ash of hazardous waste incinerators and coal-burning power plants. Some radioactive isotopes of cesium break down very slowly, and can remain in the environment for decades after release.

Major Consumer Products: No major consumer products.

Routes of Human Exposure: Although detailed exposure studies have not been conducted, researchers believe that people are exposed to relatively low levels of cesium through food, drinking water, air, and by skin contact with cesium in soil. Ingestion of cesium-contaminated food items is the most significant source of exposure to both naturally occurring and radioactive cesium. Cesium can travel long distances in the atmosphere, and be deposited onto land, water, and plant surfaces far from the original source. Cesium can be absorbed by vegetation, which is then consumed by animals and humans. Radioactive cesium has been detected in many types of food, including breast milk and pasteurized milk. People working in industries that process or use cesium, including workers in the nuclear power industry, may have above-average levels of exposure.

Health Effects: Radioactive cesium can seriously affect health, particularly following external exposure and eating contaminated food. Radioactive cesium emits beta particles and gamma rays, which can damage living cells. Reduced fertility has been observed in men exposed to radioisotopes of cesium. Animal studies have shown evidence of adverse neurological, developmental, reproductive, genotoxic, and cancer-causing effects following exposure to radioactive cesium. Long-term studies of cancer risk for humans exposed to radioactive cesium have not been conducted, and EPA has not classified cesium for human carcinogenicity. However, animal studies have suggested an increased potential for cancer, particularly at high doses.

Detecting Exposure: Urinalysis is the preferred test for analysis of cesium, although cesium can also be measured in blood, feces, and other tissues. Unlike some radioactive substances, cesium does not accumulate in any one part of the body. CDC's first National Exposure Report (2001) reported a geometric mean urinary cesium level of 4.7 µg/L in persons 6 years of age and older.

Regulations for Protection of Human Health:

Air – EPA does not regulate cesium in ambient air. The Nuclear Regulatory Commission (NRC) limits releases of cesium from commercial nuclear power plants to 2×10^{-10} µCi/mL.

Drinking Water – EPA has not established a drinking water standard or health advisory for cesium.

Occupational Exposure – NIOSH has established a Recommended Exposure Limit (REL) for cesium hydroxide of 2 mg/m³, as a time-weighted average for up to a 10-hour workday. The ACGIH has recommended a TLV of 2 mg/m³ for cesium hydroxide over an 8-hour workday. The NRC regulates occupational inhalation exposure to radioactive cesium.

Source of Information:

ATSDR. *Draft Toxicological Profile for Cesium*. Agency for Toxic Substances and Disease Registry, Atlanta, GA. 2001. Accessed online at <http://www.atsdr.cdc.gov/toxprofiles/tp157.html>

Chemical Name: COBALT

Summary: Cobalt is a relatively rare, naturally occurring metal that is used in a variety of products and industries. There is one stable (non-radioactive) form of cobalt (Co-59), and many radioactive forms. Cobalt can enter the environment naturally or through human activity. In small quantities, stable cobalt is essential for health, but in higher doses, it can have serious health effects such as lung disease and possibly effects on the heart and other organs. Radioactive cobalt is known to cause cancer, and exposure can damage organ systems throughout the body. The developing fetus is particularly sensitive to the effects of radioactive cobalt.

Uses and Sources: Small amounts of cobalt occur naturally in rocks, soil, water, plants, and animals, usually combined with other elements. Cobalt is not mined in the U.S., but is obtained from imported materials and scrap metal recycling. Cobalt is used in the military and industry; the largest use is in superalloys used for aircraft engines. Cobalt metal is also used to dry paint and porcelain enamel, in colored pigments, and as catalysts in the petroleum industry. Radioactive isotopes of cobalt, generated by nuclear reactions, are used in medical and research applications, consumer products, and for food irradiation. Human activity contributes to environmental cobalt contamination through combustion of fossil fuels, sewage sludge, fertilizers, mining, smelting, and processing of cobalt. Once released into the environment, cobalt is not destroyed, but radioactive isotopes undergo decay. According to EPA's Toxics Release Inventory, reporting industries released more than 17 million pounds of cobalt and cobalt compounds in 2000.

Major Consumer Products: Cobalt is used in artificial replacement joints.

Routes of Human Exposure: For most people, food is the most important source of cobalt exposure. Exposure to cobalt can also occur by breathing contaminated air, drinking water, and contact with soil or other materials containing cobalt. Cobalt is found in tea, coffee, many fruits and vegetables, and in some fish. The general public is not exposed to significant levels of radioactive cobalt. Workers involved in processing or working in industries using cobalt may have higher-than-average exposure to stable cobalt and workers at nuclear facilities, irradiation facilities, and nuclear waste storage sites may be exposed to radioactive forms of cobalt.

Health Effects: Cobalt is a component of vitamin B₁₂ and is an essential nutrient in very small quantities. At high levels of exposure, such as in the workplace, cobalt can cause serious health effects in humans. By inhalation, cobalt exposure can cause serious effects on the lungs, cardiovascular system, liver, kidney, and eyes. Stable cobalt has not been shown to cause cancer in humans, but animal studies suggest it may have carcinogenic potential.

Exposure to radioactive forms of cobalt can damage the lungs, gastrointestinal system, bone marrow, nervous system, and reproductive system. Radioactive cobalt is classified by EPA as a known human carcinogen. Animal studies have found that the developing fetus is particularly sensitive to the effects of exposure to radioactive cobalt.

Detecting Exposure: Analysis of urine and feces are the preferred samples for analysis of cobalt. CDC's first National Exposure Report (2001) reported a geometric mean urinary cobalt level of 0.36 µg/L in persons 6 years of age and older.

Regulations for Protection of Human Health:

Air – Cobalt compounds are regulated by EPA as Hazardous Air Pollutants under the Clean Air Act. Emissions of radioactive cobalt into air are regulated by the Department of Energy and the Nuclear Regulatory Commission.

Drinking Water – EPA has not established a drinking water standard or health advisory for stable cobalt. EPA has developed relevant standards for radioactive cobalt in drinking water. The MCL for particle and photon activity is 4 millirems/yr, and the MCL for gross alpha particle activity is 15 picoCuries per liter (pCi/L).

Occupational Exposure – OSHA has established a PEL of 0.1 mg/m³ for cobalt in workplace air.

Sources of Information:

ATSDR. *Draft Toxicological Profile for Cobalt*. Agency for Toxic Substances and Disease Registry, Atlanta, GA. September 2001. Accessed online at <http://www.atsdr.cdc.gov/toxprofiles/tp33.html>.

EPA. 2000 Toxics Release Inventory (TRI) Public Data Release Report. Office of Environmental Information, Washington, DC. EPA-260-R-02-003. Accessed online at <http://www.epa.gov/tri/tridata/tri00/pdr/index.htm>.

Klaassen CD, ed. 1996. Casarett and Doull's Toxicology: The Basic Science of Poisons. New York: McGraw-Hill.

Chemical Name: MOLYBDENUM

Summary: Molybdenum is a silvery-white metal that is an essential human nutrient in very small doses. There is limited information available regarding human health effects of exposure to excess molybdenum. Animal studies have shown potential for serious effects, including reproductive effects, organ failure, and skeletal abnormalities.

Uses and Sources: Molybdenum is used in some nickel-based alloys, which are heat-and corrosion-resistant. It is also used in electrical applications, as a catalyst in petroleum refining, in nuclear energy production, and for missile and aircraft parts. Molybdenum is recovered as a byproduct of tungsten and copper mining. Molybdenum can be released to the environment from mining/processing operations and through industrial discharges. Molybdenum releases into the environment are not tracked under EPA's Toxics Release Inventory.

Major Consumer Products: No major consumer products.

Routes of Human Exposure: Exposure to molybdenum is highest in persons involved in production or manufacture of molybdenum products. Molybdenum can also be found in ambient air, drinking water, and naturally in certain foods (soy products, grains, and nuts).

Health Effects: Small quantities of molybdenum are required for normal body functions. Molybdenum compounds have relatively low toxicity, although there are limited data for human toxicity. In laboratory animals, molybdenum exposure has caused weight loss, kidney failure, skeletal abnormalities, infertility, and other effects. The relevance of these findings to humans is uncertain. Reproductive effects have also been observed in animals exposed to molybdenum. There is no evidence that molybdenum causes cancer in humans.

Detecting Exposure: Excess molybdenum is rapidly excreted in the urine. CDC's first National Exposure Report (2001) reported a geometric mean urinary molybdenum level of 48.4 $\mu\text{g/L}$ in persons 6 years of age and older.

Regulations for Protection of Human Health:

Air – Molybdenum is not regulated in ambient air.

Drinking Water – EPA has not established an MCL for molybdenum in drinking water. EPA has developed a health advisory for children of 0.08 mg/L and a lifetime health advisory of 0.04 mg/L of molybdenum in drinking water.

Occupational Exposure – OSHA has established a PEL of 5 mg/m³ for soluble molybdenum compounds and 15 mg/m³ for insoluble compounds in workplace air.

Sources of Information:

National Academy of Sciences. Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc. Institute of Medicine. National Academy Press, Washington, DC. (2001).

National Library of Medicine. TOXNET, Hazardous Substances Data Bank (HSDB). Accessed online at <http://www.toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB>.

Chemical Name : PLATINUM

Summary: Platinum is a naturally occurring silver-gray, malleable metal. In metallic form, platinum has low toxicity. Platinum dusts and complexes can have potentially serious health effects, including neuromuscular toxicity, kidney toxicity, and cancer.

Uses and Sources: Platinum occurs in nature both as pure metal and combined with other metals. Platinum is used in jewelry, in electrical applications, as a catalyst in industrial and chemistry applications, as an anti-tumor agent, and in prosthetic and biomedical devices. Platinum releases into the environment are not tracked under EPA's Toxics Release Inventory.

Major Consumer Products: Jewelry, prosthetic devices.

Routes of Human Exposure: Potential for exposure to platinum is highest for individuals involved in mining and processing operations and in industrial operations using platinum. Exposure can occur by breathing platinum dusts.

Health Effects: Platinum is relatively non-toxic in the metallic state. Exposure to platinum dust can cause breathing difficulty, and platinum salts can cause neuromuscular toxicity and kidney toxicity. Animal studies have shown that platinum can cross the placenta and reach the fetus. Some platinum complexes may cause cancer, but there is no evidence of increased cancer risk from occupational exposure to platinum compounds.

Detecting Exposure: Animal studies have found that almost all ingested platinum is readily excreted in the urine and feces. In CDC's first National Exposure Report (2001), platinum levels were below the analytical detection limit.

Regulations for Protection of Human Health:

Air – Platinum is not regulated in ambient air.

Drinking Water – EPA has not established a drinking water standard or health advisory for platinum.

Occupational Exposure – OSHA has established a PEL of 0.002 mg/m³ for soluble platinum salts in workplace air.

Sources of Information:

Klaassen CD, ed. 1996. Casarett and Doull's Toxicology: The Basic Science of Poisons. New York: McGraw-Hill.

National Library of Medicine. TOXNET, Hazardous Substances Data Bank (HSDB). Available online at Accessed online at <http://www.toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB>

Chemical Name: THALLIUM

Summary: Thallium is a naturally occurring metal that is used in a variety of industrial applications. Little is known about the effects of long-term, low-level exposure to thallium. There is evidence from animal studies that thallium exposure can cause organ damage and may have toxic effects on the reproductive system.

Uses and Sources: Thallium is used in the manufacture of electronic devices, switches, and closures. Thallium is no longer produced in the U.S., but is imported. Thallium occurs naturally in the environment, primarily as salts. In addition to natural sources, thallium is released into the atmosphere from coal-burning power plants, cement factories, and smelters. Thallium in the air can deposit onto soil and surface water, potentially contaminating food and drinking water sources. According to EPA's Toxics Release Inventory, reporting industries released more than 5 million pounds of thallium and thallium compounds into the environment in 2000.

Major Consumer Products: No major consumer products.

Routes of Human Exposure: The primary source of exposure to thallium is consumption of fruits and vegetables that are grown in thallium-contaminated soil (including home-grown produce). Cigarette smoking is also a source of thallium exposure. People who work in power plants, cement factories, and smelters can be exposed to thallium.

Health Effects: Exposure to thallium in large amounts can damage the nervous system, lungs, heart, liver and kidneys. Symptoms of exposure to high doses include vomiting, diarrhea, and possibly death. Little is known about the human health effects of long-term, low-level exposure to thallium. Animal studies have found reproductive effects (damage to testes) after drinking small quantities of thallium-contaminated water. There is insufficient information to determine whether thallium causes cancer in humans.

Detecting Exposure: Thallium is rapidly absorbed and distributed in the body (primarily the kidneys and liver). It leaves the body slowly, and can be found in urine as long as 2 months after exposure. CDC's first National Exposure Report (2001) reported a geometric mean urinary thallium level of 0.19 µg/L in persons 6 years of age and older.

Regulations for Protection of Human Health:

Air – Thallium is not regulated in ambient air.

Drinking Water – EPA has established an MCL of 0.002 mg/L for thallium in drinking water.

Occupational Exposure – OSHA has established a PEL of 0.1 mg/m³ for thallium in workplace air.

Sources of Information:

ATSDR *Toxicological Profile for Thallium*. Agency for Toxic Substances and Disease Registry, Atlanta, GA. 1992. Accessed online at <http://www.atsdr.cdc.gov/toxprofiles/tp54.html>.

EPA. 2000 Toxics Release Inventory (TRI) Public Data Release Report. Office of Environmental Information, Washington, DC. EPA-260-R-02-003. Accessed online at <http://www.epa.gov/tri/tridata/tri00/pdr/index.htm>.

Chemical Name: TUNGSTEN

Summary: Pure tungsten is a pure-white to tin-gray metal. Very little is known about the health effects of tungsten, but there is evidence that tungsten may cause lung damage.

Uses and Sources: Most of the tungsten used in the U.S. is for cemented carbide parts for metalworking, oil and gas drilling, mining, and construction industries. Tungsten is also used in lamp filaments, electrodes, and other electrical and electronics components; in steels and alloys; and as chemicals for catalysts and pigments. Tungsten can be released into the environment through mining operations and through industrial releases to air, water, or land. Tungsten releases into the environment are not tracked under EPA's Toxics Release Inventory.

Major Consumer Products: No major consumer products.

Routes of Human Exposure: Individuals working in tungsten mining and processing or in industries using tungsten, have a higher exposure potential than the general public. The primary route of exposure is by inhalation.

Health Effects: There is very little information available regarding the toxicity of tungsten. Tungsten carbide is toxic by the inhalation route, and chronic exposure by this route can cause lung damage. Symptoms of exposure to tungsten carbide include wheezing, coughing, difficulty breathing, and cardiac failure. EPA has not evaluated the potential carcinogenicity of tungsten.

Detecting Exposure: Tungsten levels can be measured in urine. CDC's first National Exposure Report (2001) reported a geometric mean urinary tungsten level of 0.10 µg/L in persons 6 years of age and older.

Regulations for Protection of Human Health:

Air – Tungsten is not regulated in ambient air.

Drinking Water – EPA has not established a drinking water standard or health advisory for tungsten.

Occupational Exposure – OSHA has not established a standard for tungsten in workplace air.

Sources of Information:

National Toxicology Program (NTP). Chemical Repository: Tungsten Carbide. August 1991. Radian Corp. Accessed online at http://ntp-db.niehs.nih.gov/NTP_Reports/NTP_Chem_H&S/NTP_Chem1/Radian12070-12-1.txt.

Chemical Name : URANIUM

Summary: Uranium is a naturally occurring, radioactive heavy metal. Although uranium is radioactive, the weak radiation cannot penetrate the skin to enter the body. Exposure occurs by ingesting uranium in food or water or by breathing contaminated air. Available studies have shown potential for serious health effects from exposure to uranium. These include kidney disease, heart and lung damage, developmental effects, and possibly cancer.

Uses and Sources: The primary uses of uranium are in nuclear power plants, on helicopters and airplanes, as shielding to protect Army tanks, in nuclear weapons, and in ammunition. Small quantities of uranium are also used to make some ceramic glazes, light bulbs, photographic chemicals, and household products. It is found in low levels in rocks, soil, surface water and groundwater, air, and plants and animals. Uranium can also enter the environment from human activity, including uranium mining and processing; use in nuclear energy and nuclear weapons; phosphate fertilizers; or accidental releases. Uranium releases into the environment are not tracked under EPA's Toxics Release Inventory.

Major Consumer Products: No major consumer products.

Routes of Human Exposure: People can be exposed to uranium from air, water, food, and soil. Root vegetables, such as beets and potatoes, tend to take up more uranium from soil than other foods. People who work in industries where uranium is processed or used can be exposed to higher-than-normal levels of this element.

Health Effects: Kidney disease has been observed in people and animals exposed to large amounts of uranium. Animal studies suggest that the lung and heart can be damaged from exposure to uranium. There is insufficient evidence to determine whether uranium causes cancer in humans. However, it is known that long-term exposure to radioactive substances can cause cancer. It is not known whether uranium causes reproductive effects in humans. Animal studies have shown reduced sperm counts in animals exposed to high levels of uranium. There is evidence that uranium can have adverse effects on the developing fetus. Studies of animals exposed to uranium have found higher levels of fetal death, reduced growth, smaller litter sizes, and birth defects.

Detecting Exposure: When uranium is inhaled, a portion remains in the lungs. Most ingested uranium, from food or water, leaves the body in the urine and feces within a few days. Some uranium can remain in the bones, kidneys, or other soft tissues where it can remain for years.

Regulations for Protection of Human Health:

Air – EPA regulates uranium releases into ambient air from a variety of industrial sources. For more information, contact EPA or visit the EPA Office of Air and Radiation website at <http://www.epa.gov/oar/>.

Drinking Water – EPA has established an MCL of 30 µg/L for uranium in drinking water.

Occupational Exposure – OSHA has established a PEL of 0.05 mg/m³ for soluble uranium compounds and 0.25 mg/m³ for insoluble compounds in workplace air.

Source of Information:

ATSDR. *Toxicological Profile for Uranium (Update)*. Agency for Toxic Substances and Disease Registry, Atlanta, GA, 1999. Accessed online at <http://www.atsdr.cdc.gov/toxprofiles/tp150.html>