Radiological and Chemical Fact Sheets to Support Health Risk Analyses for Contaminated Areas



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These fact sheets summarize health-related information for contaminants present in the environment as a result of past industrial activities and other releases. The objective is to provide scientific context for risk analyses to guide health protection measures. Geared toward an audience familiar with basic risk concepts, they were originally developed for the U.S. Department of Energy (DOE) Richland and Chicago Operations Offices to serve as an information resource for people involved in environmental programs. The initial set was expanded to address evolving homeland security concerns, and these 51 radiological and chemical fact sheets also serve as a scientific information resource for the public.

Twenty-nine radionuclide-specific fact sheets have been prepared:

- Americium
- Cadmium
- Californium
- Carbon-14
- Cesium
- Chlorine
- Cobalt
- Curium
- Europium
- Iodine
- Iridium
- Krypton
- Neptunium
- Nickel
- Plutonium
- Polonium
- Potassium-40
- Protactinium
- Radium
- Samarium
- Selenium
- Strontium
- Technetium
- Thorium
- Tin
- Tritium
- Uranium
- Depleted uranium (DU)
  complements uranium sheet
- Zirconium

Four companion fact sheets present basic radiological concepts:

- Ionizing radiation
- Natural decay series (radium, thorium, and uranium)
- Transuranic radionuclides and decay series (plutonium and others)
- Radioactive properties, internal distribution, and risk coefficients

Two additional sheets directly address the homeland security context for a subset of radionuclides:

- Radiological dispersal device
- Health-based radionuclide concentrations in drinking water and air

Several of the radiological fact sheets also include information for chemical toxicity, notably those for cadmium, chlorine, nickel, selenium, strontium, and uranium. In addition to the radiological fact sheets, chemical-specific fact sheets have been prepared for 7 nonradioactive metals; 5 organic compounds (including one class of compounds); and 2 inorganic ions (with one covering a pair of anions):

- Arsenic
- Beryllium
- Chromium
- Copper
- Lead *(includes link to radiological information)*
- Mercury
- Zinc
- Carbon tetrachloride
- Chloroform
- Polychlorinated biphenyls (PCBs)
- Trichloroethane
- Trichloroethylene
- Cyanide
- Nitrate/nitrite

For cumulative risk analyses, the evaluation of interactions is framed by two fact sheets for mixtures:

- Mixtures concepts
- Arsenic, cadmium, chromium, and lead

These 51 fact sheets are provided in alphabetical order in this report except DU, which follows the general sheet for uranium, and the two mixtures fact sheets, which are provided at the end. Each contaminant-specific fact sheet presents brief information on:

- Key properties, origin, and use
- General environmental levels
- Distribution in the body
- Primary health effects
- Values for estimating risk

Additional information provided separately includes a summary of radionuclide morbidity and mortality risk coefficients for key isotopes (Table 1), a summary of the radioactive properties of these isotopes (Table 2), and source references for the radiological fact sheets (Table 3). For the chemicals, parallel information (chemical toxicity values, illustration of organs affected, and references) is included within the individual fact sheets.

(Note: These fact sheets have been prepared at different times to support various program needs; the preparation date is identified in the header of each. To open a given fact sheet, click on the link provided within the list on the previous page.)

Americium

What Is It? Americium is a malleable, silvery white metal that tarnishes slowly in dry air at room temperature. Americium does not occur naturally but is produced artificially by successive neutron capture reactions by plutonium isotopes. There are sixteen known isotopes of americium and all of them are radioactive. (Isotopes are different forms of an element that have the same number of protons in the nucleus but a different number of neutrons.) Americium-241 was first produced in 1944 in a nuclear reactor at the University of Chicago. Dr. Glenn Seaborg gave the new element its name in 1946 in honor of the continent on which it was discovered.

Symbol: **Am**

Atomic Number: **95**
(protons in nucleus)

Atomic Weight: **-**
(not naturally occurring)

Of the sixteen radioactive isotopes, only three have half-lives long enough to warrant concern at Department of Energy (DOE) environmental management sites: americium-241, americium-242m, and americium-243. The half-lives of these three isotopes range from 150 to 7,400 years, while those of the other isotopes are less than a day. Americium-241 is generally the most prevalent isotope at DOE sites such as Hanford. It has a half-life of 430 years and decays by emitting an alpha particle with attendant gamma radiation. The other two isotopes typically represent less than a few percent of the total americium inventory at a site. Americium-242m (the "m" means metastable) has a half-life of 150 years, and it decays by isomeric transition. Americium-243 is generally not a major concern at DOE sites given its low abundance relative to americium-241 and low specific activity.

Radioactive Properties of Key Americium Isotopes and Associated Radionuclides

Isotope	Half-Life	Specific Activity *(Ci/g)*	Decay Mode	Radiation Energy *(MeV)*		
				Alpha *(α)*	Beta *(β)*	Gamma *(γ)*
Am-241	430 yr	3.5	α	5.5	0.052	0.033
Am-242m	150 yr	9.8	IT	0.025	0.044	0.0051
Am-242	*16 hr*	*820,000*	*β, EC*	-	*0.18*	*0.018*
Am-243	7,400 yr	0.20	α	5.3	0.022	0.055
Np-239	*2.4 days*	*230,000*	*β*	-	*0.26*	*0.17*

IT = isomeric transition, EC = electron capture, Ci = curie, g = gram, and MeV = million electron volts; a dash means that the entry is not applicable. (See the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients for an explanation of terms and interpretation of radiation energies.) Americium-242 decays by two means: by emitting a beta particle (83%) and by electron capture (17%). Certain properties of americium-242 and neptunium-239 are included here because these radionuclides accompany the americium decays. Values are given to two significant figures.

Where Does It Come From? Americium is a byproduct of plutonium production activities and results from the successive capture of neutrons by plutonium. The most common isotope is americium-241, a decay product of plutonium-241. When plutonium-239 absorbs two neutrons it produces plutonium-241, which decays by emitting a beta particle with a fairly short half-life of 14 years to generate americium-241. Americium-243 is produced in a similar manner from the decay of plutonium-243, which decays by emitting a beta particle with a half-life of 5 hours. Successive neutron absorptions of the isotope americium-241can produce both americium-242m and americium-243.

How Is It Used? The most common use of americium is in smoke detectors. These detectors rely on the alpha particle associated with the decay of americium-241 to ionize the air in a gap between two electrodes, causing a very small electrical current to flow between them. When smoke enters the space between the electrodes, the alpha radiation is absorbed by the soot particles, the current is interrupted, and the alarm is sounded. Alpha particles from smoke detectors do not themselves pose a health hazard, as they are absorbed in a few centimeters of air or by the structure of the detector. Americium is also used as a portable source for gamma radiography, for crystal research, and as target material in nuclear reactors or particle accelerators to produce even heavier elements. A common neutron source is composed of americium-241 and beryllium. The alpha particle given off during the radioactive decay of americium-241 is absorbed by beryllium-9, producing carbon-12 and a neutron. Such devices can be used for the nondestructive testing of machinery and equipment and for other industrial applications.

What's in the Environment? Atmospheric testing of nuclear weapons, which ceased worldwide by 1980, generated most environmental americium. Accidents and other releases from weapons production facilities have caused localized contamination. Americium oxide is the most common form in the environment. Average americium-241 levels in surface soil are about 0.01 picocuries (pCi)/g. Americium is typically quite insoluble, although a small fraction can become soluble through chemical and biological processes. It adheres very strongly to soil, with americium concentrations associated with sandy soil particles estimated to be 1,900 times higher than in interstitial water (the water in the pore spaces between the soil particles); it binds more tightly to loam and clay soils so those concentration ratios are even higher. At DOE sites such as Hanford, americium can be present in areas that contain waste from the processing of irradiated fuel.

What Happens to It in the Body? Americium can be taken into the body by eating food, drinking water, or breathing air. Gastrointestinal absorption from food or water is a likely source of internally deposited americium in the general population. After ingestion or inhalation, most americium is excreted from the body within a few days and never enters the bloodstream; only about 0.05% of the amount taken into the body by ingestion is absorbed into the blood. After leaving the intestine or lung, about 10% clears the body. The rest of what enters the bloodstream deposits about equally in the liver and skeleton where it remains for long periods of time, with biological retention half-lives of about 20 and 50 years, respectively (per simplified models that do not reflect intermediate redistribution). The amount deposited in the liver and skeleton depends on the age of the individual, with fractional uptake in the liver increasing with age. Americium in the skeleton is deposited uniformly on cortical and trabecular surfaces of bones and slowly redistributes throughout the volume of mineral bone over time.

What Are the Primary Health Effects? Americium is generally a health hazard only if it is taken into the body, although there is a small risk associated with the gamma rays emitted by neptunium-239, a radioactive decay product of americium-243. The main means of exposure are ingestion of food and water containing americium isotopes and inhalation of americium-contaminated dust. Ingestion is generally the exposure of concern unless there is a nearby source of contaminated airborne dust. Because americium is taken up in the body much more readily if inhaled rather than ingested, both exposure routes can be important. The major health concern is tumors resulting from the ionizing radiation emitted by americium isotopes deposited on bone surfaces and in the liver.

What Is the Risk? Lifetime cancer mortality risk coefficients have been calculated for nearly all radionuclides, including americium *(see box at right).* While ingestion is generally the most common type of exposure, the risk coefficients for this route are much lower than those for inhalation. As for other nuclides, the coefficient for tap water is about 80% of that shown for dietary ingestion.

In addition to risks from internal exposures, there is an external gamma exposure risk associated with americium-243. To estimate a lifetime cancer mortality risk, if it is assumed that 100,000 people were continuously exposed to a thick layer of soil with an initial average concentration of 1 pCi/g americium-243, then 3 of these 100,000 people would be predicted to incur a fatal cancer. (This is in comparison to the 20,000 people from the group predicted to die of cancer from all other causes per the U.S. average.) This risk is largely associated with the gamma ray emitted by its short-lived decay product neptunium-239. The external risk for the other two americium isotopes is less than 10% of that for americium-243.

Radiological Risk Coefficients

This table provides selected risk coefficients for inhalation and ingestion. Recommended default absorption types were used for inhalation, and dietary values were used for ingestion. These values include the contributions from the short-lived americium decay products. Risks are for lifetime cancer mortality per unit intake (pCi), averaged over all ages and both genders (10^{-9} is a billionth, and 10^{-12} is a trillionth). Other values, including for morbidity, are also available.

Isotope	Lifetime Cancer Mortality Risk	
	Inhalation (pCi^{-1})	Ingestion (pCi^{-1})
Americium-241	2.4×10^{-8}	9.5×10^{-11}
Americium-242m	1.3×10^{-8}	6.8×10^{-11}
Americium-243	2.3×10^{-8}	9.8×10^{-11}

For more information, see the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients and the accompanying Table 1.

Cadmium

Symbol:	**Cd**
Atomic Number: *(protons in nucleus)*	**48**
Atomic Weight: *(naturally occurring)*	**112**

What Is It? Cadmium is a soft, silvery gray metal that is malleable and ductile, similar to zinc. When heated, it burns in air with a bright light to form the oxide CdO. In nature, essentially all cadmium exists as seven stable isotopes and one radioactive isotope. (Isotopes are different forms of an element that have the same number of protons in the nucleus, but a different number of neutrons.) The seven stable isotopes and their approximate abundances are cadmium-106 (1.3%), cadmium-108 (0.9%), cadmium-110 (12%), cadmium-111 (13%), cadmium-112 (24%), cadmium-114 (29%), and cadmium-116 (7.5%). The primary radioactive isotope, cadmium-113, comprises about 12% of natural cadmium and has an extremely long half-life.

Nine major radioactive isotopes of cadmium exist, of which only three – cadmium-109, cadmium-113, and cadmium-113m – have half-lives long enough to warrant potential concern. The half-lives of the other six are less than 45 days. Cadmium-109 decays by electron capture with a half-life of 1.3 years, so any that was produced more than 20 years ago has long since decayed away. The other two cadmium isotopes decay by emitting a beta particle. The very low specific activity of cadmium-113 limits its radioactive hazards. Thus, cadmium-113m (the "m" means metastable) is the isotope of most concern at Department of Energy environmental management sites such as Hanford. Cadmium-113m decays by emitting a beta particle with no gamma radiation.

Radioactive Properties of Key Cadmium Isotopes

Isotope	Half-Life *(yr)*	Natural Abundance *(%)*	Specific Activity *(Ci/g)*	Decay Mode	Radiation Energy *(MeV)*		
					Alpha *(α)*	Beta *(β)*	Gamma *(γ)*
Cd-109	1.3	<<1	2,600	EC	-	0.083	0.026
Cd-113	9.3 million billion	12	0.34 trillionth	β	-	0.093	-
Cd-113m	14	<<1	240	β	-	0.019	-

EC = electron capture, Ci = curie, g = gram, and MeV = million electron volts; a dash means the entry is not applicable. (See the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients for an explanation of terms and interpretation of radiation energies.) Values are given to two significant figures.

Where Does It Come From? Cadmium is found in rare ores such as sphalerite and greenockite, and it is formed as a byproduct during production of zinc, copper, and lead. The United States is among the top ten producers, refining over 1,000 metric tons of cadmium a year. The majority of cadmium that enters the environment is from mining, smelting, oil and coal combustion, and waste incineration. Cadmium-113m is produced by neutron activation of the stable isotope cadmium-112 and as a fission product. When a fissile nuclide such as uranium-235 fissions, it generally splits asymmetrically into two large fragments – fission products with mass numbers in the range of about 90 and 140 – and two or three neutrons. In a nuclear reactor, these neutrons can cause additional fissions (producing a chain reaction), escape from the reactor, or irradiate nearby materials. Cadmium is used in reactor components such as control rods and shields to absorb neutrons, resulting in the formation of various isotopes including cadmium-113m. The fission yield of cadmium-113m is very low (about 0.0002%). This radioactive isotope is present in spent nuclear fuel and radioactive wastes associated with operating nuclear reactors and fuel reprocessing plants.

How Is It Used? Most cadmium in the United States (about 75%) is used in nickel-cadmium batteries. It has also been used as an anticorrosive coating for steel and cast iron, and it is a component of certain specialty alloys. Cadmium is used in semiconductors (such as cadmium selenide and telluride), in dyes and pigments, as a stabilizer in plastics such as polyvinyl chloride, and as a neutron absorber in nuclear reactor control rods and shields. Its use in the United States has recently decreased by about 50% in response to environmental concerns.

What's in the Environment? Nonradioactive cadmium is present in U.S. soil at an average concentration of about 0.15 milligram per kilogram (mg/kg). Trace amounts of cadmium-113m are present in soil around the world from radioactive fallout due to past atmospheric weapons tests. It may also be present at certain nuclear facilities, such as reactors and facilities that process spent nuclear fuel. Cadmium is usually relatively immobile, with concentrations in sandy soil estimated to be 80 times higher than in interstitial water (water in the pore space between the soil particles); it is even less mobile

in clay soils, with estimated concentration ratios above 500. The typical ratio of the concentration of cadmium in plants to that in soil is estimated at 0.15 (or 15%). (Cadmium-113m is generally not a major contaminant in groundwater at DOE sites like Hanford because of its relatively low concentration in wastes as a result of its low fission yield.)

What Happens to It in the Body? Cadmium can be taken into the body by eating food, drinking water, breathing air, or smoking a cigarette. Children, and to a lesser extent adults, can also be exposed by ingesting soil. Gastrointestinal absorption from food or water is the principal source of internally deposited cadmium in the general population. Gastrointestinal absorption is generally quite low, with only about 5% of the amount ingested being transferred to the bloodstream; the unabsorbed cadmium is excreted in the feces. Cadmium absorption via inhalation exposure is higher, estimated at 30 to 60%. Little metabolic conversion of cadmium compounds occurs in the body, but cadmium does bind to proteins and other components of macromolecules. Thirty percent of cadmium that reaches the blood deposits in the liver, another 30% deposits in the kidneys, and the rest distributes throughout all other organs and tissues of the body (per simplified models that do not reflect intermediate redistribution). Absorbed cadmium is excreted primarily in the urine, but because daily excretion is only about 0.01% of the total body burden, the biological half-life is about 25 years. Dermal contact is not usually of concern because the amount absorbed is small (about 0.1%).

What Are the Primary Health Effects? Cadmium-113m is a health hazard only if it is taken into the body. It does not pose an external hazard because it decays by emitting a relatively low-energy beta particle with no gamma radiation. While it concentrates in the liver and kidneys, cadmium can also deposit in other organs and tissues depending on its chemical form. The main concern is cancer induction from the beta particles associated with its radioactive decay, but cadmium also exhibits chemical toxicity, with health effects following inhalation exposure to high levels that can include damage to the respiratory system (bronchial and pulmonary irritation), headache, chest pains, muscular weakness, pulmonary edema, and death. Chronic exposure may result in emphysema and chronic bronchitis. Repeated low exposures may also cause permanent kidney damage, leading to kidney stones and other health problems. In its narrative for the cancer weight of evidence, the U.S. Environmental Protection Agency (EPA) states that occupational studies of cadmium smelter workers developing lung cancer provide limited evidence for the carcinogenicity of cadmium in humans following inhalation exposure, and that there is sufficient evidence of carcinogenicity in rats and mice by inhalation and intramuscular and subcutaneous injection. Cadmium was classified as a probable human carcinogen under the EPA 1996 cancer guidelines. In contrast, cadmium has not been shown to cause cancer when ingested. Information on the joint toxicity of cadmium with other metals is provided in the companion chemical mixtures fact sheet.

What Is the Risk? Lifetime cancer mortality risk coefficients have been calculated for nearly all radionuclides, including cadmium *(see box at right).* While the coefficients for ingestion are lower than for inhalation, ingestion is generally the most common means of entry into the body. Similar to other radionuclides, the risk coefficients for tap water are about 80% of those for dietary ingestion. The EPA has also developed toxicity values to estimate the risk of developing cancer or other adverse health effects as a result of inhaling or ingesting cadmium. The toxicity value for estimating the risk of cancer for inhalation exposure is called a unit risk (UR), which is an estimate of the chance that a person will get cancer from continuous exposure to a chemical in air at a concentration of 1 milligram per cubic meter (mg/m^3). The value for the non-cancer effect following oral exposure is a reference dose (RfD), which is an estimate of the highest dose that can be taken in every day without causing an adverse non-cancer effect. The UR is based on studies of humans exposed to cadmium in the workplace. Oral RfDs for food and water, shown at right, were developed using a toxicokinetic model that relates cadmium intake to concentrations in the kidney. To illustrate how the RfD is applied, a 150-pound (lb) person could safely ingest food containing 0.068 mg or drink water containing 0.034 mg of cadmium every day without expecting any adverse effects (2.2 lb = 1 kg, or 1,000 g, or 1 million mg). In contrast to the RfD, which represents a "safe daily dose" (and so is compared to the amount an individual takes in, as a ratio), the UR is multiplied by the air concentration to estimate the cancer risk. Using the UR, the EPA estimates that a person would have a one-in-a-million chance of developing cancer if exposed to air containing 0.0006 microgram ($\mu g)/m^3$ every day over a lifetime. (A microgram is one millionth of a gram.)

Radiological Risk Coefficients

The following table provides selected radiological risk coefficients for inhalation and ingestion. Maximum values are given for inhalation (no default absorption types were provided), and dietary values were used for ingestion. Risks are for lifetime cancer mortality per unit intake (picocurie, pCi), averaged over all ages and both genders (10^{-9} is a billionth, and 10^{-12} is a trillionth). Other values, including for morbidity, are also available.

Isotope	Lifetime Cancer Mortality Risk	
	Inhalation (pCi^{-1})	Ingestion (pCi^{-1})
Cadmium-109	2.0×10^{-11}	4.2×10^{-12}
Cadmium-113	8.1×10^{-11}	2.0×10^{-11}
Cadmium-113m	9.3×10^{-11}	2.5×10^{-11}

For more information, see the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients and the accompanying Table 1.

Chemical Toxicity Values

Cancer Risk	Non-Cancer Effect	
Inhalation UR	*Oral RfD: Water*	*Oral RfD: Food*
1.8 per mg/m^3	0.0005 mg/kg-d	0.001 mg/kg-d

Californium

Symbol: **Cf**

Atomic Number: **98**
(protons in nucleus)

Atomic Weight: **-**
(not naturally occurring)

What Is It? Californium is a silvery-white or gray metal with a density somewhat greater than that of lead. Californium does not occur naturally but is produced artificially in nuclear reactors and particle accelerators. Ten isotopes of californium are known to exist and all are radioactive. (Isotopes are different forms of an element that have the same number of protons in the nucleus but a different number of neutrons.) Californium was the sixth transuranic element to be discovered and was first produced in 1950 in a cyclotron at the University of California at Berkeley by bombarding curium-242 with helium ions. Californium was named for the state and the University of California.

Of the ten known isotopes, only five have half-lives long enough to warrant concern: californium-248, californium-249, californium-250, californium-251, and californium-252. The half-lives of these isotopes range from 0.91 to 900 years, while those of the other isotopes are less than two months. All five of these isotopes decay by emitting an alpha particle, and all but californium-248 also decay by spontaneous fission (SF), a process in which the atom self-disintegrates into two smaller atoms accompanied by a burst of neutrons and a release of energy. About 3% of the radioactive decays of californium-252 are by SF, while only a very small fraction of the decays of the other three isotopes are by SF. Californium is not a major radionuclide at Department of Energy legacy sites such as Hanford because it was not produced in large quantities.

Radioactive Properties of Key Californium Isotopes

Isotope	Half-Life *(yr)*	Specific Activity *(Ci/g)*	Decay Mode	Radiation Energy *(MeV)*		
				Alpha *(α)*	Beta *(β)*	Gamma *(γ)*
Cf-248	0.91	1,600	*α*	6.3	0.0060	0.0013
Cf-249	350	4.1	*α*	5.8	0.044	0.33
Cf-250	13	110	*α*	6.0	0.0057	0.0012
Cf-251	900	1.6	*α*	5.8	0.20	0.13
Cf-252	2.6	540	*α*	5.9	0.0056	0.0012

Ci = curie, g = gram, and MeV = million electron volts. (See the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients for an explanation of terms and interpretation of radiation energies.) About 3% of the decays of californium-252 are by spontaneous fission (SF), with alpha-particle decay occurring 97% of the time. A very small fraction (<0.1%) of the decays of californium-248, californium-249, and californium-250 are also by SF. Values are given to two significant figures.

Where Does It Come From? Californium is a byproduct of plutonium production and can be formed by various neutron capture and radioactive decay routes. Californium-249 results from the beta decay of berkelium-249, while the heavier (higher-numbered) isotopes are produced by intense neutron irradiation, typically in a nuclear reactor. Californium can also be produced in particle accelerators. Although it has never been detected in nature, very minute amounts of californium might exist in some uranium ores and it has been suggested (but not confirmed) that it could be produced in stellar explosions (supernova).

How Is It Used? The only californium isotope that has a commercial use is californium-252. Because this radionuclide is only available in very small quantities its uses are quite limited. Californium-252 is a very strong neutron emitter, with one microgram emitting 170 million neutrons per minute. Thin foils containing californium-252 can be used as a source of fission fragments for research purposes. Californium-252 can also be used as a portable neutron source to identify gold or silver ores through neutron activation analysis, and it can be used in moisture gauges to locate water and oil-bearing layers in oil wells. In addition, californium-252 is used in brachytherapy to treat various types of cancer.

What's in the Environment? Atmospheric testing of nuclear weapons, which ceased worldwide by 1980, would have generated a small amount of environmental californium but fallout levels are extremely low. Californium is typically quite insoluble and adheres well to soil. The concentration in soil particles is estimated to be about 500 times higher than in interstitial water.

What Happens to It in the Body? Californium can be taken into the body by eating food, drinking water, or breathing air. Gastrointestinal absorption from food or water is a likely source of internally deposited californium in the general population. After ingestion or inhalation, most californium is excreted from the body within a few days and never enters the bloodstream; only about 0.05% of the amount taken into the body by ingestion is absorbed into the blood. After leaving the intestine or lung, about 65% of the californium that does enter the bloodstream deposits in the skeleton, 25% deposits in the liver, and the rest deposits in other organs or is excreted, primarily in urine. The biological half-lives in the skeleton and liver are about 50 and 20 years, respectively. (This information is per simplified models that do not reflect intermediate redistribution.) Californium in the skeleton is deposited on bone surfaces and slowly redistributes throughout the bone volume over time.

What Is the Primary Health Effect? Californium is generally a health hazard only if it is taken into the body, although there is an external risk associated with the gamma rays emitted by californium-249 and californium-251. The main means of exposure are ingestion of food and water containing californium isotopes and inhalation of californium-contaminated dust. Ingestion is generally the exposure of concern unless there is a nearby source of contaminated airborne dust. Because californium is taken up in the body much more readily if inhaled rather than ingested, both exposure routes can be important. The major health concern is cancer resulting from the ionizing radiation emitted by californium isotopes deposited on bone surfaces and in the liver.

What Is the Risk? Lifetime cancer mortality risk coefficients have been calculated for nearly all radionuclides, including californium *(see box at right)*. While ingestion is generally the most common route of exposure, the risk coefficients for this route are much lower than those for inhalation. Similar to other radionuclides, the risk coefficients for tap water are about 80% of those for dietary ingestion. In addition to risks from internal exposures, there is an external gamma risk associated with exposure to californium-249 and californium-251. To estimate a lifetime cancer mortality risk, if it is assumed that 100,000 people were continuously exposed to a thick layer of soil with an initial average concentration of 1 pCi/g, then 6 of these 100,000 people would be predicted to incur a fatal cancer if the soil contained californium-249, and 2 if it contained californium-251. (This is in comparison to the 20,000 people from the group predicted to die of cancer from all other causes per the U.S. average.)

Radiological Risk Coefficients

This table provides selected risk coefficients for inhalation and ingestion. Maximum values are given for inhalation as no default absorption types were provided, and dietary values were used for ingestion. Risks are for lifetime cancer mortality per unit intake (pCi), averaged over all ages and both genders (10^{-9} is a billionth, and 10^{-12} is a trillionth). Other values, including for morbidity, are also available. Because risk values are not available for californium-252, these were estimated by multiplying the risk factors for californium-250 by the ratio of the dose conversion factors for californium-252 to californium-250.

Isotope	Lifetime Cancer Mortality Risk	
	Inhalation (pCi^{-1})	Ingestion (pCi^{-1})
Californium-248	2.4×10^{-8}	3.8×10^{-11}
Californium-249	4.0×10^{-8}	1.2×10^{-10}
Californium-250	3.5×10^{-8}	8.0×10^{-11}
Californium-251	4.1×10^{-8}	1.3×10^{-10}
Californium-252	2.1×10^{-8}	4.1×10^{-11}

For more information, see the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients and the accompanying Table 1.

Californium-252, with about 3% of the decays by spontaneous fission, is a significant source of neutrons and gamma rays. As noted in Federal Guidance Report 12, no detailed information has been compiled on the radiation field (both neutron and gamma rays) associated with distributed sources of radionuclides that decay by spontaneous fission, such as californium-252 in soil. This is because radionuclides with a significant spontaneous fission yield are not typically present in the environment, or if present they are at extremely low concentrations. Hence, no standard values are currently available to estimate the dose or risk from external exposure to californium-252 that accounts for the spontaneous fission dose component.

Carbon-14

Symbol:	**C(-14)**
Atomic Number: *(protons in nucleus)*	**6**
Atomic Weight: *(naturally occurring)*	**12**

What Is It? Carbon-14 is a naturally occurring radioactive isotope of carbon. (An isotope is a different form of an element that has the same number of protons in the nucleus but a different number of neutrons.) Carbon is widely distributed in nature and is present in all organic compounds. Natural forms include diamonds and graphite, which are among the hardest and softest minerals known, respectively. The nucleus of a carbon-14 atom contains six protons and eight neutrons. There are two stable (nonradioactive) isotopes of carbon: carbon-12, which has six protons and six neutrons, and carbon-13, which has six protons and seven neutrons. Carbon-12 comprises most (about 99%) of naturally occurring carbon, and carbon-13 accounts for about 1.1%. Naturally occurring carbon contains an extremely small fraction (about two trillionths) of radioactive carbon-14.

There are several radioactive isotopes of carbon in addition to carbon-14. These isotopes are very short-lived – with half-lives ranging from 20 minutes for carbon-11 to less than a second – so they are not a health concern for Department of Energy (DOE) environmental management sites. The half-life of carbon-14 is about 5,700 years, and it decays by emitting a beta particle with no attendant gamma radiation to produce nitrogen-14. Carbon-14 is an important radionuclide in the low-level radioactive wastes previously disposed of at Hanford.

Radioactive Properties of Carbon-14

Isotope	Half-Life *(yr)*	Natural Abundance *(%)*	Specific Activity *(Ci/g)*	Decay Mode	Radiation Energy *(MeV)*		
					Alpha *(α)*	Beta *(β)*	Gamma *(γ)*
C-14	5,700	<<1	4.5	β	-	0.049	-

Ci = curie, g = gram, and MeV = million electron volts; a dash means the entry is not applicable. (See companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients for explanation of terms and interpretation of radiation energies.) Values are given to two significant figures.

Where Does It Come From? Carbon-14 is produced naturally in the upper atmosphere by the reaction of neutrons originating from cosmic rays with nitrogen and, to a lesser extent, with oxygen and carbon. The natural steady-state inventory of carbon-14 in the biosphere is about 300 million Ci, most of which is in the oceans. Large amounts of carbon-14 have also been released to the atmosphere as a result of nuclear weapons testing. Weapons testing through 1963 added about 9.6 million Ci, an increase of 3% above natural steady-state levels. Carbon-14 is also made commercially for use in medical or biological tracer research. Carbon-14 is produced in nuclear reactors by the capture of neutrons by nitrogen, carbon, or oxygen present as components of the fuel, moderator, or structural hardware. The contribution to the carbon-14 global inventory from commercial nuclear reactors and DOE facilities in the United States has been less than 600 Ci per year, or less than 1/500,000th of the natural steady-state level. Carbon-14 was produced at Hanford by neutron activation of carbon in graphite-moderated plutonium-production reactors in the 100 Area. Carbon-14 is present in the graphite moderator of these shutdown reactors and in certain wastes associated with previous reactor operations, as well as in wastes from ongoing decommissioning activities, including for spent graphite.

How Is It Used? Two main uses of carbon-14 are in diagnostic medical procedures and radiocarbon dating to determine the age of previously living animals and plants. In medicine, carbon-14 can be injected to study abnormalities of metabolism that underlie diabetes, gout, anemia, and acromegaly (adult "gigantism"), and to trace the metabolism of new drugs. However, its main use to date has been to determine the age of fossils and other dead organic material. All living organisms absorb carbon from the environment, which contains carbon-12 and carbon-14 in a fixed ratio. When an organism dies, it no longer takes in carbon through respiration so the amount of carbon-14 will decrease at a constant rate due to radioactive decay, resulting in a lower ratio of carbon-14 to carbon-12 over time. Because this is constant in all living organisms, one can determine when an organism died by measuring the ratio of these

two isotopes. Radiocarbon dating is considered one of the most reliable means of determining the age of artifacts containing plant or animal matter, including some prehistoric materials up to 50,000 years old.

What's in the Environment? Carbon-14 is present in the atmosphere, oceans, and all organic material, and it behaves in the environment in the same manner as other carbon isotopes. The largest source is in the upper atmosphere where nitrogen interacts with neutrons from cosmic rays, with about 38,000 Ci of carbon-14 being produced by this process each year. Carbon-14 occurs in the ratio of 6 picocuries (pCi) of carbon-14 per gram of total carbon, and it is assimilated into tissues of all plants and animals just like other carbon isotopes. The atmospheric inventory is estimated at 13 million Ci, and it is generally present as carbon dioxide with less than 1% in the form of carbon monoxide, methane, formaldehyde, and other molecules. Carbon-14 distributes throughout the atmosphere and surface ocean waters over a period of several years. Transfer to deep ocean waters proceeds much more slowly, taking hundreds to thousands of years. The carbon-14 concentration in the troposphere has been reported to be 3.4 pCi per kilogram of air, and its concentration in soil is about 0.2 pCi/g. Carbon-14 can be present at sites with graphite-moderated reactors. It is not a major contaminant in site groundwater due to its low leachability from graphite waste and limited presence in soil. Concentrations in sandy soil are estimated to be 5 times higher than in the interstitial water (in the pore spaces between the soil particles). Thus, carbon-14 that does leach from solids to soil can move downward fairly quickly with percolating water to groundwater.

What Happens to It in the Body? Carbon-14 can be taken into the body by drinking water, eating food, or breathing air. Carbon-14 is present in the human body at a level of about 0.1 microcurie (or 100,000 pCi) in adults, and it behaves in the same manner as other carbon isotopes. Most carbon-14 is almost completely absorbed upon ingestion, moving quickly from the gastrointestinal tract to the bloodstream. However, some carbon-containing compounds in food, such as cholesterol, fat-soluble vitamins, cellulose, and polysaccharides, may be less completely absorbed. The fractional uptake of carbon-14 by inhalation is strongly dependent on its chemical form. For carbon dioxide gas and organic compounds, essentially all inhaled carbon-14 is absorbed into the bloodstream, while for carbon monoxide gas the absorption fraction is about 40%. The absorption fraction for carbon-14 on inorganic particulate aerosols is significantly lower. The carbon-14 that enters the bloodstream after either ingestion or inhalation is quickly distributed to all organs and tissues of the body, as for other isotopes of carbon. Carbon-14 is eliminated from the body with a biological half-life of 40 days.

What Is the Primary Health Effect? Carbon-14 poses a health hazard only if it is taken into the body, because it decays by emitting a weak beta particle with no gamma radiation. The beta particle emitted by carbon-14 has low energy and cannot penetrate deeply into tissue or travel far in air. Carbon-14 behaves the same as ordinary carbon, both in the environment and in the human body. Hence, a significant fraction of the carbon-14 taken in by either ingestion or inhalation is absorbed into the bloodstream, where it is transferred to all organs of the body. The health hazard of carbon-14 is associated with cell damage caused by the ionizing radiation that results from radioactive decay, with the potential for subsequent cancer induction.

What Is the Risk? Lifetime cancer mortality risk coefficients have been calculated for nearly all radionuclides, including carbon-14 *(see box at right)*. Additional values are also available, including for inhalation of carbon-14 as a gaseous oxide, i.e., as carbon monoxide and carbon dioxide. As for other radionuclides, the risk coefficient for tap water is about 80% of that for dietary ingestion.

Radiological Risk Coefficients

This table provides selected risk coefficients for inhalation and ingestion. The recommended default absorption type was used for inhalation as an organic particulate, and the dietary value was used for ingestion. Risks are for lifetime cancer mortality per unit intake (pCi), averaged over all ages and both genders (10^{-12} is a trillionth). Other values, including for morbidity, are also available.

Isotope	Lifetime Cancer Mortality Risk	
	Inhalation (pCi^{-1})	Ingestion (pCi^{-1})
Carbon-14	6.5×10^{-12}	1.4×10^{-12}

For more information, see the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients and the accompanying Table 1.

Cesium

What Is It? Cesium is a soft, silvery white-gray metal that occurs in nature as cesium-133. The natural source yielding the greatest quantity of cesium is the rare mineral pollucite. American ores of pollucite, found in Maine and South Dakota, contain about 13% cesium oxide. Although it is a metal, cesium melts at the relatively low temperature of 28° C (82° F), so like mercury it is liquid at moderate temperatures. This most alkaline of metals reacts explosively when it comes in contact with cold water.

Symbol:	**Cs**
Atomic Number: *(protons in nucleus)*	**55**
Atomic Weight: *(naturally occurring)*	**133**

There are 11 major radioactive isotopes of cesium. (Isotopes are different forms of an element that have the same number of protons in the nucleus but a different number of neutrons.) Only three have half-lives long enough to warrant concern: cesium-134, cesium-135 and cesium-137. Each of these decays by emitting a beta particle, and their half-lives range from about 2 to 2 million years. The half-lives of the other cesium isotopes are less than two weeks. Of these three, the isotope of most concern for Department of Energy (DOE) environmental management sites and other areas is cesium-137 which has a half-life of 30 years. Its decay product, barium-137m (the "m" means metastable) stabilizes itself by emitting an energetic gamma ray with a half-life of about 2.6 minutes. It is this decay product that makes cesium an external hazard (that is, a hazard without being taken into the body). Cesium-135 and cesium-134 are typically of less concern because of their radiological decay characteristics. The very long half-life of cesium-135 means it has a very low specific activity, and the slow decay rate combined with its low decay energy contribute to its low hazard. Cesium-134 has a half-life of 2.1 years and decays by emitting a beta particle. The relatively small amount of cesium-134 produced more than 20 years ago would essentially all be gone today due to radioactive decay.

Radioactive Properties of Key Cesium Isotopes and an Associated Radionuclide

Isotope	Half-Life	Specific Activity *(Ci/g)*	Decay Mode	Radiation Energy *(MeV)*		
				Alpha *(α)*	Beta *(β)*	Gamma *(γ)*
Cs-134	2.1 yr	1,300	β	-	0.16	1.6
Cs-135	2.3 million yr	0.0012	β	-	0.067	-
Cs-137	30 yr	88	β	-	0.19	-
Ba-137m (95%)	*2.6 min*	*540 million*	*IT*	-	*0.065*	*0.60*

IT = isomeric transition, Ci = curie, g = gram, and MeV = million electron volts; a dash indicates that the entry is not applicable. (See the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients for an explanation of terms and interpretation of radiation energies.) Certain properties of barium-137m are included here because this radionuclide accompanies the cesium decays. Values are given to two significant figures.

Where Does It Come From? Cesium is naturally present as the isotope 133 in various ores and to a lesser extent in soil. The three radioactive cesium isotopes identified above are produced by nuclear fission. When an atom of uranium-235 (or other fissile nuclide) fissions, it generally splits asymmetrically into two large fragments – fission products with mass numbers in the range of about 90 and 140 – and two or three neutrons. (The mass number is the sum of the number of protons and neutrons in the nucleus of the atom.) Cesium radionuclides are such fission products, with cesium-135 and cesium-137 being produced with relatively high yields of about 7% and 6%, respectively. That is, about 7 atoms of cesium-135 and 6 atoms of cesium-137 are produced per 100 fissions. Cesium-137 is a major radionuclide in spent nuclear fuel, high-level radioactive wastes resulting from the processing of spent nuclear fuel, and radioactive wastes associated with the operation of nuclear reactors and fuel reprocessing plants.

How Is It Used? Cesium metal is used in photoelectric cells and various optical instruments, and cesium compounds are used in the production of glass and ceramics. Cesium-137 is also used in brachytherapy to treat various types of cancer. (Brachytherapy is a method of radiation treatment in which sealed sources are used to deliver a radiation dose at a distance of up to a few centimeters by surface, intracavitary, or interstitial application.)

What's in the Environment? Cesium-133 exists naturally as a stable isotope. The concentration of cesium in the earth's crust is 1.9 milligrams per kilogram (mg/kg), and the concentration in seawater is about 0.5 micrograms/kg. Cesium has been shown to biomagnify in aquatic food chains. Radioactive cesium is present in soil around the world largely as a result of fallout from past atmospheric nuclear weapons tests. The concentration of cesium-137 in surface soil from fallout ranges from about 0.1 to 1 picocurie (pCi)/g, averaging less than 0.4 pCi/g (or 0.3 billionth of a milligram per kilogram soil). Cesium is also present as a contaminant at certain locations, such as nuclear reactors and facilities that process spent nuclear fuel.

Cesium is generally one of the less mobile radioactive metals in the environment. It preferentially adheres quite well to soil, and the concentration associated with sandy soil particles is estimated to be 280 times higher than in interstitial water (water in the pore space between soil particles); concentration ratios are much higher (about 2,000 to more than 4,000) in clay and loam soils. Thus, cesium is generally not a major contaminant in groundwater at DOE sites or other locations.

What Happens to It in the Body? Cesium can be taken into the body by eating food, drinking water, or breathing air. After being taken in, cesium behaves in a manner similar to potassium and distributes uniformly throughout the body. Gastrointestinal absorption from food or water is the principal source of internally deposited cesium in the general population. Essentially all cesium that is ingested is absorbed into the bloodstream through the intestines. Cesium tends to concentrate in muscles because of their relatively large mass. Like potassium, cesium is excreted from the body fairly quickly. In an adult, 10% is excreted with a biological half-life of 2 days, and the rest leaves the body with a biological half-life of 110 days. Clearance from the body is somewhat quicker for children and adolescents. This means that if someone is exposed to radioactive cesium and the source of exposure is removed, much of the cesium will readily clear the body along the normal pathways for potassium excretion within several months.

What Are the Primary Health Effects? Cesium-137 presents an external as well as internal health hazard. The strong external gamma radiation associated with its short-lived decay product barium-137m makes external exposure a concern, and shielding is often needed to handle materials containing large concentrations of cesium. While in the body, cesium poses a health hazard from both beta and gamma radiation, and the main health concern is associated with the increased likelihood for inducing cancer.

What Is the Risk? Lifetime cancer mortality risk coefficients have been calculated for nearly all radionuclides, including cesium *(see box at right)*. While the coefficients for ingestion are somewhat lower than for inhalation, ingestion is generally the most common means of entry into the body. Similar to other radionuclides, the risk coefficients for tap water are about 80% of those for dietary ingestion.

In addition to risks from internal exposures, there is a risk from external gamma exposure. Using the external gamma risk coefficient to estimate a lifetime cancer mortality risk, if it is assumed that 100,000 people were continuously exposed to a thick layer of soil with an initial average concentration of 1 pCi/g cesium-137, then 6 of these 100,000 people would be predicted to incur a fatal cancer. (This is in comparison to about 20,000 people from the group predicted to die of cancer from all other causes per the general U.S. average.) This risk is largely associated with the gamma ray from barium-137m.

Radiological Risk Coefficients

This table provides selected risk coefficients for inhalation and ingestion. Recommended default absorption types were used for inhalation, and dietary values were used for ingestion. The cesium-137 values include the contribution from the decay product barium-137m. (See text at left for information on the risk from external exposure.) Risks are for lifetime cancer mortality per unit intake (pCi), averaged over all ages and both genders (10^{-12} is a trillionth). Other values, including for morbidity, are also available.

Isotope	Lifetime Cancer Mortality Risk	
	Inhalation (pCi^{-1})	Ingestion (pCi^{-1})
Cesium-134	1.1×10^{-11}	3.5×10^{-11}
Cesium-135	1.3×10^{-12}	4.0×10^{-12}
Cesium-137	8.1×10^{-12}	2.5×10^{-11}

For more information, see the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients and the accompanying Table 1.

Chlorine

Symbol:	**Cl**
Atomic Number: *(protons in nucleus)*	**17**
Atomic Weight: *(naturally occurring)*	**35**

What Is It? Chlorine in its pure form is a greenish-yellow gas with a disagreeable, suffocating odor. About two and a half times denser than air, chlorine occurs in nature as two stable isotopes. (Isotopes are different forms of an element that have the same number of protons in the nucleus but a different number of neutrons.) Chlorine-35 is the most prevalent of these stable forms; it comprises about 76% of natural chlorine with chlorine-37 accounting for the rest.

Of the seven radioactive chlorine isotopes, only one – chlorine-36 – has a half-life long enough to warrant concern. The half-lives of all other chlorine isotopes are less than 1 hour. Chlorine-36 decays with a half-life of 300,000 years by emitting a beta particle and electron capture; most of the decays (98%) are by beta-particle emission. Chlorine-36 is present at the Hanford Site as a contaminant in former plutonium-production reactors that are currently being decommissioned. The long half-life of chlorine-36 (with its subsequent low-specific activity) combined with the relatively low energy of its beta particle and small amount of gamma radiation limit the hazards associated with this radionuclide.

Radioactive Properties of the Key Chlorine Isotope

Isotope	Half-Life *(yr)*	Specific Activity *(Ci/g)*	Decay Mode	Radiation Energy *(MeV)*		
				Alpha *(α)*	Beta *(β)*	Gamma *(γ)*
Cl-36	300,000	0.033	β, EC	-	0.027	<

EC =electron capture, Ci = curie, g = gram, and MeV = million electron volts; a dash means the entry is not applicable, and a "<" means the radiation energy is less than 0.001 MeV. (See the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients for an explanation of terms and interpretation of radiation energies.) Chlorine-36 decays by both emitting a beta particle (98%) and electron capture (2%). Values are given to two significant figures.

Where Does It Come From? Chlorine is a very reactive element that does not occur uncombined in nature. It is usually found bound with elements such as sodium, potassium, and magnesium. The most common chlorine compound is sodium chloride (table salt), which is present in seawater, salt wells, and large salt deposits, often in association with other chlorides. Chlorine is produced commercially by the electrolysis of sodium chloride, and it can also be produced by oxidizing the hydrogen chloride in hydrochloric acid. Chlorine-36 is generated in the atmosphere by the spallation of argon-36 with cosmic ray protons, and in soil and rock by neutron activation of chlorine-35. Large amounts of chlorine-36 were produced by irradiating seawater during certain nuclear weapons tests conducted between 1952 and 1958.

The graphite used as a neutron moderating material in the plutonium-production reactors in the 100 Area of the Hanford Site was treated with chlorine gas at high temperatures to remove the impurity boron (which is a good neutron absorber). A small amount of chlorine remained in the graphite that was charged to the nuclear reactors. When a fissile nuclide such as an atom of uranium-235 fissions, it generally splits asymmetrically into two large fragments – fission products with mass numbers in the range of about 90 and 140 – and two or three neutrons. (The mass number is the sum of the number of protons and neutrons in the nucleus of the atom.) These neutrons can cause additional fissions (producing a chain reaction), escape from the reactor, or irradiate nearby materials. The chlorine-35 remaining in the graphite moderator absorbed neutrons to become chlorine-36. Thus, chlorine-36 is present in the graphite moderator of these shutdown reactors and in certain wastes associated with previous reactor operations as well as in wastes from ongoing decommissioning activities, including spent graphite.

How Is It Used? Chlorine has a number of industrial and commercial uses. Its primary use is as a disinfectant, and it has been used for this purpose against a wide range of life-threatening infections, viruses, and bacteria for more than 150 years. Chlorine is used to disinfect water for both drinking and swimming, and to kill harmful levels of bacteria such as *Salmonella* and *E. coli* during food processing in restaurants and in meat and poultry packaging plants. It is also used in the manufacture of bleaching powder, household cleansers, dyes, explosives, textiles, pharmaceuticals, synthetic rubber, paper and petroleum products,

plastics, and poisonous gases. Chlorinated hydrocarbons have been used extensively as pesticides, and their long persistence has made them troublesome environmental pollutants. The freon refrigerants are hydrocarbons that have been reacted with chlorine and fluorine, and carbon tetrachloride and trichloroethylene are two common solvents.

What's in the Environment? Chlorine is present in crustal rock at a concentration of about 170 milligrams per kilogram (mg/kg), and its concentration in seawater is about 20 g/liter. In pure water, chlorine forms elemental chlorine (Cl_2), chloride ion (Cl^-) and hypochlorous acid (HOCl). Although it does preferentially adhere to soil particles, it is one of the more mobile radionuclides in soil and can move downward with percolating water to underlying soil and groundwater. Chlorine-36 is generally not a major contaminant in groundwater at contaminated sites, mainly because of its limited presence in waste and soil. Its mobility combined with its long half-life makes chlorine-36 a radionuclide of potential concern for long-term management for wastes associated with decommissioning plutonium-production reactors (such as at the DOE Hanford site; chlorine-36 is associated with the graphite-moderated reactors).

What Happens to It in the Body? Chlorine can be taken into the body by eating food, drinking water, or breathing air; small amounts could be absorbed through the skin. Gastrointestinal absorption from food or water is the principal source of internally deposited chorine in the general population. Chorine as chloride is an essential nutrient in the human diet and is necessary for healthy nervous and digestive systems. Once taken in, chlorine-36 behaves in the body the same way as other chlorine isotopes. Chlorine is almost completely absorbed upon ingestion, moving quickly from the gastrointestinal tract into the bloodstream. The chlorine-36 that enters the bloodstream after ingestion or inhalation is quickly distributed to all organs and tissues of the body. Chlorine-36 is eliminated from the body with a biological half-life of 10 days.

What Are the Primary Health Effects? Chlorine is a health hazard only if it is taken into the body. External gamma exposure is not a concern because chlorine-36 decays by emitting a relatively low-energy beta particle with only a small amount of gamma radiation. While in the body, chlorine poses a hazard from the beta particles and gamma radiation. The main concern is the increased likelihood of cancer induction. In addition to its radiological effects, chlorine is a very toxic gas, and acute exposures to high levels can cause respiratory distress and death. Chronic ingestion of chlorine has been shown to decrease organ and body weights in animals, notably at high doses.

What Is the Risk? Lifetime cancer mortality risk coefficients have been calculated for nearly all radionuclides, including chlorine *(see box at right)*. While the coefficients for ingestion are lower than for inhalation, ingestion is generally the most common means of entry into the body. Similar to other radionuclides, the risk coefficients for tap water are about 75% of those for dietary ingestion. The EPA uses a reference dose (RfD) for estimating the non-cancer health effects from oral exposure, which is an estimate of the highest dose that can be ingested every day over a lifetime without causing an adverse effect. The RfD for chlorine, shown at right, was developed by analyzing the biological effects of test animals given relatively large amounts of chlorine and then adjusting and normalizing those results to a mg/kg-day basis for humans. A toxicity value for non-cancer effects following chronic inhalation exposure to chlorine is not defined.

Radiological Risk Coefficients

This table provides selected risk coefficients for inhalation and ingestion. Maximum values are given for inhalation (no default absorption types were provided), and dietary values were used for ingestion. Risks are for lifetime cancer mortality per unit intake (picocuries, pCi), averaged over all ages and both genders (10^{-9} is a billionth, and 10^{-12} is a trillionth). Other values, including for morbidity, are also available.

Isotope	Lifetime Cancer Mortality Risk	
	Inhalation (pCi^{-1})	Ingestion (pCi^{-1})
Chlorine-36	9.6×10^{-11}	2.9×10^{-12}

For more information, see the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients and accompanying Table 1.

Chemical Toxicity Value
Non-Cancer Effect: *Oral RfD*
0.1 mg/kg-day

Cobalt

Symbol: **Co**

Atomic Number: **27**
(protons in nucleus)

Atomic Weight: **59**
(naturally occurring)

What Is It? Cobalt is a hard, silvery-white metal that occurs in nature as cobalt-59. Cobalt is a constituent of the minerals cobaltite, smaltite, erythrite, and other ores, and it is usually found in association with nickel, silver, lead, copper, and iron. Pure cobalt metal is prepared by reducing its compounds with aluminum, carbon, or hydrogen. It is similar to iron and nickel in its physical properties. Cobalt has relatively low strength and little ductility at normal temperatures and is a component of several alloys.

Radioactive Properties of Key Cobalt Isotopes

Isotope	Half-Life	Specific Activity *(Ci/g)*	Decay Mode	Radiation Energy *(MeV)*		
				Alpha *(α)*	Beta *(β)*	Gamma *(γ)*
Co-57	270 days	8,600	EC	-	0.019	0.13
Co-60	5.3 yr	1,100	β	-	0.097	2.5

EC = electron capture, Ci = curie, g = gram, and MeV = million electron volts; a dash means the entry is not applicable. (See the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients for an explanation of terms and interpretation of radiation energies.) Values are given to two significant figures.

There are nine major radioactive cobalt isotopes. (Isotopes are different forms of an element that have the same number of protons in the nucleus but a different number of neutrons.) Of these, only cobalt-57 and cobalt-60 have half-lives long enough to warrant concern. The half-lives of all other isotopes are less than 80 days. Cobalt-57 decays with a half-life of 270 days by electron capture and cobalt-60 decays with a half-life of 5.3 years by emitting a beta particle with two energetic gamma rays; the combined energy of these two gamma rays is 2.5 MeV (one has an energy of 1.2 MeV and the other has an energy of 1.3 MeV). Cobalt-60 is the isotope of most concern at Department of Energy (DOE) environmental management sites such as Hanford, for the cobalt-57 produced more than 20 years ago has long since decayed away. The two energetic gamma rays that accompany the radioactive decay of cobalt-60 make this isotope an external hazard (that is, it can be hazardous without being taken into the body).

Where Does It Come From? Cobalt is naturally present as the isotope 59 in various ores and to a lesser extent in soil. Cobalt-60 is produced by neutron activation of components in nuclear reactors; it can also be produced in a particle accelerator. When an atom of uranium-235 (or other fissile nuclide) fissions, it generally splits asymmetrically into two large fragments – fission products with mass numbers in the range of about 90 and 140 – and two or three neutrons. (The mass number is the sum of the number of protons and neutrons in the nucleus of the atom.) These neutrons can cause additional fissions (producing a chain reaction), escape from the reactor, or irradiate nearby materials. A number of reactor components are made of various alloys of steel that contain chromium, manganese, nickel, iron and cobalt, and these elements can absorb neutrons to produce radioactive isotopes, including cobalt-60. Cobalt-60 is a radionuclide of concern in spent nuclear fuel (as a component of the fuel hardware) and in the radioactive wastes associated with nuclear reactors and fuel reprocessing plants.

How Is It Used? Cobalt is used as a component of several alloys, including carboloy and stellite that are used to make very hard cutting tools. Cobalt is also used in some stainless steels. Alnico, an alloy of aluminum, nickel, cobalt, and other metals, is used to make high-strength, permanent magnets. Cobalt is also used in electroplating to give a hard surface that is resistant to oxidation, and as a blue colorant in pottery enamels and glass. High-energy gamma rays emitted during the radioactive decay of cobalt-60 can be used to detect flaws in metal components and in brachytherapy to treat various types of cancer. (Brachytherapy is a method of radiation treatment in which sealed sources are used to deliver a radiation dose at a distance of up to a few centimeters by surface, intracavitary, or interstitial application.)

What's in the Environment? Cobalt-59 is present in soil as a stable isotope at a concentration of about 1 to 2 milligram per kilogram (mg/kg). Trace amounts of cobalt-60 are also present around the globe from radioactive fallout as a result of past atmospheric weapons tests. It may also be present as a contaminant at certain locations, such as in nuclear reactors and facilities that process spent nuclear fuel, principally in the hardware associated with the spent fuel. Transport of cobalt in the environment is strongly influenced by its chemical form. It is generally one of the less mobile radioactive metals in soil, although certain forms can move downward with percolating water into underlying layers of soil. Under certain conditions, mobility can be enhanced based on the form and environmental setting, for example, when complexes form with cyanide or ferrocyanide. In other settings, cobalt has been found to preferentially adhere to soil. For sandy soil, the concentration in soil particles is estimated to be about 60 times higher than in water between the soil particles, and cobalt binds even more tightly to loam where the estimated concentration ratio is 1,300.

What Happens to It in the Body? Cobalt can be taken into the body by eating food, drinking water, or breathing air. Gastrointestinal absorption from food or water is the principal source of internally deposited cobalt in the general population. Estimates of the gastrointestinal absorption of cobalt range from 5 to 30%, depending on the chemical form and amount ingested; 10% is a typical value for adults and 30% for children. Cobalt is an essential element found in most body tissues, with the highest concentration in the liver. Vitamin B12 is a cobalt-containing vitamin essential for red blood cell formation in humans, and the intestinal absorption of cobalt in this vitamin is high. Fifty percent of cobalt that reaches the blood is excreted right away, mainly in urine; 5% deposits in the liver, and the remaining 45% deposits evenly in other tissues of the body. Of the cobalt that deposits in the liver and other tissues, 60% leaves the body with a biological half-life of 6 days and 20% clears with a biological half-life of 60 days; the last 20% is retained much longer, with a biological half-life of 800 days. On the basis of animal studies, retention of cobalt was determined to be the same for all age groups. Inhaled cobalt oxide moves from the lung to body tissues quite readily.

What Are the Primary Health Effects? Cobalt-60 poses both an internal and external hazard, and the main health concern is associated with the increased likelihood of cancer. External exposure is a concern because of the strong external gamma radiation, and shielding is often needed to handle wastes and other materials with high concentrations of this isotope. Inside the body, cobalt presents a hazard from both beta and gamma radiation.

What Is the Risk? Lifetime cancer mortality risk coefficients have been calculated for nearly all radionuclides, including cobalt *(see box at right).* While the coefficients for ingestion are somewhat lower than for inhalation, ingestion is generally the most common means of entry into the body. Similar to other radionuclides, the risk coefficients for tap water are about 70% of those for dietary ingestion. In addition to the risk from internal exposure, there is a risk from external gamma exposure. Using the external gamma risk coefficient to estimate a lifetime cancer mortality risk, if it is assumed that 100,000 people were continuously exposed to a thick layer of soil with an initial average concentration of 1 pCi/g cobalt-60, then 6 of these 100,000 people would be predicted to incur a fatal cancer. (This is compared to the 20,000 people from the group predicted to die of cancer from all other causes per the U.S. average.) The external risk for cobalt-57 is less than 1% of this risk.

Radiological Risk Coefficients

This table provides selected risk coefficients for inhalation and ingestion. Recommended default absorption types were used for inhalation, and dietary values were used for ingestion. (See text at left for information on the external gamma exposure.pathway) Risks are for lifetime cancer mortality per unit intake (pCi), averaged over all ages and both genders (10^{-9} is a billionth, and 10^{-12} is a trillionth). Other values, including for morbidity, are also available.

Isotope	Lifetime Cancer Mortality Risk	
	Inhalation (pCi^{-1})	Ingestion (pCi^{-1})
Cobalt-57	1.7×10^{-12}	9.0×10^{-13}
Cobalt-60	3.0×10^{-11}	1.4×10^{-11}

For more information, see the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients and the accompanying Table 1.

Curium

Symbol: **Cm**

Atomic Number: **96**
(protons in nucleus)

Atomic Weight: **-**
(not naturally occurring)

What Is It? Curium is a hard, brittle, silvery metal that tarnishes slowly in dry air at room temperature. Curium does not occur naturally; it is typically produced artificially in nuclear reactors through successive neutron captures by plutonium and americium isotopes. Sixteen isotopes of curium are known to exist, and all are radioactive. (Isotopes are different forms of an element that have the same number of protons in the nucleus but a different number of neutrons.) Curium was first produced in 1944 by bombarding plutonium-239 with alpha particles in a cyclotron at the University of California at Berkeley. Curium was isolated in visible amounts as the hydroxide in 1947 and is named in honor of Pierre and Marie Curie, who pioneered the study of radioactivity.

Eight of the sixteen curium isotopes have half-lives greater than one month. Curium-243 and curium-244 are the two isotopes of most concern at Department of Energy (DOE) environmental management sites such as Hanford. The curium-242 produced more than 20 years ago has essentially all decayed away, and the low specific activities of the other curium isotopes limit their radiological hazards. In addition, the longer-lived isotopes typically represent much less than 1% of the curium inventory at a site. Curium generally decays to plutonium by emitting an alpha particle; gamma radiation is associated with some of these decays. A relatively small percentage (14%) of curium-250 decays are by beta-particle emission to berkelium-250. Curium-248 and curium-250 also decay by spontaneous fission (SF), a process in which the atom self-disintegrates into two smaller atoms accompanied by a release of energy. (A very small fraction of curium-242, curium-244, and curium-246 decays are also by SF.)

Radioactive Properties of Key Curium Isotopes and Associated Radionuclides

Isotope	Half-Life	Specific Activity *(Ci/g)*	Decay Mode	Radiation Energy *(MeV)*		
				Alpha *(α)*	Beta *(β)*	Gamma *(γ)*
Cm-242	160 days	3,400	α	6.1	0.010	0.0018
Cm-243	29 yr	52	α	5.8	0.14	0.13
Cm-244	18 yr	82	α	5.8	0.086	0.0017
Cm-245	8,500 yr	0.17	α	5.4	0.065	0.096
Cm-246	4,700 yr	0.31	α	5.4	0.0080	0.0015
Cm-247	16 million yr	0.000094	α	4.9	0.021	0.32
Pu-243	*5.0 hr*	*2.6 million*	*β*	-	*0.17*	*0.026*
Cm-248	340,000 yr	0.0043	α	4.7	0.0060	0.0012
Cm-250	6,900 yr	0.21	α, β	1.3	0.0016	-
Pu-246 (25%)	*11 days*	*49,000*	*β*	-	*0.13*	*0.14*
Bk-250 (14%)	*3.2 hr*	*3.9 million*	*β*	-	*0.29*	*0.89*
Am-246 (25%)	*39 min*	*20 million*	*β*	-	*0.66*	*0.70*

Ci = curie, g = gram, and MeV = million electron volts; a dash indicates the entry is not applicable. (See the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients for an explanation of terms and interpretation of radiation energies.) About 8% of the decays of curium-248 are by spontaneous fission (SF), with alpha-particle decay occurring 92% of the time. 61% of the curium-250 decays are by SF, with alpha-particle emission (25%) and beta-particle emission (14%) accounting for the remainder. Certain properties of plutonium-243, plutonium-246, berkelium-250, and americium-246 are included here because these radionuclides accompany the curium. Values are given to two significant figures.

Where Does It Come From? Although the presence of natural curium has never been detected, minute amounts may exist in some uranium ores. Curium is a byproduct of plutonium production activities and results from the successive capture of neutrons by plutonium and americium, generally in nuclear reactors.

How Is It Used? Curium has few uses outside of research activities, and it is only available in extremely small quantities. Curium isotopes can be used without heavy shielding as sources of thermoelectric power in satellites and crewless space probes. Curium-242 has been used in isotopic power generators because it produces about 3 watts of heat energy (from radioactive decay) per gram. Curium-242 was used on lunar missions to bombard the moon's soil with alpha particles to determine what it was made of. Instruments analyzed the characteristics of the scattered alpha particles from the moon's surface, from which it was determined that lunar soil was

similar in composition to basalt, a common terrestrial volcanic rock. The high specific activity of curium-242 coupled with its low external hazard made this isotope an ideal choice for such an application.

What's in the Environment? Atmospheric testing of nuclear weapons, which ceased worldwide by 1980, generated most environmental curium. Accidents and other releases from weapons production facilities have caused localized contamination. Curium oxide is the most common form in the environment. Curium is typically quite insoluble and adheres very tightly to soil particles. The concentration of curium in sandy soil particles is estimated to be about 4,000 times higher than in interstitial water (in pore spaces between soil particles), and it binds even more tightly to loam soil where concentration ratios are even higher (18,000).

What Happens to It in the Body? Curium can be taken into the body by eating food, drinking water, or breathing air. Gastrointestinal absorption from food or water is the most likely source of any internally deposited curium in the general population. After ingestion, most curium is excreted from the body within a few days and never enters the bloodstream; only about 0.05% of the amount ingested is absorbed into the bloodstream. Of the curium that reaches the blood, about 45% deposits in the liver where it is retained with a biological half-life of 20 years, and 45% deposits in bone where it is retained with a biological half-life of 50 years (per simplified models that do not reflect intermediate redistribution). Most of the remaining 10% is directly excreted. Curium in the skeleton is deposited mainly on the endosteal surfaces of mineral bone and only slowly redistributes throughout the bone volume.

What Are the Primary Health Effects? Curium is generally a health hazard only if it is taken into the body; however, a small external risk is associated with curium-243, curium-245, curium-247, and curium-250 (note that the SF contribution from curium-250 is not quantified). People can be exposed by ingesting contaminated food or water or by inhaling contaminated dust. Ingestion is generally the exposure route of concern unless a nearby source of dust contamination exists. Because curium is absorbed within the body much more readily if inhaled rather than ingested, both exposure routes can be important. The main health concern is bone tumors resulting from ionizing radiation emitted by curium isotopes deposited on bone surfaces.

What Is the Risk? Lifetime cancer mortality risk coefficients have been calculated for nearly all radionuclides, including curium *(see box at right)*. Although ingestion is generally the most common exposure, these risk coefficients are much lower than those for inhalation. As for other nuclides, the risk coefficient for tap water is about 80% of that shown for dietary ingestion. In addition to risks from internal exposures, a risk from external gamma exposure is associated with curium-243, curium-245, curium-247, and curium-250. Using the external gamma risk coefficients to estimate lifetime cancer mortality risks, if it is assumed that 100,000 people were continuously exposed to a thick layer of soil with an initial average concentration of 1 pCi/g, then 1 person would be predicted to incur a fatal cancer if the soil contained curium-243, 1 if it contained curium-245, 6 if it contained curium-247, and 7 if it contained curium-250 (from its short-lived decay products). (This compares to 20,000 people from the group predicted to die of cancer from all other causes per the U.S. average.) The external risk coefficients for the other curium isotopes are less than 1% of those for these four. About 8% of curium-248 decays and 61% of curium-250 decays are by SF. No detailed information has been compiled on the SF radiation field (neutron and gamma rays) associated with distributed sources of radionuclides in soil. This is because radionuclides with a significant SF yield are not usually found in the

Radiological Risk Coefficients

This table provides selected risk coefficients for inhalation and ingestion. Recommended default absorption types were used for inhalation, and dietary values were used for ingestion. Risks are for lifetime cancer mortality per unit intake (picocurie, pCi), averaged over all ages and both genders (10^{-9} is a billionth, and 10^{-12} is a trillionth). Other values, including for morbidity, are also available. Because risk values are not available for curium-248 and curium-250, these were estimated by multiplying the risk factors for curium-246 by the ratios of the dose conversion factors for curium-248 and curium-250 to curium-246.

Isotope	Lifetime Cancer Mortality Risk	
	Inhalation (pCi^{-1})	Ingestion (pCi^{-1})
Curium-242	1.4×10^{-8}	3.2×10^{-11}
Curium-243	2.4×10^{-8}	8.5×10^{-11}
Curium-244	2.3×10^{-8}	7.5×10^{-11}
Curium-245	2.4×10^{-8}	9.5×10^{-11}
Curium-246	2.4×10^{-8}	9.3×10^{-11}
Curium-247	2.2×10^{-8}	9.1×10^{-11}
Curium-248	*8.8×10^{-8}*	*3.4×10^{-10}*
Curium-250	*5.0×10^{-7}*	*2.0×10^{-9}*

For more information, see the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients and the accompanying Table 1.

environment, and if present they are at extremely low levels. Thus, no standard values are available to estimate the dose or risk from external exposure to curium-248 or curium-250 that accounts for the SF dose component.

Europium

Symbol: **Eu**

Atomic Number: **63**
(protons in nucleus)

Atomic Weight: **152**
(naturally occurring)

What Is It? Europium is a silvery-white metal. It is the softest, least dense, and most volatile member of the lanthanide series, and it ignites in air at high temperatures (150 to 180°C). In nature, europium occurs as two stable isotopes. (Isotopes are different forms of an element that have the same number of protons in the nucleus but a different number of neutrons.) Europium-153 accounts for 52% of natural europium, and europium-151 makes up the remaining 48%.

Of the fourteen major radioactive isotopes, only four have half-lives long enough to warrant potential concern. The half-lives of all other europium isotopes are less than four months. Of these four longer-lived isotopes, three (europium-152, europium-154, and europium-155) are produced by the fissioning of uranium and plutonium and are present at Department of Energy (DOE) environmental management sites such as Hanford. These three isotopes have half-lives ranging from 5 to 13 years, and they decay by emitting a beta particle. A fraction (28%) of the europium-152 decays is by electron capture. A significant amount of energy in the form of gamma rays accompanies the decays of europium-152 and europium-154.

Radioactive Properties of Key Europium Isotopes

Isotope	Half-Life *(yr)*	Specific Activity *(Ci/g)*	Decay Mode	Radiation Energy *(MeV)*		
				Alpha *(α)*	Beta *(β)*	Gamma *(γ)*
Eu-150	34	70	EC	-	0.044	1.5
Eu-152	13	180	β, EC	-	0.14	1.2
Eu-154	8.8	270	β	-	0.29	1.2
Eu-155	5.0	470	β	-	0.063	0.061

EC = electron capture, Ci = curie, g = gram, and MeV = million electron volts; a dash means the entry is not applicable. (See the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients for an explanation of terms and interpretation of radiation energies.) Europium-152 decays both by emitting a beta particle (28%) and electron capture (72%). A second isotope of europium-150 having a half-life of 13 hours also exists. Values are given to two significant figures.

Where Does It Come From? Europium is found in a variety of ores, primarily bastnasite, monazite, and xenotime. These ores contain different mixes of rare earth metals, which are the elements from lanthanum through lutetium in the periodic table. Europium generally makes up less than 0.2% of this mixture. China currently produces the vast majority of the rare earth metals, totaling about 70,000 metric tons (MT) annually. The United States comes in a distant second, producing around 5,000 MT annually, and europium makes up only a small fraction of this amount.

While europium-152, europium-154, and europium-155 are produced primarily as fission products, europium-152 can also be produced by neutron activation of nuclear reactor control rods. When a fissile nuclide such as an atom of uranium-235 fissions, it generally splits asymmetrically into two large fragments – which can include the three europium isotopes – and two or three neutrons. The fission yield of europium-155 is about 0.03% while the yield of the other two isotopes is much lower. That is, about 3 atoms of europium-155 are produced per 10,000 fissions. In order to control this fission reaction, isotopes that can absorb excess neutrons are used in nuclear reactor control rods. Because europium-151 is a very good neutron absorber, it is often used in these control rods. Neutron activation of this stable isotope produces europium-152.

How Is It Used? The primary use of europium is in nuclear reactor control rods, because of its effectiveness in absorbing neutrons. Other uses have been limited because it is rare and thus very expensive. Europium-doped plastics have been used as laser materials, and europium oxide serves as a phosphor activator. For example, europium has been used to activate yttrium vanadate for its use in the red phosphors of color television tubes.

What's in the Environment? Europium is present in the earth's crust at a concentration of about 1.8 milligrams per kilogram (mg/kg), while its concentration in seawater is about 0.00013 micrograms per liter. Trace amounts of europium-152, europium-154, and europium-155 are present in soil around the globe from radioactive fallout. It can also be present at certain nuclear facilities, such as reactors and spent fuel reprocessing plants. Europium is generally one of the more immobile radioactive metals in the environment. It preferentially adheres fairly tightly to soil, and the concentration associated with soil particles is estimated to be about 240 times higher than in interstitial water (the water in the pore space between soil particles). Thus, europium is generally not a major contaminant in groundwater at DOE sites.

What Happens to It in the Body? Europium can be taken into the body by eating food, drinking water, or breathing air. Gastrointestinal absorption from food or water is the principal source of internally deposited europium in the general population. Europium is not well absorbed into the body after intake, with only about 0.05% of the amount ingested being absorbed into the bloodstream through the digestive tract. Of the europium that reaches the blood, 40% is deposited in the liver, and another 40% is deposited on the surface of the bone, where it can irradiate the bone-forming cells; this deposited europium is retained in the body with a biological half-life of almost 10 years (3,500 days); an additional 6% of the absorbed europium is deposited in the kidneys, where it is retained with a short biological half-life of 10 days (per simplified models that do not reflect intermediate redistribution). The remainder of the absorbed europium is excreted.

What Are the Primary Health Effects? Europium poses an external as well as an internal health hazard. The strong gamma radiation associated with europium-152 and europium-154 makes external exposure to these two isotopes a concern. While in the body, europium poses a health hazard from both the beta particles and gamma rays, and the main health concern is associated with the increased likelihood of inducing cancer in the liver and bone.

What Is the Risk? Lifetime cancer mortality risk coefficients have been calculated for nearly all radionuclides, including europium *(see box at right)*. While the coefficients for ingestion are somewhat lower than for inhalation, ingestion is generally the most common means of entry into the body. Similar to other radionuclides, the risk coefficients for tap water are about 70% of those for dietary ingestion.

In addition to the risks from internal exposure, a risk from external gamma exposure is associated with europium-152 and europium-154. If it is assumed that 100,000 people were continuously exposed to a thick layer of soil with an initial average concentration of 1 picocurie (pCi)/g, then the number of fatal cancers estimated for these 100,000 people is 7 for exposure to europium-152 and 5 for exposure to europium-154. (This can be compared to the 20,000 people from this group who would be predicted to die of cancer from all other causes per the U.S. average.)

Radiological Risk Coefficients

This table provides selected risk coefficients for inhalation and ingestion. Maximum values are given for inhalation since no default absorption types were provided, and dietary values were used for ingestion. Risks are for lifetime cancer mortality per unit intake (pCi), averaged over all ages and both genders (10^{-9} is a billionth, and 10^{-12} is a trillionth). Other values, including for morbidity, are also available.

Isotope	Lifetime Cancer Mortality Risk	
	Inhalation *(per pCi^{-1})*	**Ingestion** *(per pCi^{-1})*
Europium-150	2.1×10^{-10}	3.6×10^{-12}
Europium-152	1.5×10^{-10}	5.0×10^{-12}
Europium-154	1.7×10^{-10}	8.5×10^{-12}
Europium-155	1.7×10^{-11}	1.6×10^{-12}

For more information, see the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients and the accompanying Table 1.

Iodine

What Is It? Iodine is a bluish-black, lustrous solid that mainly occurs in nature as stable iodine-127. A small amount of radioactive iodine-129 is produced naturally in the upper atmosphere by the interaction of high-energy particles with xenon. Iodine volatilizes at ambient temperatures into a pretty blue-violet gas with an irritating odor. Iodine exhibits some metal-like properties and is only slightly soluble in water. It occurs in nature as iodide ions, and it is in this form that it is taken into our bodies.

Symbol: **I**

Atomic Number: **53**
(protons in nucleus)

Atomic Weight: **127**
(naturally occurring)

Of the fourteen major radioactive isotopes of iodine, only iodine-129 has a half-life sufficiently long to warrant concern for Department of Energy (DOE) environmental management sites such as Hanford. (Isotopes are different forms of an element that have the same number of protons in the nucleus but a different number of neutrons.) Iodine-129 decays by emitting a beta particle with a half-life of about 16 million years; the half-lives of all other iodine radionuclides are less than 60 days. The very long half-life of iodine-129 (with its subsequent low-specific activity) combined with the low energy of its beta particle and minimal gamma radiation limit the hazards of this radionuclide. Iodine-131 has a short half-life (8 days) and is not generally a major isotope of concern for DOE environmental management sites. However, information is included here for this radionuclide because it was released during past operations of nuclear reactors at Hanford.

Radioactive Properties of Key Iodine Isotopes

Isotope	Half-Life	Specific Activity *(Ci/g)*	Decay Mode	Radiation Energy *(MeV)*		
				Alpha *(α)*	Beta *(β)*	Gamma *(γ)*
I-129	16 million yr	0.00018	β	-	0.064	0.025
I-131	8.0 days	130,000	β	-	0.19	0.38

EC = electron capture, Ci = curie, g = gram, and MeV = million electron volts; a dash means the entry is not applicable. (See the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients for an explanation of terms and interpretation of radiation energies.) Values are given to two significant figures.

Where Does It Come From? Stable iodine (iodine-127) is naturally present in seaweeds, sponges, and other materials. Radioactive isotopes of iodine are produced by nuclear fission. When an atom of uranium-235 (or other fissile nuclide) fissions, it generally splits asymmetrically into two large fragments – fission products with mass numbers in the range of about 90 and 140 – and two or three neutrons. (The mass number is the sum of the number of protons and neutrons in the nucleus of the atom.) Iodine-129 and iodine-131 are two such products. The fission yield of iodine-129 is about 1% and the yield of iodine-131 is close to 3%. That is, about one atom of iodine-129 and three atoms of iodine 131 are produced per 100 fissions. Iodine-129 is present in spent nuclear fuel, high-level radioactive wastes resulting from processing spent nuclear fuel, and radioactive wastes associated with the operation of nuclear reactors and fuel reprocessing plants.

How Is It Used? Iodine is used to treat cuts and scrapes on the skin as a tincture of iodine, which is a dilute mixture of alcohol and iodine. Iodine is also used in photography and lasers (silver iodide), in dyes, and as a nutrient added to table salt. Iodine-131 is used for a number of medical procedures, including to monitor and trace the flow of thyroxin from the thyroid. With its short half-life of 8 days, it is essentially gone in less than three months. Iodine-129 has no important commercial uses.

What's in the Environment? Iodine is present in nature in various materials, with soil, rock, and all living organisms containing low concentrations. Iodine is assimilated by seaweeds and sponges (from which it may be recovered) and is found in Chilean saltpeter, caliche, and brine associated with salt

deposits. The ratio of stable iodine-127 to radioactive iodine-129 in the environment is more than 10 million to 1. The human body contains 10 to 20 milligrams of iodine, of which more than 90% is contained in the thyroid gland. Iodine-129 is present in soil around the world as a result of fallout from past atmospheric nuclear weapons tests; any iodine-131 that may have been present in soil from fallout has long since decayed away. Iodine may also be found as a contaminant at facilities where spent nuclear fuel was processed.

Iodine-129 is one of the more mobile radionuclides in soil and can move downward with percolating water to groundwater. Iodine concentrations in sandy soil are about the same as in interstitial water (in the pore spaces between soil particles). It binds more preferentially to loam, where the concentration in soil is estimated to be 5 times higher than in interstitial water.

What Happens to It in the Body? Iodine can be taken into the body by eating food, drinking water, or breathing air. It is a constituent of thyroid hormone and as such is a required element for humans. Iodine is readily taken into the bloodstream from both the lungs and the gastrointestinal tract (essentially 100%) after inhalation and ingestion. Upon entering the bloodstream, 30% is deposited in the thyroid, 20% is quickly excreted in feces, and the remainder is eliminated from the body within a short time (per simplified models that do not reflect intermediate redistribution). Clearance from the thyroid is age-dependent, with biological half-lives ranging from 11 days in infants to 23 days in a five-year-old child and 80 days in adults.

What Are the Primary Health Effects? Iodine is an essential component of the human diet, and lack of dietary iodine is a cause of goiter. Elemental iodine (I_2) can be toxic, and its vapor irritates the eyes and lungs. While iodine is generally a health hazard only if it is taken into the body in substantial doses, iodine-131 emits fairly high-energy beta particles and a number of gamma rays. The gamma rays are of sufficient energy to be measured outside the body if deposited in tissue such as the thyroid. Because iodine selectively deposits in the thyroid, the primary health hazard for iodine is thyroid tumors resulting from ionizing radiation emitted by iodine-129 and iodine-131. Historically, the major exposure pathway has been ingestion of milk from cows grazing on iodine-contaminated crops. Other pathways include ingestion of fruits and vegetables and inhalation.

What Is the Risk? Lifetime cancer mortality risk coefficients have been calculated for nearly all radionuclides, including iodine *(see box at right).* Additional values are also available, including for inhalation of iodine vapor and methyl iodide. Similar to other radionuclides, the risk coefficients for tap water are about 75% of those shown for dietary ingestion.

Thyroid cancer is the main risk associated with radioactive iodine. Based on epidemiological studies for external radiation, children are more susceptible than adults to cancer from thyroid irradiation. Data available for iodine-131 have not shown it to be carcinogenic in the human thyroid. While certain studies have indicated a potential effect from exposures to iodine-131 (e.g., at Chernobyl, where external radiation was also quite high), others have not.

Radiological Risk Coefficients

This table provides selected risk coefficients for inhalation and ingestion. Recommended default absorption types were used for inhalation of particulates, and milk consumption values were used for ingestion. Risks are for lifetime cancer mortality per unit intake (pCi), averaged over all ages and both genders (10^{-9} is a billionth, and 10^{-12} is a trillionth). Other values, including for morbidity, are also available.

Isotope	Lifetime Cancer Mortality Risk	
	Inhalation (pCi^{-1})	Ingestion (pCi^{-1})
Iodine-129	6.2×10^{-12}	3.3×10^{-11}
Iodine-131	2.1×10^{-12}	1.4×10^{-11}

For more information, see the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients and the accompanying Table 1.

Iridium

What Is It? Iridium is a silvery white metal named after the Latin word for rainbow because its salts are highly colored. Iridium is hard and brittle with low ductility, which makes it very difficult to machine and form. It is quite dense, about twice as dense as lead, and occurs in nature as two stable isotopes. (Isotopes are different forms of an element that have the same number of protons in the nucleus but a different number of neutrons.) Iridium-193 is the most prevalent form, comprising about 63% of natural iridium, with iridium-191 accounting for the rest.

Symbol: **Ir**

Atomic Number: **77**
(protons in nucleus)

Atomic Weight: **192**
(naturally occurring)

Of the 15 major radioactive iridium isotopes, only three have half-lives longer than a month and warrant concern; these are shown in the box at right. The half-lives of the other isotopes are less than 2 weeks. Iridium-192 has a half-life of 74 days, and it decays to stable platinum-192 and osmium-192 by emitting a beta particle and by electron capture; most of these decays (95%) are by beta emission. Iridium-192m (the "m" means metastable) has a half-life of 240 years, and it decays to iridium-192 by isomeric transition and by emitting a relatively low-energy gamma ray. Iridium-194m has a half-life of 170 days, and it decays to stable platinum-194 by emitting a beta particle and several gamma rays. Of these three radioactive isotopes, iridium-192m presents the lowest acute or short-term hazard from external exposures as a result of its relatively low specific activity and gamma radiation; the other two iridium isotopes have high specific activities and significant gamma radiation. Iridium-192 is the isotope of most concern based on general availability; it is used in a number of industrial and medical applications.

Radioactive Properties of Key Iridium Isotopes

Isotope	Half-Life	Specific Activity *(Ci/g)*	Decay Mode	Radiation Energy *(MeV)*		
				Alpha *(α)*	Beta *(β)*	Gamma *(γ)*
Ir-192	74 days	9,200	β, EC	-	0.22	0.82
Ir-192m	240 yr	7.8	IT	-	-	0.16
Ir-194m	170 days	4,000	β	-	0.16	2.3

EC = electron capture, IT = isomeric transition, Ci = curie, g = gram, and MeV = million electron volts; a dash means the entry is not applicable. (See the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients for an explanation of terms and interpretation of radiation energies.) Iridium-192 decays by two means: emitting a beta particle (95%) and electron capture (5%). Values are given to two significant figures.

Where Does It Come From? Iridium is found uncombined in nature with platinum and other metals in the platinum group, and it can be obtained from platinum ores and as a byproduct of nickel mining. Naturally occurring iridium alloys include osmiridium and iridiosmium, which are mixtures of iridium and osmium. The highest iridium concentration on earth is at its core, and it can be released to the earth's surface in volcanoes. Iridium-192 is produced by neutron activation of iridium metal, usually in nuclear reactors. The strength of an iridium-192 source is related to the amount of neutron irradiation. Natural iridium contains 37% iridium-191, and when this isotope absorbs a neutron it produces iridium-192. Neutron absorption by iridium-193, the other naturally occurring isotope, produces iridium-194. Iridium-194 has a short half-life of 19 hours, and it decays to stable platinum-194 in a few days; very little iridium-192m and iridium-194m are produced in this process.

How Is It Used? Iridium is used as an alloying agent with a number of other metals to produce composites that are extremely hard and have good corrosion resistance. Its principal use is as a hardening agent in platinum alloys. Iridium is used for high-temperature applications, including in crucibles and thermocouples and as electrodes in spark plugs for severe operating conditions, such as those experienced by jet engine igniters. Radioactive iridum-192 is used industrially as a radiotracer in the oil industry and in gamma radiography to identify flaws in metal castings and welded joints. These radiographic sources are constructed of metal discs or pellets in a welded stainless steel capsule, and their activity levels range from less than one curie to several hundred curies.

Iridium-192 is also used medically in brachytherapy to treat various types of cancer. (Brachytherapy is a method of radiation treatment in which sealed sources are used to deliver a radiation dose at a distance of up to a few centimeters by surface, intracavitary, or interstitial application.) Iridium-192 implants are used especially in the head and breast. They are produced in wire form and are introduced through a catheter to the target area. After

being left in place for the time required to deliver the desired dose, the implant wire is removed. This procedure is very effective at providing localized radiation to the tumor site while minimizing the patient's whole-body dose.

What's in the Environment? Iridium is naturally present in the earth's crust at a concentration of about 1 microgram per kilogram (µg/kg), or part per billion. In contrast, the concentration of iridium in some meteorites is about 500 µg/kg. A thin layer of iridium exists worldwide in a layer of deep sediment that was put down at the end of the Cretaceous period (roughly 150 million years ago), which is seen as evidence that the earth was struck by a large meteor or asteroid at that time. Dust from that impact would have spread around the globe, depositing iridium. As a very corrosion-resistant metal, iridium is quite insoluble in water. This means the iridium that is present in soil and rock tends to stay in place rather than being mobilized and transported in solution toward groundwater. There is no evidence that the three radioactive isotopes are present in soil around the world. These isotopes are not fission products so they would not exist in radioactive fallout from past atmospheric weapons tests. Only one isotope (iridium-192m) has a half-life longer than a year, and that isotope is not produced in any significant quantities.

What Happens to It in the Body? Iridium can be taken into the body by eating food, drinking water, or breathing air. Gastrointestinal absorption from food or water is the likely source of internally deposited iridium in the general population. After ingestion or inhalation, most iridium is excreted from the body and never enters the bloodstream; only about 1% of the amount taken into the body by ingestion is absorbed into the blood. Twenty percent of the iridium that reaches the blood is excreted right away, 20% deposits in the liver, 4% deposits in the kidney, 2% deposits in the spleen, and the remaining 54% is evenly distributed among other organs and tissues of the body. Of the iridium that deposits in any organ or tissue, 20% leaves the body with a biological half-life of 8 days and 80% clears with a biological half-life of 200 days. On the basis of animal studies, retention of iridium was determined to be the same for all age groups. Most inhaled iridium compounds appear to clear the lungs quite rapidly.

What Are the Primary Health Effects? The three radioactive iridium isotopes pose both an internal and external hazard, and the main health concern is associated with the increased likelihood of cancer. External exposure is a concern because of the strong gamma radiation (especially for iridium-192 and iridium-194m), and shielding is needed to handle iridium-192 radiographic and medical sources. Iridium can concentrate in several organs depending on its chemical form, so while there is no dominant organ of health concern the liver is a main organ of deposition. Inside the body, these iridium isotopes can pose a hazard from both beta and gamma radiation.

What Is the Risk? Lifetime cancer mortality risk coefficients have been calculated for nearly all radionuclides, including iridium *(see box at right).* While ingestion is generally the most common means for entry into the body, the risk coefficients for that route are lower than for inhalation. Similar to other radionuclides, the risk coefficients for tap water are about 70% of those for dietary ingestion.

In addition to the risks from internal exposures, a risk from external gamma radiation is associated with these iridium isotopes. To estimate the lifetime cancer mortality risk, if it is assumed that 100,000 people were continuously exposed to a thick layer of soil with an initial average concentration of 1 pCi/g iridium-192m, then 10 of these people would be predicted to incur a fatal cancer. (This is in comparison to the 20,000 people from this group predicted to die of cancer from all other causes, per the U.S. average.) While the gamma radiation for iridium-192 and iridium-194m is much greater than for iridium-192m, these radionuclides will decay to very low levels in a few years. Because of this decay, less than one additional cancer fatality is predicted to occur among 100,000 people exposed to soil with an initial concentration of 1 pCi/g of either isotope. However, the respective external gamma radiation risks for acute exposures to iridium-192 and iridium-194m are 6 and 19 times higher than for iridium-192m.

Radiological Risk Coefficients

This table provides selected risk coefficients for inhalation and ingestion. Maximum values are given for inhalation (no default absorption types were provided), and dietary values were used for ingestion. Risks are for lifetime cancer mortality per unit intake (pCi), averaged over all ages and both genders (10^{-9} is a billionth and 10^{-12} is a trillionth). Other values, including for morbidity, are also available.

Isotope	Lifetime Cancer Mortality Risk	
	Inhalation (pCi^{-1})	Ingestion (pCi^{-1})
Iridium-192	2.1×10^{-11}	6.0×10^{-12}
Iridium-192m	1.7×10^{-11}	8.7×10^{-13}
Iridium-194m	4.0×10^{-11}	7.3×10^{-12}

For more information, see the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients and the accompanying Table 1.

Krypton

Symbol:	**Kr**
Atomic Number: *(protons in nucleus)*	**36**
Atomic Weight:	**84**

What Is It? Krypton is a colorless, odorless, tasteless gas about three times heavier than air. It was discovered in 1898 by Sir William Ramsay and Morris Travers in the residue left after evaporating water, oxygen, nitrogen, helium, and argon from a sample of liquid air. The name comes from the Greek work *kryptos*, meaning hidden. As a noble gas, krypton is generally inert and forms very few chemical compounds. It occurs in nature as six stable isotopes. (Isotopes are different forms of an element that have the same number of protons in the nucleus but a different number of neutrons.) Krypton-84 is the most prevalent, comprising about 57% of natural krypton. The other five stable isotopes and their relative abundances are krypton-78 (0.4%), krypton-80 (2.3%), krypton-82 (12%), krypton-83 (11%), and krypton-86 (17%).

Eleven major radioactive isotopes of krypton exist of which only two – krypton-81 and krypton-85 – have half-lives long enough to warrant concern. Krypton-81 has a half-life of 210,000 years, and krypton-85 has a half-life of 11 years; the half-lives of the other krypton isotopes are less than two days. Krypton-85 is the isotope of concern at Department of Energy (DOE) environmental management sites such as Hanford. It is produced by the fissioning of uranium and plutonium and is present in spent nuclear fuel. The low specific activity of krypton-81 limits its radioactive hazards.

Radioactive Properties of Key Krypton Isotopes

Isotope	Half-Life *(yr)*	Specific Activity *(Ci/g)*	Decay Mode	Radiation Energy *(MeV)*		
				Alpha *(α)*	Beta *(β)*	Gamma *(γ)*
Kr-81	210,000	0.021	EC	-	0.0051	0.012
Kr-85	11	400	β	-	0.25	0.0022

EC = electron capture, Ci = curie, g = gram, and MeV = million electron volts; a dash means the entry is not applicable. (See the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients for an explanation of terms and interpretation of radiation energies.) Values are given to two significant figures.

Where Does It Come From? Krypton is naturally present in meteorites and minerals in trace quantities. It exists naturally in the atmosphere at a concentration of about 1 cubic centimeter per cubic meter (cm^3/m^3). Radioactive krypton-85 is present in the natural environment in minute quantities due to the spontaneous and neutron-induced fission of uranium and other actinides. Krypton-81 and krypton-85 are both present in the atmosphere due to neutron capture reactions from cosmic ray neutrons interacting with stable krypton isotopes. Krypton can be obtained as a byproduct from the liquefaction and separation of air.

The major source of krypton-85 is nuclear fission. When an atom of uranium-235 (or other fissile nuclide) fissions, it generally splits asymmetrically into two large fragments – fission products with mass numbers in the range of about 90 and 140 – and two or three neutrons. (The mass number is the sum of the number of protons and neutrons in the nucleus of the atom.) Krypton-85 is one such fission product with a fission yield of about 0.3%. That is, three atoms of krypton-85 are produced per 1,000 fissions. An estimated 5 million curies of krypton-85 were released to the atmosphere as a result of nuclear weapons tests from 1945 through 1962. A large commercial nuclear power plant produces about 300,000 curies of krypton-85 per year, essentially all of which is retained within the fuel elements. This gaseous radionuclide is a component of spent nuclear fuel and is generally released to the atmosphere when the fuel is reprocessed. About 50,000 curies of krypton-85 were released to the atmosphere as a result of the accident at Three Mile Island in which a large number of fuel elements ruptured.

How Is It Used? Krypton has a number of industrial and medical applications. It is used alone or in combination with argon and neon in fluorescent lights. It emits a characteristic bright orange-red color and is used in lights at airports because the red light is visible for long distances and penetrates fog and haze to a greater extent than ordinary light. Krypton is also used in tungsten-filament projection lamps for home movies and slide projectors. A krypton gas laser produces a very intense and concentrated light,

and these lasers are used for medical applications such as surgery on the retina of the eye. The intense krypton laser light causes the blood to clot during the surgery, thus preventing further bleeding with subsequent loss of vision, and the laser is so accurate that surrounding tissues are not damaged.

Krypton is also used as a standard because the spectral lines of its isotopes are very sharp. In 1960, the International Commission on Weights and Measures defined the length of the standard meter as exactly 1,650,763.73 wavelengths (in a vacuum) of the orange-red line in the emission spectrum of krypton-86. This unit was redefined in October 1983 as the path length of light in a vacuum during a time interval of 1/299,792,458 of a second. Radioactive krypton-85 is used to detect leaks from sealed containers, with the escaping atoms being identified through their radiation. Krypton-85 is also used to excite phosphors in light sources with no external source of energy and in medicine to detect abnormal heart openings.

What's in the Environment? The highest concentrations of krypton are in the atmosphere. Krypton is present in air at a concentration of about 1 cm^3/m^3, or parts per million by volume. On a mass basis, the concentration is about 3 mg/kg. For comparison, the krypton concentration in the atmosphere of Mars is about 1/3 this amount (0.3 cm^3/m^3). Krypton is naturally present in the earth's crust at a concentration of about 0.15 micrograms per kilogram (µg/kg), and its concentration in seawater is about 0.21 µg/liter. Krypton-85 has been released to the atmosphere during nuclear fuel reprocessing activities and as a result of past aboveground nuclear weapons tests. In 1970, the concentration of krypton-85 in the atmosphere reached about 10 picocuries (pCi)/m^3 (or 10 trillionths of a curie per m^3), mainly from nuclear weapons tests and plutonium production activities. The concentration is significantly lower now due to the relatively short half-half of this radionuclide, the cessation of aboveground nuclear weapons tests worldwide by 1980, and the shutdown of plutonium production facilities at DOE sites. Neither the oceans nor the land surfaces act as significant sinks for this radionuclide. Krypton-85 is in spent nuclear fuel stored at certain sites (such as the DOE Hanford Site).

What Happens to It in the Body? As a noble gas, krypton does not generally participate in any biological processes. After being taken into the body, a very small amount can be dissolved in the bloodstream and distributed to organs and tissues throughout the body. Nevertheless, the tissue of most concern from exposure to a cloud of krypton-85 gas is generally the skin, with most of the dose resulting from the beta particles associated with its radioactive decay.

What Are the Primary Health Effects? The main health concern is the increased likelihood for cancer induction, and the exposure pathway of most concern is external exposure in a cloud of gas. The radiation dose for krypton-85 (the primary isotope of concern) from an external cloud of gas is more than 130 times higher than the dose from any gas in the lungs and more than 200 times higher than that from any gas in body organs and tissues after being taken into the body. For kypton-81, most of the dose is associated with gamma rays that will irradiate all tissues and organs of the body. In contrast, much of the dose for kypton-85 is from beta particles, and the skin is the primary tissue of concern.

What Is the Risk? Radiation doses from inhaling or ingesting krypton are small compared to the dose from external radiation, such as could occur in a cloud of krypton gas. In contrast to most other radionuclides, lifetime cancer mortality risk coefficients have not been developed for the inhalation and ingestion of krypton isotopes. The only pathway for which cancer mortality risk coefficients have been developed is external exposure. External gamma risk coefficients for krypton-81 and krypton-85 were used to estimate lifetime cancer mortality risks for submersion in krypton clouds. If it is assumed that krypton releases occurred and 100,000 people were continuously exposed to a cloud of air with an average concentration of 1 pCi/cm^3 over a period of one year, then the estimated number of fatal cancers in this group of 100,000 would be 2 for krypton-81 and less than 1 for krypton-85. (This is in comparison to the 20,000 people from this group who would be predicted to die of cancer from all other causes per the U.S. average.) This risk is due to the beta particles and gamma rays associated with the two krypton isotopes. *(For more information, see the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients and the accompanying Table 1.)*

Neptunium

Symbol: **Np**

Atomic Number: **93**
(protons in nucleus)

Atomic Weight: **-**
(not naturally occurring)

What Is It? Neptunium is a ductile, silver-colored metal about twice as dense as lead. It does not occur naturally but is produced artificially by neutron capture reactions by uranium. There are seventeen known isotopes of neptunium, and all are radioactive. (Isotopes are different forms of an element that have the same number of protons in the nucleus but a different number of neutrons.) The first neptunium isotope to be identified was neptunium-239, which has a half-life of 2.4 days. This isotope was first produced in 1940 in a cyclotron at the University of California at Berkeley by bombarding uranium-238 with high-energy neutrons. Neptunium was the first transuranic element to be formed and was named for the planet Neptune.

Of the seventeen neptunium isotopes, only three have half-lives long enough to warrant concern at Department of Energy (DOE) environmental management sites: neptunium-235, neptunium-236, and neptunium-237. The half-lives of these three isotopes range from 1.1 to 2.1 million years, while those of the other isotopes are less than five days. Of the three, neptunium-237 is the most prevalent isotope at DOE sites such as Hanford. It has a half-life of 2.1 million years and decays by emitting an alpha particle. The other two isotopes typically represent less than a few percent of the total neptunium inventory at a site. Neptunium-235 has a half-life of 1.1 years and decays by electron capture; essentially all of this isotope that was produced more than 20 years ago has long since decayed away. Neptunium-236 has a half-life of 120,000 years and decays by emitting a beta particle and electron capture.

Radioactive Properties of Key Neptunium Isotopes and Associated Radionuclides

Isotope	Half-Life *(yr)*	Specific Activity *(Ci/g)*	Decay Mode	Radiation Energy *(MeV)*		
				Alpha *(α)*	Beta *(β)*	Gamma *(γ)*
Np-235	1.1	1,400	EC	<	0.010	0.0071
Np-236	120,000	0.013	β, EC	-	0.21	0.14
Pu-236 (9%)	*2.9*	*540*	*α*	*5.8*	*0.013*	*0.0021*
Np-237	2.1 million	0.00071	α	4.8	0.070	0.035
Pa-233	*0.074*	*21,000*	*β*	-	*0.20*	*0.20*

EC = electron capture, Ci = curie, g = gram, and MeV = million electron volts; a "<" means the radiation energy is less than 0.001 MeV, and a dash means the entry is not applicable. (See the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients for explanation of terms and interpretation of radiation energies.) About 0.001% of the neptunium-235 decays are by alpha-particle emission. The isotope neptunium-236 decays both by emitting a beta particle (9%) and by electron capture (91%); another isotope of neptunium-236 with a half-life of 23 hours also exists. Certain properties of plutonium-236 and protactinium-233 are included here because these radionuclides accompany the neptunium decays. Values are given to two significant figures.

Where Does It Come From? Neptunium is a byproduct of plutonium production activities and results from the capture of neutrons by uranium isotopes, usually in a nuclear reactor. Neptunium isotopes can be formed by a variety of neutron capture and radioactive decay routes. Neptunium is present in spent nuclear fuel, high-level radioactive wastes resulting from the processing of spent nuclear fuel, and radioactive wastes associated with the operation of reactors and fuel reprocessing plants. Although neptunium is essentially not naturally present in the environment, very minute amounts may be associated with uranium ores.

How Is It Used? There are no major commercial uses of neptunium, although neptunium-237 is used as a component in neutron detection instruments. Neptunium-237 can also be used to make plutonium-238 (by absorption of a neutron). Neptunium is considered useable in nuclear weapons, although no country is known to have used it to make a nuclear explosive device.

What's in the Environment? Atmospheric testing of nuclear weapons, which ceased worldwide by 1980, generated most environmental neptunium. The level of neptunium in soil from fallout is quite low; for

example, the concentration of neptunium-237 is less than 1% of that for plutonium-239 (on the order of 0.0001 picocuries per gram, pCi/g). Accidents and other releases from weapons production facilities have caused localized contamination. Neptunium typically occurs in the environment as an oxide, although other forms can be present. It is generally more mobile than other transuranic elements such as plutonium, americium, and curium, and it can move down with percolating water to underlying layers of soil. Neptunium preferentially adheres to soil particles, with the concentration associated with sandy soil particles estimated to be about 5 times higher than in interstitial water (water in pore spaces between the soil particles); it bonds more tightly to clay soils, where concentration ratios are typically higher (55). Neptunium is readily taken up by plants, and plant concentrations are typically similar to soil concentrations.

What Happens to It in the Body? Neptunium can be taken into the body by eating food, drinking water, or breathing air. Gastrointestinal absorption from food or water is a likely source of internally deposited neptunium in the general population. After ingestion or inhalation, most neptunium is excreted from the body within a few days and never enters the bloodstream; only about 0.05% of the amount taken into the body by ingestion is absorbed into the blood. After leaving the intestine or lung, about 50% of the neptunium that does enter the bloodstream deposits in the skeleton, about 10% deposits in the liver, about 5% deposits in other soft tissues, and the rest is excreted, primarily in urine. The biological half-lives in the skeleton and liver are about 50 and 20 years, respectively. (This information is per simplified models that do not reflect intermediate redistribution.) The amount deposited in the liver and skeleton depends on the age of the individual, with fractional uptake in the liver increasing with age. Neptunium in the skeleton is deposited on bone surfaces and slowly redistributes throughout the bone volume over time.

What Is the Primary Health Effect? Neptunium is generally a health hazard only if it is taken into the body, although there is an external risk associated with the gamma rays emitted by neptunium-236 and neptunium-237 and its short-lived decay product protactinium-233. The main means of exposure are ingestion of food and water containing neptunium isotopes and inhalation of neptunium-contaminated dust. Ingestion is generally the exposure of concern unless there is a nearby source of contaminated airborne dust. Because neptunium is taken up in the body much more readily if inhaled rather than ingested, both exposure routes can be important. The major health concern is cancer resulting from the ionizing radiation emitted by neptunium isotopes deposited on bone surfaces and in the liver.

What Is the Risk? Lifetime cancer mortality risk coefficients have been calculated for nearly all radionuclides, including neptunium *(see box at right)*. While ingestion is generally the most common route of exposure, the risk coefficients for this route are much lower than those for inhalation. Similar to other radionuclides, the risk coefficients for tap water are about 70 to 75% of those for dietary ingestion.

In addition to risks from internal exposures, there is an external gamma risk associated with exposure to neptunium-236 and neptunium-237. To estimate a lifetime cancer mortality risk, if it is assumed that 100,000 people were continuously exposed to a thick layer of soil with an initial average concentration of 1 pCi/g, then 2 of these 100,000 people would be predicted to incur a fatal cancer if the soil contained neptunium-236, and 4 if it contained neptunium-237. (This is in comparison to the 20,000 people from the group predicted to die of cancer from all other causes per the U.S. average.) The external risk for neptunium-237 is largely due to its short-lived decay product protactinium-233.

Radiological Risk Coefficients

This table provides selected risk coefficients for inhalation and ingestion. Recommended default absorption types were used for inhalation, and dietary values were used for ingestion. Values include contributions from the short-lived neptunium decay products. Risks are for lifetime cancer mortality per unit intake (pCi), averaged over all ages and both genders (10^{-9} is a billionth, and 10^{-12} is a trillionth). Other values, including for morbidity, are also available.

Isotope	Lifetime Cancer Mortality Risk	
	Inhalation (pCi^{-1})	Ingestion (pCi^{-1})
Neptunium-235	1.0×10^{-12}	2.8×10^{-13}
Neptunium-236	2.6×10^{-9}	1.5×10^{-11}
Neptunium-237	1.5×10^{-8}	5.8×10^{-11}

For more information, see the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients and the accompanying Table 1.

Nickel

Symbol: **Ni**

Atomic Number: **28**
(protons in nucleus)

Atomic Weight: **59**
(naturally occurring)

What Is It? Nickel is a hard, silvery-white metal that is malleable and ductile. It occurs in nature as five stable isotopes. (Isotopes are different forms of an element that have the same number of protons in the nucleus but a different number of neutrons.) Nickel-58 is the most prevalent form, comprising about two-thirds of natural nickel. The other four stable isotopes and their relative abundance are nickel-60 (26%), nickel-61 (1.1%), nickel-62 (3.6%), and nickel-64 (0.9%).

Of the six major radioactive isotopes, only two – nickel-59 and nickel-63 – have half-lives long enough to warrant concern. The half-lives of all other nickel isotopes are less than six days. Nickel-59 decays with a half-life of 75,000 years by electron capture, and nickel-63 decays with a half-life of 96 years by emitting a beta particle. Both isotopes are present in wastes resulting from the reprocessing of spent nuclear fuel. Nickel-63 is generally the isotope of most concern at U.S. Department of Energy (DOE) environmental management sites such as Hanford. The long half-life of nickel-59 (with its subsequent low specific activity) combined with its low decay energy limits the radioactive hazards associated with this isotope.

Radioactive Properties of Key Nickel Isotopes

Isotope	Half-Life *(yr)*	Specific Activity *(Ci/g)*	Decay Mode	Radiation Energy *(MeV)*		
				Alpha *(α)*	Beta *(β)*	Gamma *(γ)*
Ni-59	75,000	0.082	EC	-	0.0046	0.0024
Ni-63	96	60	β	-	0.017	-

EC =electron capture, Ci = curie, g = gram, and MeV = million electron volts; a dash means the entry is not applicable. (See the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients for an explanation of terms and interpretation of radiation energies.) Values are given to two significant figures.

Where Does It Come From? Nickel is naturally present in various ores and to a lesser extent in soil. It occurs in minerals such as garnierite, millerite, niccolite, pentlandite, and pyrrhotite, with the latter two being the principal ores. It is also found in most meteorites and often serves as one of the criteria for distinguishing a meteorite from other minerals. Most of the world's supply of nickel is mined in Canada; other sources include Cuba, the former Soviet Union, China, and Australia. The United States has no large deposits of nickel and accounts for less than 1% of the annual world output. Most nickel used in the United States is imported, and about 30% of the annual consumption is from recycled sources.

The two radioactive isotopes of potential concern are produced by neutron activation of components in nuclear reactors. When a fissile nuclide such as an atom of uranium-235 fissions, it generally splits asymmetrically into two large fragments – fission products with mass numbers in the range of about 90 and 140 – and two or three neutrons. (The mass number is the sum of the number of protons and neutrons in the nucleus of the atom.) These neutrons can cause additional fissions (producing a chain reaction), escape from the reactor, or irradiate nearby materials. A number of reactor components are made of various alloys of steel that contain chromium, manganese, nickel, iron, and cobalt, and these elements can absorb neutrons resulting in radioactive isotopes, including nickel-59 and nickel-63. Nickel-59 and nickel-63 are radionuclides of concern in spent nuclear fuel (as a component of the fuel hardware) and the radioactive wastes associated with operating nuclear reactors and fuel reprocessing plants. Nickel-63 is present in much higher concentrations than nickel-59.

How Is It Used? Nickel is used in various coins and as a component of several alloys, including nichrome and permalloy, and in some stainless steels. Alnico, an alloy of aluminum, nickel, cobalt, and other metals, is used to make high-strength, permanent magnets. Nickel alloy steels are used in heavy machinery, manufacturing, armaments, tools, and high-temperature equipment, including gas turbines and environmental devices used to control emissions such as scrubbers. Nickel is also used as a protective and ornamental coating for metals susceptible to corrosion, particularly iron and steel. The nickel plate is deposited by electrolysis in a nickel solution, and the coating can be highly polished.

What's in the Environment? Nickel is present in crustal rock at a concentration of about 90 milligrams per kilogram (mg/kg). Its concentration in seawater is about 2 mg per liter (mg/L). Trace amounts of nickel-59 and nickel-63 are present around the globe from radioactive fallout. It can also be present at certain nuclear facilities as a contaminant from operating reactors and processing spent fuel. Nickel is generally one of the less mobile radioactive metals in the environment. The typical ratio of the concentration of nickel in plants to that in soil is low, estimated at 0.06 (or 6%). It also adheres quite well to soil. The

concentration of nickel associated with sandy soil particles is typically about 400 times higher than in interstitial water (in the pore spaces between the soil particles); it binds even more tightly to clay soil where concentration ratios can exceed 600. Thus, nickel is generally not a major contaminant in groundwater at DOE sites.

What Happens to It in the Body? Nickel can be taken into the body by eating food, drinking water, or breathing air. Children, and to a lesser extent adults, can also be exposed by ingesting soils. Gastrointestinal absorption from food or water is the principal source of internally deposited nickel in the general population. About 5% of the amount ingested is absorbed into the bloodstream through the intestines, while 20 to 35% of inhaled nicked is absorbed through the lungs. Of the nickel that reaches the blood, 68% is rapidly excreted in urine, while 2% remains in the kidneys with a very short biological half-life of 0.2 days (about 5 hours). The remaining 30% is evenly distributed to all remaining tissues of the body, including the kidneys, and clears with a biological half-life of more than 3 years (1,200 days). (This information is based on simplified models that do not reflect intermediate redistribution.) Nickel can be absorbed into the skin where it may stay, instead of being absorbed into the blood.

What Are the Primary Health Effects? Nickel is a radiogenic health hazard only if it is taken into the body. External gamma exposure is not a concern because nickel-63 and nickel-59 do not emit significant gamma radiation. Nickel-63 decays by emitting a beta particle and nickel-59 decays by electron capture, in which low-energy gamma radiation is emitted. While in the body, radioactive nickel presents a health hazard from the beta particles and gamma radiation; the main health concern is associated with the increased likelihood of inducing cancer. Nickel also exhibits chemical toxicity. The most common effect is an allergic reaction of the skin, with about 10 to 15% of the population sensitive to nickel (e.g., in jewelry). Less frequently, nickel induces asthmatic attacks. Both human and animal studies indicate that the respiratory system is the primary target following acute inhalation of high concentrations of nickel. Acute toxicity following ingestion of high concentrations includes effects on the gastrointestinal system, blood, and kidneys. Effects reported in studies of workers chronically exposed to airborne nickel dusts include chronic bronchitis, reduced lung function, and cancer of the lung and nasal sinus. The U.S. Environmental Protection Agency (EPA) has classified nickel subsulfide (a relatively insoluble form of nickel) as a known human carcinogen.

What Is the Risk? Lifetime cancer mortality risk coefficients have been calculated for nearly all radionuclides, including nickel *(see box at right).* While the coefficients for ingestion are somewhat lower than for inhalation, ingestion is generally the most common means of entry into the body. Similar to other radionuclides, the risk coefficients for tap water are about 70% of those for dietary ingestion. The EPA has developed toxicity values to estimate the risk of getting cancer or other adverse health effects associated with the chemical toxicity of nickel *(see box at right).* The toxicity value for estimating cancer risk following inhalation exposure is called a unit risk (UR), which is an estimate of the chance that a person will get cancer from continuous exposure to a chemical in air at a concentration of 1 milligram per cubic meter (mg/m^3). For example, using the inhalation UR, the EPA estimates that a person would have a one-in-a-million chance of developing cancer if exposed daily over a lifetime to air containing 0.002 microgram per cubic meter ($\mu g/m^3$) nickel subsulfide. The toxicity value for estimating non-cancer effects is a reference dose (RfD), which is an estimate of the highest dose that can be taken in every day without causing an adverse effect. The RfD for nickel was developed by studying test animals given relatively high doses over their lifetimes, then adjusting and normalizing those results to a mg per kg-day basis for humans. EPA has not derived a toxicity value for evaluating non-cancer effects of nickel following inhalation exposure.

Radiological Risk Coefficients

This table provides selected risk coefficients for inhalation and ingestion. Recommended default absorption types were used for inhalation, and dietary values were used for ingestion. Risks are for lifetime cancer mortality per unit intake (pCi), averaged over all ages and both genders (10^{-12} is a trillionth). Other values, including for morbidity, are also available.

Isotope	Lifetime Cancer Mortality Risk	
	Inhalation (pCi^{-1})	Ingestion (pCi^{-1})
Nickel-59	3.6×10^{-13}	2.3×10^{-13}
Nickel-63	1.4×10^{-12}	5.7×10^{-13}

For more information, see the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients and the accompanying Table 1.

Chemical Toxicity Values

Form of Nickel	Cancer Risk	Non-Cancer Effect
	Inhalation UR	*Oral RfD*
Soluble salts	None established	0.02 mg/kg-day
Nickel carbonyl	None established	None established
Nickel refinery dust	2.4×10^{-1} per mg/m^3	None established
Nickel subsulfide	4.8×10^{-1} per mg/m^3	None established

Plutonium

Symbol:	**Pu**
Atomic Number: *(protons in nucleus)*	**94**
Atomic Weight: *(not naturally occurring)*	**-**

What Is It? Plutonium in its pure form is a very heavy, silver-colored, radioactive metal about twice as dense as lead. Essentially all the plutonium on earth has been created within the past six decades by human activities involving fissionable materials. Several plutonium isotopes exist, all of which are radioactive. (Isotopes are different forms of an element that have the same number of protons in the nucleus but a different number of neutrons.)

The main plutonium isotopes at Department of Energy (DOE) environmental management sites are plutonium-238, plutonium-239, plutonium-240, and plutonium-241. Except for plutonium-241, these isotopes decay by emitting an alpha particle. Plutonium-241 decays by emitting a low-energy beta particle to americium-241, an alpha-emitting radionuclide with a half-life of 430 years that is much more radiotoxic than its parent. The maximum activity of americium-241 is about 3% of the initial activity of plutonium-241 and occurs 73 years later. An extremely small fraction of the decays of plutonium-236, plutonium-238, plutonium-240, and plutonium-242, are by spontaneous fission (SF), as are about 0.1% of the plutonium-244 decays. Plutonium-242 and plutonium-244 are generally present in relatively minute activity concentrations.

Radioactive Properties of Key Plutonium Isotopes

Isotope	Half-Life	Specific Activity *(Ci/g)*	Decay Mode	Radiation Energy *(MeV)*		
				Alpha *(α)*	Beta *(β)*	Gamma *(γ)*
Pu-236	2.9 yr	540	α	5.8	0.013	0.0021
Pu-238	88 yr	17	α	5.5	0.011	0.0018
Pu-239	24,000 yr	0.063	α	5.1	0.0067	<
Pu-240	6,500 yr	0.23	α	5.2	0.011	0.0017
Pu-241	14 yr	100	β	<	0.0052	<
Pu-242	380,000 yr	0.0040	α	4.9	0.0087	0.0014
Pu-244	83,000,000 yr	0.000018	α	4.6	0.0071	0.0012
U-240	*14 hr*	*940,000*	*β*	-	*0.14*	*0.0076*
Np-240m	*7.4 min*	*110 million*	*β*	-	*0.68*	*0.34*

Ci = curie, g = gram, and MeV = million electron volts; a "<" means the radiation energy is less than 0.001 MeV, and a dash means the entry is not applicable. (See the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients for an explanation of terms and interpretation of radiation energies.) A very small fraction (about 0.002%) of the plutonium-241 decays are by alpha-particle emission. Certain properties of uranium-240 and neptunium-240m are included here because these radionuclides accompany the plutonium-244 decays. Values are given to two significant figures.

Where Does It Come From? Plutonium was first made in large quantities by American scientists in the 1940s as part of the Manhattan Project to create the atomic bomb, and this production continued through the Cold War. Plutonium is formed when the nucleus of a uranium atom captures one or more neutrons, changing the atomic structure and creating a new element. This process occurs in nuclear reactors and mainly involves transforming uranium-238 into plutonium. (Extremely small quantities of plutonium were created naturally in sustained underground nuclear reactions estimated to have occurred about 1.9 billion years ago in Gabon, Africa. This phenomenon occurred because concentrations of uranium-235 were much higher at that time. The current uranium-235 concentration, about 0.72%, will not sustain such natural reactions.)

How Is It Used? The nuclear properties of plutonium-239, as well as our ability to produce large amounts of nearly pure plutonium-239, led to its use in nuclear weapons and nuclear power. The fissioning of uranium-235 in the reactor of a nuclear power plant produces two to three neutrons, and these neutrons can be absorbed by uranium-238 to produce plutonium-239 and other isotopes. Plutonium-239 can also absorb neutrons and fission along with the uranium-235. Plutonium fissions provide about one-third of the total energy produced in a typical commercial nuclear power plant. The use of plutonium in power plants occurs without it ever being removed from the nuclear reactor fuel, i.e., it is fissioned in the same fuel rods in which it is produced. Another isotope, plutonium-238, is used as a heat source in radiothermal generators to produce electricity for a variety of purposes including unmanned spacecraft and interplanetary probes. The United States recovered or acquired about 110,000 kilograms (kg) of plutonium between 1944 and 1994, and about 100,000 kg remains in inventory. Of this amount, over 80% is in the form of weapons-grade plutonium, primarily plutonium-239. Plutonium was generated in production reactors at DOE's Hanford and Savannah River sites, and weapons components were produced at the Rocky Flats facility. Surplus plutonium is currently stored at the Pantex Plant and other sites.

What's in the Environment? Atmospheric testing of nuclear weapons, which ceased worldwide by 1980, generated most environmental plutonium. About 10,000 kg were released to the atmosphere during these tests. Average plutonium levels in surface soil from fallout range from about 0.01 to 0.1 picocurie per gram (pCi/g).

Accidents and other releases from weapons production facilities have caused greater localized contamination. The most common form in the environment is plutonium oxide. Plutonium is typically very insoluble, with the oxide being less soluble in water than ordinary sand (quartz). It adheres tightly to soil particles and tends to remain in the top few centimeters of soil as the oxide. In aquatic systems, plutonium tends to settle out and adhere strongly to sediments, again remaining in upper layers. Typically one part of plutonium will remain in solution for every 2,000 parts in sediment or soil. A small fraction of plutonium in soil can become soluble through chemical or biological processes, depending on its chemical form. While plutonium can bioconcentrate in aquatic organisms, data have not indicated that it biomagnifies in aquatic or terrestrial food chains.

What Happens to It in the Body? When plutonium is inhaled, a significant fraction can move from the lungs through the blood to other organs, depending on the solubility of the compound. Little plutonium (about 0.05%) is absorbed from the gastrointestinal tract after ingestion, and little is absorbed through the skin following dermal contact. After leaving the intestine or lung, about 10% clears the body. The rest of what enters the bloodstream deposits about equally in the liver and skeleton where it remains for long periods of time, with biological retention half-lives of about 20 and 50 years, respectively, per simplified models that do not reflect intermediate redistribution. The amount deposited in the liver and skeleton depends on the age of the individual, with fractional uptake in the liver increasing with age. Plutonium in the skeleton deposits on the cortical and trabecular surfaces of bones and slowly redistributes throughout the volume of mineral bone with time.

Plutonium metal. *Plutonium isotopes are primarily alpha-emitters so they pose little risk outside the body. Here the plastic bag, gloves, and outer (dead) layer of skin would each alone stop the emitted alpha particles from getting into the body.*

What Is the Primary Health Effect? Plutonium generally poses a health hazard only if it is taken into the body because all isotopes except plutonium-241 decay by emitting an alpha particle, and the beta particle emitted by plutonium-241 is of low energy. Minimal gamma radiation is associated with these radioactive decays. However, there is an external gamma radiation hazard associated with plutonium-244 from it short-lived decay product neptunium-240m. Inhaling airborne plutonium is the primary concern for all isotopes, and cancer resulting from the ionizing radiation is the health effect of concern. The ingestion hazard associated with common forms of plutonium is much lower than the inhalation hazard because absorption into the body after ingestion is quite low. Laboratory studies with experimental animals have shown that exposure to high levels of plutonium can cause decreased life spans, diseases of the respiratory tract, and cancer. The target tissues in those animals were the lungs and associated lymph nodes, liver, and bones. However, these observations in experimental animals have not been corroborated by epidemiological investigations in humans exposed to lower levels.

What Is the Risk? Lifetime cancer mortality risk coefficients have been calculated for nearly all radionuclides, including plutonium (s*ee box at right).* While ingestion is generally the most common route of exposure, the risk coefficients for this route are much lower than those for inhalation. As for other radionuclides, the risk coefficients for tap water are about 80% of those for dietary ingestion. In addition to risks from internal exposures, there is an external gamma exposure risk associated with plutonium-244. To estimate a lifetime cancer mortality risk, if it is assumed that 100,000 people were continuously exposed to a thick layer of soil with an initial concentration of 1 pCi/g of plutonium-244, then 7 of these 100,000 people would be predicted to incur a fatal cancer. (This is in comparison to the 20,000 people from this group predicted to die of cancer from all other causes per the U.S. average.) This risk is largely associated with the gamma rays emitted by its short-lived decay product neptunium-240m. The external risk for the other plutonium isotopes is less than 1% of that for plutonium-244. As a note, for inhalation (the exposure of highest risk), breathing in 5,000 respirable plutonium particles of about 3 microns each is estimated to increase an individual's risk of incurring a fatal cancer about 1% above the U.S. average.

Radiological Risk Coefficients

This table provides selected risk coefficients for inhalation and ingestion. Recommended default absorption types were used for inhalation, and dietary values were used for ingestion. Risks are for lifetime cancer mortality per unit intake (pCi), averaged over all ages and both genders (10^{-9} is a billionth, and 10^{-12} is a trillionth). Other values, including for morbidity, are also available. Because values are not available for plutonium-244, these were estimated by multiplying the risk factors for plutonium-242 by the ratio of the dose conversion factors for plutonium-244 to plutonium-242.

Isotope	Lifetime Cancer Mortality Risk	
	Inhalation (pCi^{-1})	Ingestion (pCi^{-1})
Plutonium-236	2.1×10^{-8}	6.9×10^{-11}
Plutonium-238	3.0×10^{-8}	1.3×10^{-10}
Plutonium-239	2.9×10^{-8}	1.3×10^{-10}
Plutonium-240	2.9×10^{-8}	1.3×10^{-10}
Plutonium-241	2.8×10^{-10}	1.9×10^{-12}
Plutonium-242	2.8×10^{-8}	1.3×10^{-10}
Plutonium-244	2.7×10^{-8}	1.3×10^{-10}

For more information, see the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients and accompanying Table 1.

Polonium

Symbol: **Po**

Atomic Number: **84**
(protons in nucleus)

Atomic Weight: **210**
(naturally occurring)

What Is It? Polonium is a radioactive element that occurs naturally in very low concentrations in the earth's crust (at about one part in 10^{15}, or one millionth of a trillionth). Polonium was the first element discovered by Marie and Pierre Curie in 1898, while seeking the cause of radioactivity of pitchblende ore containing uranium. Polonium in its pure form is a low-melting, fairly volatile metal. Over 25 isotopes of polonium are known, with atomic masses ranging from 192 to 218 (isotopes are different forms of an element that have the same number of protons in the nucleus but a different number of neutrons.) All polonium isotopes are radioactive, with only three having appreciable half-lives: polonium-208, polonium-209, and polonium-210.

Polonium-210, historically called "radium F," is the predominant naturally occurring isotope of polonium and the one most widely used. Polonium-210 is a radioactive decay product in the natural uranium-238 decay series; along with lead-210 it is one of two relatively long-lived decay products of radon-222. Polonium-210 has a half-life of 138 days, and it decays to stable lead-206 by emitting an alpha particle. One-thousandth of a gram (1 mg) of polonium-210 emits as many alpha particles as 5 g of radium-226. The energy released by its decay is so large (140 watts/g) that a capsule containing about half a gram reaches a temperature above 500ºC.

Radioactive Properties of Key Polonium Isotopes

Isotope	Half-Life	Specific Activity *(Ci/g)*	Decay Mode	Radiation Energy *(MeV)*		
				Alpha *(α)*	Beta *(β)*	Gamma *(γ)*
Po-208	2.9 yr	590	α	5.1	<	<
Po-209	100 yr	17	α	4.9	<	<
Po-210	140 days	4,500	α	5.3	<	<

Ci = curie, g = gram, and MeV = million electron volts; a "<" means the radiation energy is less than 0.001 MeV. (See the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients for an explanation of terms and interpretation of radiation energies.) Values are given to two significant figures. Polonium-210 is a decay product of radium-226 and is also shown on that fact sheet. The basic properties of polonium-208 and polonium-209 (which are not in the natural decay series) are also given here because they are included in the general discussion below.

Where Does It Come From? Because it is produced during the decay of naturally ubiquitous uranium-238, polonium-210 is widely distributed in small amounts in the earth's crust. Although it can be produced by the chemical processing of uranium ores or minerals, uranium ores contain less than 0.1 mg polonium-210 per ton. Originally, polonium-210 was obtained from the rich pitchblende ore found in Bohemia, but it can also be obtained from aged radium salts that contain about 0.2 mg per gram of radium. Although a number of other polonium isotopes are present in the natural decay series, their short half-lives preclude any appreciable concentrations.

Due to its scarcity, polonium-210 is usually produced artificially in a nuclear reactor by bombarding bismuth-209 (a stable isotope) with neutrons. This forms radioactive bismuth-210, which has a half-life of 5 days. Bismuth-210 decays to polonium-210 through beta decay. Milligram amounts of polonium-210 have been produced by this method. The longer-lived isotopes polonium-209 (half-life 103 years) and polonium-208 (half-life 2.9 years) are also produced in reactors or particle accelerators, but these are very expensive.

How Is It Used? Polonium-210 is used mainly in static eliminators, which are devices designed to eliminate static electricity in machinery where it can be caused by processes such as paper rolling, manufacturing sheet plastics, and spinning synthetic fibers. The polonium-210 is generally electroplated onto a backing foil and inserted into a brush, tube, or other holder. Alpha particles from the polonium ionize adjacent air, and the air ions then neutralize static electricity on the surfaces in contact with the air. These devices generally need to be replaced every year because of the short half-life of this radioisotope. Polonium-210 is also used in brushes to remove dust from photographic films and camera lenses. Static eliminators typically contain from tens to hundreds of mCi (thousandth of a curie) of radioactivity. Polonium-210 can also be combined with beryllium to produce neutron sources, and in fact it was used as neutron-producing initiators of at least the first generation of atomic weapons. In addition, polonium-210 has been investigated as a heat source for thermoelectric power devices for space applications.

What's in the Environment? Polonium-210 is naturally present in all environmental media at very low concentrations. In soils, the concentration is similar to that of uranium, averaging about 1 pCi/g (or one trillionth curie per gram). Because polonium-210 is produced from the decay of radon-222 gas, it can be found in the atmosphere from which it is deposited on the earth's surface. Average annual air concentrations range from 0.005 to 0.04 pCi/m^3. Polonium-210 is also emitted to the atmosphere during the calcining of phosphate rock as part of the production of elemental phosphorous. Although direct root uptake by plants is generally small, polonium-210 can be deposited on broad-leaved vegetables. Deposition from the atmosphere on tobacco leaves results in elevated concentrations of polonium-210 in tobacco smoke, resulting in greater intakes in smokers compared to non-smokers. It is estimated that the average Western diet includes from 1 to 10 pCi of polonium-210 per day. Polonium-210 can be significantly elevated in residents of northern lands who subsist on reindeer that consume lichens, which absorb trace elements from the atmosphere.

What Happens to It in the Body? Polonium can be taken into the body by eating food, drinking water, or breathing air. Between 50% and 90% of the polonium taken in by ingestion will promptly leave the body in feces. The fraction remaining in the body enters the bloodstream. In general, the spleen and kidneys concentrate polonium more than other tissues except for temporary deposition in the lung after inhalation of an insoluble form. It is estimated that approximately 45% of ingested polonium will be deposited in the spleen, kidneys, and liver, with 10% deposited in bone marrow and the remainder distributed throughout the body. The amount of polonium in the body will decrease with a half-time of 50 days.

Studies of smokers have shown that inhaled polonium can be highly localized in the lungs, with about twice as much polonium found in the ribs of smokers compared to nonsmokers. It is estimated that the dose to the skeleton is elevated about 30% in smokers. Another source of polonium-210 in the body is its gradual ingrowth from the decay of radium-226 and lead-210 deposited in bone. The average amount of polonium-210 in the body is approximately 1 nCi (one billionth of a curie).

What Are the Primary Health Effects? Polonium-210 is a health hazard only if it is taken into the body. External exposure is not a concern because polonium is an alpha emitter. The primary means of exposure are ingestion of food and water containing polonium-210 and inhalation of polonium-contaminated dust. Inhalation is of particular concern in the vicinity of a source of airborne dust, such as a phosphate plant, and in areas of high radon concentrations, or for cigarette smokers.

Substantial radiation doses from polonium can be expected in many tissues of the body; it supplies a more nearly whole-body dose than almost all other alpha emitters. Effects are more common in the kidney than the spleen, despite a higher dose in the spleen. The lymph nodes and liver can also be affected. Polonium that is inhaled, either from radon in the air or cigarette smoke, can be deposited on the mucous lining of the respiratory tract. When alpha particles are then emitted within the lung, the cells lining the airways can be damaged, potentially leading to lung cancer over time.

What Is the Risk? Lifetime cancer mortality risk coefficients have been calculated for nearly all radionuclides, including polonium-210 *(see box at right).* Risk coefficients for inhalation are about 6 times higher than for dietary ingestion. Similar to other radionuclides, the risk coefficients for ingestion of tap water containing polonium-210 are about 75% of those shown for dietary ingestion. Polonium-210 poses no external risk when outside the body.

Radiological Risk Coefficients

This table provides risk coefficients for inhalation and absorption. Recommended default absorption types were used for inhalation, and dietary values were used for ingestion. Risks are for lifetime cancer mortality per unit intake (pCi), averaged over all ages and both genders (10^{-9} is a billionth). Other values, including for morbidity, are also available. No risk or dose conversion factors are available for Po-208 or Po-209.

Isotope	Lifetime Cancer Mortality Risk	
	Inhalation (pCi^{-1})	Ingestion (pCi^{-1})
Po-210	1.0×10^{-8}	1.6×10^{-9}

For more information, see the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients and the accompanying Table 1.

Potassium-40

Symbol:	**K(-40)**
Atomic Number: *(protons in nucleus)*	**19**
Atomic Weight: *(naturally occurring)*	**39**

What Is It? Potassium is a soft, silver-white metal. An important constituent of soil, it is widely distributed in nature and is present in all plant and animal tissues. Potassium-40 is a naturally occurring radioactive isotope of potassium. (An isotope is a different form of an element that has the same number of protons in the nucleus but a different number of neutrons.) Two stable (nonradioactive) isotopes of potassium exist, potassium-39 and potassium-41. Potassium-39 comprises most (about 93%) of naturally occurring potassium, and potassium-41 accounts for essentially all the rest. Radioactive postassium-40 comprises a very small fraction (about 0.012%) of naturally occurring potassium.

Several radioactive isotopes of potassium exist in addition to potassium-40. These isotopes all have half-lives of less than one day so they are not of concern for Department of Energy (DOE) environmental management sites such as Hanford. The half-life of potassium-40 is 1.3 billion years, and it decays to calcium-40 by emitting a beta particle with no attendant gamma radiation (89% of the time) and to the gas argon-40 by electron capture with emission of an energetic gamma ray (11% of the time). Potassium-40 is an important radionuclide in terms of the dose associated with naturally occurring radionuclides.

Radioactive Properties of Potassium-40

Isotope	Half-Life *(yr)*	Natural Abundance *(%)*	Specific Activity *(Ci/g)*	Decay Mode	Radiation Energy *(MeV)*		
					Alpha *(α)*	Beta *(β)*	Gamma *(γ)*
K-40	1.3 billion	0.012	0.0000071	β, EC	-	0.52	0.16

EC = electron capture, Ci = curie, g = gram, and MeV = million electron volts; a dash means that the entry is not applicable. (See the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients for explanation of terms and interpretation of radiation energies.) Potassium-40 decays by both emitting a beta particle (89%) and electron capture (11%). Values are given to two significant figures.

Where Does It Come From? Potassium-40 is present as a very small fraction of naturally occurring potassium, which is an element found in large amounts throughout nature. Potassium is the seventh most abundant element in the crust of the earth and the sixth most abundant element in solution in the oceans. It is present in mineral waters and brines, and in various minerals such as carnallite, feldspar, saltpeter, greensand, and sylvite. Potassium is an important constituent of fertile soil and is an essential nutrient for plant growth and in the human diet.

How Is It Used? Potassium metal, which is so soft it can be cut with a knife, is used in photoelectric cells. Potassium is one of the most reactive metals in nature, and it forms a number of compounds that have many commercial uses. For example, the white solid potassium bromide is used in photography, engraving, and lithography. The red crystal potassium chromate and yellow crystal potassium bichromate are powerful oxidizing agents used in matches and fireworks, and they are also used to dye textiles and tan leather. The white crystal potassium iodide is very soluble in water and is used in photography to prepare gelatin emulsions. It is also used in medicine to treat rheumatism and overactive thyroid glands. Potassium nitrate is a white solid used in matches, explosives, and fireworks, and it is also used to pickle meat. The purple crystal potassium permanganate is used as a disinfectant and germicide and as an oxidizing agent in various chemical reactions. The white solid potassium carbonate is used to make glass and soft soap. The white solids potassium sulfate and potassium chloride are used to fertilize soil, because potassium (along with nitrogen and phosphorous) is an essential element for plant growth. Potassium is also an essential element for humans, as a key electrolyte for maintaining basic cardiovascular functions; many people take potassium supplements as capsules or tablets. There are no specific commercial or medical uses associated with the radioactive properties of potassium-40.

What's in the Environment? Potassium is present in the earth's crust, oceans, and all organic material. Its concentration in the earth's crust is about 15,000 milligrams per kilogram (mg/kg) or 1.5%, and its concentration in seawater is about 416 mg per liter (mg/L). Because potassium-40 represents 0.012% of naturally occurring potassium, its concentration in the earth's crust is about 1.8 mg/kg, or 13 picocurie per gram (pCi/g). Potassium binds preferentially to soil, with the concentration associated with sandy soil particles estimated to be 15 times higher than in the interstitial water (in pore spaces between soil particles); it binds more tightly to loam and clay soil, so those concentration ratios are higher (above 50). Together with nitrogen and phosphorous, potassium is a major soil fertilizer, so levels of potassium-40 in soils are strongly influenced by fertilizer use; it is estimated that about 3,000 Ci of potassium-40 are added annually to U.S. soils. Potassium-40 behaves in the environment the same as other potassium isotopes, being assimilated into the tissues of all plants and animals through normal biological processes. It is the predominant radioactive component in human tissues and in most food. For example, milk contains about 2,000 pCi/L of natural potassium-40.

What Happens to It in the Body? Potassium-40 can be taken into the body by drinking water, eating food, or breathing air. Once taken in, potassium-40 behaves in the body in the same manner as other potassium isotopes. Humans require potassium to sustain biological processes, with most (including potassium-40) being almost completely absorbed upon ingestion, moving quickly from the gastrointestinal tract to the bloodstream. The potassium-40 that enters the bloodstream after ingestion or inhalation is quickly distributed to all organs and tissues. Potassium-40 is eliminated from the body with a biological half-life of 30 days. The potassium content of the body is under strict homeostatic control (in which the amount retained is actively regulated by the body to achieve the normal range required for system functions), and it is not influenced by variations in environmental levels. Hence, the potassium-40 content in the body is constant, with an adult male having about 0.1 microcurie or 100,000 pCi. Each year this isotope delivers doses of about 18 millirem (mrem) to soft tissues of the body and 14 mrem to bone.

What Is the Primary Health Effect? Potassium-40 can present both an external and an internal health hazard. The strong gamma radiation associated with the electron-capture decay process (which occurs 11% of the time) makes external exposure to this isotope a concern. While in the body, potassium-40 poses a health hazard from both the beta particles and gamma rays. Potassium-40 behaves the same as ordinary potassium, both in the environment and within the human body – it is an essential element for both. Hence, what is taken in is readily absorbed into the bloodstream and distributed throughout the body, with homeostatic controls regulating how much is retained or cleared. The health hazard of potassium-40 is associated with cell damage caused by the ionizing radiation that results from radioactive decay, with the general potential for subsequent cancer induction.

What Is the Risk? Lifetime cancer mortality risk coefficients have been calculated for nearly all radionuclides, including potassium-40 *(see box at right)*. While ingestion is generally the most common type of exposure, the risk coefficients for this route are lower than those for inhalation. As for other radionuclides, the risk coefficient for tap water is about 70% of that for dietary ingestion. In addition to risks from internal exposures, an external gamma exposure risk also exists for potassium-40. To estimate a lifetime cancer mortality risk, if it is assumed that 100,000 people were continuously exposed to a thick layer of soil with an initial average concentration of 1 pCi/g potassium-40, then 4 of these 100,000 people would be predicted to incur a fatal cancer over their lifetime. (This is in comparison to the 20,000 people from the group predicted to die of cancer from all other causes per the U.S. average.)

Radiological Risk Coefficients

This table provides selected risk coefficients for inhalation and ingestion. Maximum values are given for inhalation since no default absorption types were provided, and dietary values were used for ingestion. Risks are for lifetime cancer mortality per unit intake (pCi), averaged over all ages and both genders (10^{-9} is a billionth, and 10^{-12} is a trillionth). Other values, including for morbidity, are also available.

Isotope	Lifetime Cancer Mortality Risk	
	Inhalation (pCi^{-1})	Ingestion (pCi^{-1})
Potassium-40	2.1×10^{-10}	2.2×10^{-11}

For more information, see the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients and the accompanying Table 1.

Protactinium

Symbol: **Pa**

Atomic Number: **91**
(protons in nucleus)

Atomic Weight: **231**
(naturally occurring)

What Is It? Protactinium is a malleable, shiny, silver-gray radioactive metal that does not tarnish rapidly in air. It has a density greater than that of lead and occurs in nature in very low concentrations as a decay product of uranium. There are three naturally occurring isotopes, with protactinium-231 being the most abundant. (Isotopes are different forms of an element that have the same number of protons in the nucleus but a different number of neutrons.) The other two naturally occurring isotopes are protactinium-234 and protactinium-234m (the "m" meaning metastable), both of which have very short half-lives (6.7 hours and 1.2 minutes, respectively) and occur in extremely low concentrations. Protactinium was first identified in 1913 by Kasimir Fajans and O.H. Gohring (as the isotope protactinium-234m), and protactinium-231 was identified in 1917. The name comes from the Greek work *protos* (meaning first) and the element actinium, because protactinium is the precursor of actinium.

Of the 20 known isotopes of protactinium, only protactinium-231 has a half-life greater than one year and is a concern for Department of Energy (DOE) environmental management sites. The half-lives of all other protactinium isotopes are less than a month. Protactinium-231 is a decay product of uranium-235 and is present at sites that processed uranium ores and associated wastes. This isotope decays by emitting an alpha particle with a half-life of 33,000 years to actinium-227, which has a half-life of 22 years and decays by emitting an alpha or beta particle. Actinium-227 and its decay products are included with the list of radionuclides associated with protactinium-231 in the table to the right for completeness, as these radionuclides are typically present with protactinium-231. Much of the hazard associated with protactinium-231 is attributable to actinium-227.

Radioactive Properties of the Key Protactinium Isotope and Associated Radionuclides

Isotope	Half-Life	Natural Abundance (%)	Specific Activity (Ci/g)	Decay Mode	Radiation Energy (MeV)		
					Alpha (α)	Beta (β)	Gamma (γ)
Pa-231	33,000 yr	>99	.048	α	5.0	0.065	0.048
Ac-227	*22 yr*		*73*	*α, β*	*0.068*	*0.016*	<
Th-227 (99%)	*19 days*		*31,000*	*α*	*5.9*	*0.053*	0.11
Fr-223 (1%)	*22 min*		*39 million*	*β*	-	*0.40*	*0.059*
Ra-223	*11 days*		*52,000*	*α*	*5.7*	*0.076*	*0.13*
Rn-219	*4.0 sec*		*13 billion*	*α*	*6.8*	*0.0063*	*0.056*
Po-215	*0.0018 sec*		*30 trillion*	*α*	*7.4*	<	<
Pb-211	*36 min*		*25 million*	*β*	-	*0.46*	*0.051*
Bi-211	*2.1 min*		*420 million*	*α, β*	*6.6*	*0.010*	*0.047*
Tl-207	*4.8 min*		*190 million*	*β*	-	*0.49*	*0.0022*

Ci = curie, g = gram, and MeV = million electron volts; a "<" means the radiation energy is less than 0.001 MeV, and a dash means the entry is not applicable. (See the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients for an explanation of terms and interpretation of radiation energies.) Thorium-227 decays by both emitting an alpha particle (1%) and a beta particle (99%). Certain properties of additional radionuclides are included here because they accompany the protactinium decays. Values are given to two significant figures.

Where Does It Come From? Protactinium is widely distributed in very small amounts in the earth's crust, and it is one of the rarest and most expensive naturally occurring elements. It is present in uranium ores at a concentration of about 1 part protactinium to 3 million parts uranium. Of the three naturally occurring isotopes, protactinium-231 is a decay product of uranium-235, and protactinium-234 and protactinium-234m are decay products of uranium-238. Essentially all (99.8%) of the decays of thorium-234, which is the immediate decay product of uranium-238, are to protactium-234m; only about 0.2% are to protactinium-234. *(See the companion fact sheets on Uranium and Natural Decay Series for additional information.)*

How Is It Used? There are no industrial or commercial uses of protactinium due to its scarcity, expense, and radiotoxicity. Its only uses are associated with basic scientific research activities.

What's in the Environment? Protactinium is naturally present in soil, rocks, surface water, groundwater, plants, and animals in very low concentrations – on the order of one part per trillion, or 0.1 picocuries (pCi)/g. Higher levels are present in uranium ores and other geologic materials. Essentially all naturally occurring protactinium is present as protactinium-231. Protactinium preferentially adheres quite well to soil, and the concentration associated with sandy soil particles is typically 550 times higher than in interstitial water (water in the pore space between the soil particles); concentration ratios are even higher (about 2,000 and above) for loam and clay soils. Protactinium is generally not a major contaminant at DOE sites and is not a concern for groundwater.

What Happens to It in the Body? Protactinium can be taken into the body by eating food, drinking water, or breathing air. When protactinium is inhaled, a significant fraction can move from the lungs through the blood to other organs, depending on the solubility of the compound. Gastrointestinal absorption from food or water is a likely source of internally deposited protactinium in the general population. Most of the protactinium taken in by ingestion will promptly leave the body in feces; only about 0.05% of the amount ingested is absorbed from the gastrointestinal tract into the bloodstream. After leaving the intestine or lung, about 40% of the protactinium that does enter the bloodstream deposits in the skeleton, about 15% deposits in the liver, about 2% deposits in the kidneys, and the rest is excreted. The biological half-life in the skeleton is about 50 years. Of the protactinium deposited in the liver, 70% is assumed to be retained with a biological half-life of 10 days, with the remaining 30% having a biological half-life of 60 days. Of the protactinium deposited in the kidneys, 20% is assumed to be retained with a biological half-life of 10 days, with the remaining 80% having a biological half-life of 60 days. (This information is per simplified models that do not reflect intermediate redistribution.)

What Are the Primary Health Effects? Protactinium is generally a health hazard only if it is taken into the body, although there is a small external risk associated with the gamma rays emitted by protactinium-231 and a number of short-lived decay products of actinium-227. The main means of exposure are ingestion of food and water containing protactinium and inhalation of protactinium-contaminated dust. Ingestion is generally the exposure of concern unless there is a nearby source of contaminated airborne dust. Because protactinium is taken up in the body much more readily if inhaled rather than ingested, both exposure routes can be important. The major health concern is cancer resulting from the ionizing radiation emitted by protactinium deposited in the skeleton, liver, and kidneys. The health risks associated with protactinium-234m are included with those for uranium-238 *(see the companion fact sheet on Uranium)*. Protactinium-234m decays by emitting an energetic beta particle so precautions against this radiation are needed when handling uranium; for example, heavy rubber gloves are worn to protect the hands and forearms.

What Is the Risk? Lifetime cancer mortality risk coefficients have been calculated for nearly all radionuclides, including protactinium *(see box at right)*. The inhalation risk factor for protactinium-231 represents one of the largest risk factors for any radionuclide. Actinium-227 and its decay products account for more than 80% of this inhalation risk. While the risk factor for ingestion is much lower than for inhalation, ingestion is generally the most common means of entry into the body. Similar to other radionuclides, the risk coefficient for tap water is about 75% of that shown for dietary ingestion.

Radiological Risk Coefficients

This table provides selected risk coefficients for inhalation and absorption. Maximum values are given for inhalation as no default absorption types were provided, and dietary values were used for ingestion. These values include the contributions from the actinium-227 and its short-lived decay products. Risks are for lifetime cancer mortality per unit intake (pCi), averaged over all ages and both genders (10^{-9} is a billionth, and 10^{-12} is a trillionth). Other values, including for morbidity, are also available.

Isotope	Lifetime Cancer Mortality Risk	
	Inhalation (pCi^{-1})	Ingestion (pCi^{-1})
Pa-231	2.5×10^{-7}	6.0×10^{-10}

For more information, see the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients and accompanying Table 1.

In addition to risks from internal exposures, there is a risk from external gamma exposure to protactinium-231. Using the external gamma risk coefficients to estimate lifetime cancer mortality risks, if it is assumed that 100,000 people were continuously exposed to a thick layer of soil with an initial average concentration of 1 pCi/g protactinium-231, then 8 of these 100,000 people would be predicted to incur a fatal cancer. (This is in comparison to the 20,000 people from this group predicted to die of cancer from all other causes per the U.S. average.) As for internal exposures, much of this risk is from actinium-227 and its decay products.

Radium

What Is It? Radium is a radioactive element that occurs naturally in very low concentrations (about one part per trillion) in the earth's crust. Radium in its pure form is a silvery-white heavy metal that oxidizes immediately upon exposure to air. Radium has a density about one-half that of lead and exists in nature mainly as radium-226, although several additional isotopes are present. (Isotopes are different forms of an element that have the same number of protons in the nucleus but a different number of neutrons.) Radium was first discovered in 1898 by Marie and Pierre Curie, and it served as the basis for identifying the activity of various radionuclides. One curie of activity equals the rate of radioactive decay of one gram (g) of radium-226.

Symbol: **Ra**

Atomic Number: **88**
(protons in nucleus)

Atomic Weight: **226**
(naturally occurring)

Of the 25 known isotopes of radium, only two – radium-226 and radium-228 – have half-lives greater than one year and are of concern for Department of Energy environmental management sites. Radium-226 is a radioactive decay product in the uranium-238 decay series and is the precursor of radon-222. Radium-228 is a radioactive decay product in the thorium-232 decay series. Both isotopes give rise to many additional short-lived radionuclides, resulting in a wide spectrum of alpha, beta and gamma radiations. Lead-210, which has a 22-year half-life, is included in the list of short-lived radionuclides associated with radium-226 for completeness, as this isotope and its short-lived decay products are typically present with radium-226. Radium-226 decays slowly (half-life of 1,600 years) by emitting an alpha particle. Radium-228 has a much shorter half-life (5.8 years) and decays by emitting a beta particle. While radium-226 poses a hazard due to its long half-life, radium-228 poses a long-term hazard only if its parent (thorium-232) is present.

Radioactive Properties of Key Radium Isotopes and Associated Radionuclides

Isotope	Half-Life	Natural Abundance (%)	Specific Activity (Ci/g)	Decay Mode	Radiation Energy (MeV)		
					Alpha (α)	Beta (β)	Gamma (γ)
Ra-226	1,600 yr	>99	1.0	α	4.8	0.0036	0.0067
Rn-222	*3.8 days*		*160,000*	*α*	*5.5*	<	<
Po-218	*3.1 min*		*290 million*	*α*	*6.0*	<	<
Pb-214	*27 min*		*33 million*	*β*	-	*0.29*	*0.25*
Bi-214	*20 min*		*45 million*	*β*	-	*0.66*	*1.5*
Po-214	*0.00016 sec*		*330 trillion*	*α*	*7.7*	<	<
Pb-210	*22 yr*		*77*	*β*	-	*0.038*	*0.0048*
Bi-210	*5.0 days*		*130,000*	*β*	-	*0.39*	-
Po-210	*140 days*		*4,500*	*α*	*5.3*	<	<
Ra-228	5.8 yr	<<1	280	β	-	0.017	<
Ac-228	*6.1 hr*		*2.3 million*	*β*	-	*0.48*	*0.97*
Th-228	*1.9 yr*		*830*	*α*	*5.4*	*0.021*	0.0033
Ra-224	*3.7 days*		*160,000*	*α*	*5.7*	*0.0022*	*0.010*
Rn-220	*56 sec*		*930 million*	*α*	*6.3*	<	<
Po-216	*0.15 sec*		*350 billion*	*α*	*6.8*	<	<
Pb-212	*11 hr*		*1.4 million*	*β*	-	*0.18*	*0.15*
Bi-212	*61 min*		*15 million*	*α, β*	*2.2*	*0.47*	*0.19*
Po-212 (64%)	*0.00000031 sec*		*180,000 trillion*	*α*	*8.8*	-	-
Tl-208 (36%)	*3.1 min*		*300 million*	*β*	-	*0.60*	*3.4*

Ci = curie, g = gram, and MeV = million electron volts; a "<" means the radiation energy is less than 0.001 MeV, and a dash means the entry is not applicable. (See the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients for an explanation of terms and interpretation of radiation energies.) Bismuth-212 decays by both emitting an alpha particle (36%) and a beta particle (64%). Certain properties of additional radionuclides are included here because they accompany the radium decays. Values are given to two significant figures.

Where Does It Come From? Radium is widely distributed in small amounts in the earth's crust. It is present in all uranium and thorium minerals; its concentration in uranium ores is about one part radium to 3 million parts uranium. The chemical properties of radium are similar to those of barium, and the two substances are removed from uranium ore by precipitation and other chemical processes. Originally, radium was obtained from the rich pitchblende ore found in Bohemia. The carnotite sands of Colorado furnish some radium, but richer ores are found in the Republic of Zaire and the Great Lake Region of Canada. Radium is a major contaminant in mine and milling wastes, such as uranium mill tailings, and is present in various radioactive wastes associated with past uranium processing activities.

How Is It Used? Radium-226 is the only radium isotope used commercially. Historically, the main use of radium has been as a component in luminous paint used on the dials of watches, clocks, and other instruments, although it is no longer used for

this purpose. Radium is currently used in brachytherapy to treat various types of cancer. (Brachytherapy is a method of radiation treatment in which sealed sources are used to deliver a radiation dose at a distance of up to a few centimeters by surface, intracavitary, or interstitial application.)

What's in the Environment? Essentially all naturally occurring radium is present as radium-226. Radium exists naturally in soil, rocks, surface water, groundwater, plants, and animals in generally low concentrations – on the order of one part per trillion, or 1 picocurie (pCi)/g. Higher levels are present in uranium ores and other geologic materials. Because of the separation process used to extract uranium from ores, radium-226 is a major contaminant in uranium mill tailings. The concentration of radium in plants is typically about 0.03 (or 3%) of that in soil. However, Brazil nuts in areas of high natural radium have much higher (orders of magnitude) concentration ratios. The average concentration of radium in food has been estimated at less than 0.01 to 0.03 pCi/g. Radium preferentially adheres well to soil particles, with concentrations in sandy soil generally on the order of 500 times higher than in interstitial water (water in the pore spaces between soil particles); it is even less mobile in clay soils, with concentration ratios over 9,000. The maximum contaminant level developed by the U.S. Environmental Protection Agency for radium (as radium-226 and radium-228, combined) in drinking water supplies is 5 pCi per liter (pCi/L).

What Happens to It in the Body? Radium can be taken into the body by eating food, drinking water, or breathing air. Most of the radium taken in by ingestion (about 80%) will promptly leave the body in feces. The remaining 20% enters the bloodstream and is carried to all parts of the body. Inhaled radium can remain in the lungs for several months and will gradually enter the bloodstream and be carried throughout the body. The metabolic behavior of radium in the body is similar to that of calcium. For this reason, an appreciable fraction is preferentially deposited in bone and teeth. The amount in bone decreases with time from the exposure, generally dropping below 10% in a few months to 1% and less in a few years. Release from the bone is slow, so a portion of inhaled and ingested radium will remain in the bones throughout a person's lifetime.

What Are the Primary Health Effects? Radium poses an external as well as an internal health hazard. The strong external gamma radiation associated with several short-lived decay products of radium-226 and radium-228 makes external exposure a concern, and shielding is often needed to handle waste and other materials containing large concentrations of these radionuclides. The majority of epidemiological data on the health effects of radium-226 and radium-228 in humans comes from studies of radium dial painters, radium chemists, and technicians exposed through medical procedures in the early 1900s. These studies, as well as studies on experimental animals, indicate that chronic exposure to radium can induce bone sarcomas. The minimum latency period is seven years after the first exposure, but tumors can continue to appear throughout a lifetime.

The inhalation risk is associated primarily with radium decay products, i.e., radon and its short-lived daughters. Each of the two radium isotopes decays into a gaseous radon isotope. Radon-222 is a short-lived decay product of radium-226, and radon-220 is a short-lived decay product of radium-228. The primary hazard associated with radon arises from the inhalation of its short-lived decay products, which are charged ions that readily attach to dust particles. These particles can be inhaled into the lungs and deposited on the mucous lining of the respiratory tract. Unattached decay products tend to be inhaled deeper into the lungs where the residence time is longer. When alpha particles are then emitted within the lung, the cells lining the airways can be damaged, potentially leading to lung cancer over time.

What Is the Risk? Lifetime cancer mortality risk coefficients have been calculated for nearly all radionuclides, including radium *(see box at right).* The ingestion and inhalation coefficients for radium-226 and radium-228 are generally comparable. While ingestion is the most common means of radium entry into the body, risk coefficients for that exposure route are lower than for inhalation. Similar to other radionuclides, the risk coefficients for tap water are about 75% of those shown for dietary ingestion.

Radiological Risk Coefficients

This table provides selected risk coefficients for inhalation and absorption. Recommended default absorption types were used for inhalation, and dietary values were used for ingestion. These values include the contributions from the short-lived radium decay products. (See text for information on the external exposure pathway.) Risks are for lifetime cancer mortality per unit intake (pCi), averaged over all ages and both genders (10^{-9} is a billionth). Other values, including for morbidity, are also available.

Isotope	Lifetime Cancer Mortality Risk	
	Inhalation (pCi^{-1})	Ingestion (pCi^{-1})
Ra-226	2.4×10^{-8}	2.9×10^{-9}
Ra-228	9.0×10^{-8}	1.3×10^{-9}

For more information, see the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients and the accompanying Table 1.

In addition to risks from internal exposures, a risk from external gamma exposure is associated with these two isotopes. Using the external gamma risk coefficients to estimate lifetime cancer mortality risks, if it is assumed that 100,000 persons were continuously exposed to a thick layer of soil with an initial average concentration of 1 pCi/g, then 40 of these 100,000 people would be predicted to incur a fatal cancer if the soil contained radium-226, and 7 if it contained radium-228. (This is in comparison to the 20,000 people from the group predicted to die of cancer from all other causes per the U.S. average.) These risks are associated with the gamma rays emitted by various decay products of these two radium isotopes.

Samarium

Symbol: **Sm**

Atomic Number: **62**
(protons in nucleus)

Atomic Weight: **150**
(naturally occurring)

What Is It? Samarium is a very hard, silvery-white metal. It is a member of the lanthanide series and ignites in air at high temperatures (150°C). In nature, samarium occurs as seven isotopes, three of which are radioactive with extremely long half-lives. (Isotopes are different forms of an element that have the same number of protons in the nucleus but a different number of neutrons.) The isotopes and their natural occurrences are samarium-144 (3.1%), samarium-147 (15%), samarium-148 (11%), samarium-149 (14%), samarium-150 (7.4%), samarium-152 (27%), and samarium-154 (23%). The three naturally occurring radioactive isotopes and their half-lives are samarium-147 (110 billion years), samarium-148 (8,000 trillion years), and samarium-149 (10,000 trillion years). Their extremely long half-lives make these three radioactive isotopes essentially indistinguishable from the stable (nonradioactive) isotopes.

Nine major radioactive isotopes of samarium exist in addition to the three naturally occurring ones. Of these nine, only two have half-lives long enough to warrant concern at Department of Energy (DOE) environmental management sites such as Hanford. The half-lives of the other samarium isotopes are less than one year. Radioactive samarium isotopes are produced by the fissioning of uranium and plutonium, generally in nuclear reactors. The shorter-lived samarium isotopes (those with half-lives less than one year) produced more than 20 years ago have long since decayed away. Of the two longer-lived isotopes, samarium-146 is not a major concern at DOE sites because of its low prevalence and very long half-life (and hence low specific activity) that limits its radioactive hazards. The isotope of most concern is samarium-151, a radionuclide with a half-life of 90 years that decays by emitting a relatively low-energy beta particle.

Radioactive Properties of Key Samarium Isotopes

Isotope	Half-Life *(yr)*	Specific Activity *(Ci/g)*	Decay Mode	Radiation Energy *(MeV)*		
				Alpha *(α)*	Beta *(β)*	Gamma *(γ)*
Sm-146	100,000,000	0.000024	α	2.5	-	-
Sm-151	90	27	β	-	0.020	<

Ci = curie, g = gram, and MeV = million electron volts; a "<" means the radiation energy is less than 0.001 MeV, and a dash means the entry is not applicable. (See the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients for an explanation of terms and interpretation of radiation energies.) The three naturally occurring samarium isotopes are not included here. Values are given to two significant figures.

Where Does It Come From? Samarium is found in a variety of minerals such as monazite, bastnasite, cerite, gadolinite, and samarskite. These minerals contain different mixes of rare earth metals, which are the elements from lanthanum through lutetium in the periodic table. Samarium generally makes up less than a few percent of these minerals. China currently produces the vast majority of the rare earth metals, totaling about 70,000 metric tons (MT) annually. The United States comes in a distant second, producing around 5,000 MT annually, and samarium makes up only a small fraction of this amount.

Samarium-151 is produced by nuclear fission. When an atom of uranium-235 (or another fissile nuclide) fissions, it generally splits asymmetrically into two large fragments – fission products with mass numbers in the range of about 90 and 140 – and two or three neutrons. (The mass number is the sum of the number of protons and neutrons in the nucleus of the atom.) The fission yield of samarium-151is about 0.4%. That is, about four atoms of samarium-151 are produced per 1,000 fissions. Samarium-151 is a significant radionuclide in spent nuclear fuel, high-level radioactive wastes resulting from the processing of spent nuclear fuel, and radioactive wastes associated with the operation of nuclear reactors and fuel reprocessing plants. In addition, samarium-149 is a very good absorber of neutrons and is therefore often used in nuclear reactor control rods; capture of two neutrons by samarium-149 results in samarium-151.

How Is It Used? Samarium has a variety of commercial uses. It is used as a catalyst for certain organic reactions and in pyrophoric alloys for cigarette lighter flints. Samarium oxide is also used in special infrared absorbing glass and in the cores of carbon arc-lamp electrodes. An alloy of samarium with cobalt is used to make a magnetic material with the highest resistance to demagnetization of any known material. Because one isotope (samarium-149) is a very good neutron absorber, this element has also been used as a component in nuclear reactor control rods

What's in the Environment? Samarium is present in the earth's crust at a concentration of about 6 milligram per kilogram (mg/kg), while its concentration in seawater is about 0.00045 micrograms per liter (µg/L). Trace amounts of samarium-151 are present in soil around the globe from radioactive fallout. It can also be present at certain nuclear facilities, such as reactors and spent fuel reprocessing plants.

The transport of samarium in the environment is strongly influenced by its chemical form. It is generally one of the less mobile radioactive metals in soil, although certain forms can move downward with percolating water some distance to underlying layers of soil. Samarium preferentially adheres well to soil particles, with concentrations in sandy soil generally more than 200 times higher than in interstitial water (the water in the pore space between soil particles); it is even less mobile in clay soils, with concentration ratios over 1,000. Thus, samarium is generally not a major contaminant in groundwater at DOE sites.

What Happens to It in the Body? Samarium can be taken into the body by eating food, drinking water, or breathing air. Gastrointestinal absorption from food or water is the principal source of internally deposited samarium in the general population. Samarium is not well absorbed into the body after intake. Only about 0.05% of the amount ingested being absorbed into the bloodstream through the digestive tract, with the rest being excreted. Of the samarium that reaches the blood, 45% is deposited in the liver and another 45% is deposited on the surface of the bone, where it can irradiate the bone-forming cells; the remaining 10% is excreted. The samarium that is internally deposited is retained in the body with a biological half-life of almost 10 years (3,500 days). (This information is per simplified models that do not reflect intermediate redistribution.)

What Are the Primary Health Effects? Samarium-151 is a health hazard only if it is taken into the body. External gamma exposure is not a major concern because samarium-151 emits only a small amount of low-energy gamma radiation. While in the body, samarium-151 poses a health hazard from both the beta particles and gamma rays it emits, and the main health concern is associated with the increased likelihood of inducing cancer in the liver and bone.

What Is the Risk? Lifetime cancer mortality risk coefficients have been calculated for nearly all radionuclides, including samarium *(see box at right)*. While the coefficients for ingestion are somewhat lower than for inhalation, ingestion is generally the most common means of samarium entry into the body. Similar to other radionuclides, the risk coefficients for tap water are about 70% of those for dietary ingestion.

Radiological Risk Coefficients

This table provides selected risk coefficients for inhalation and ingestion. Maximum values are given for inhalation since no default absorption types were provided, and dietary values were used for ingestion Risks are for lifetime cancer mortality per unit intake (picocurie, pCi), averaged over all ages and both genders (10^{-9} is a billionth, and 10^{-12} is a trillionth). Other values, including for morbidity, are also available.

Isotope	Lifetime Cancer Mortality Risk	
	Inhalation (pCi^{-1})	Ingestion (pCi^{-1})
Samarium-146	1.2×10^{-8}	4.0×10^{-11}
Samarium-151	8.6×10^{-12}	4.6×10^{-13}

For more information, see the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients and the accompanying Table 1.

Selenium

What Is It? Selenium is a non-metallic mineral that resembles sulfur and can exist as a gray crystal, red powder, or vitreous black form. It occurs in nature as six stable isotopes. (Isotopes are different forms of an element that have the same number of protons in the nucleus but a different number of neutrons.) Selenium-80 is the most prevalent, comprising about half of natural selenium. The other five stable isotopes and their relative abundances are selenium-74 (0.9%), selenium-76 (9.4%), selenium-77 (7.6%), selenium-78 (24%), and selenium-82 (8.7%).

Symbol:	**Se**
Atomic Number: *(protons in nucleus)*	**34**
Atomic Weight: *(naturally occurring)*	**79**

Of the nine major radioactive selenium isotopes, only one – selenium-79 – has a half-life long enough to warrant concern at U.S. Department of Energy (DOE) environmental management sites such as Hanford. The half-life of selenium-75 is 120 days and the half-lives of all other isotopes are less than eight hours. Selenium-79 decays by emitting a beta particle with a half-life of 650,000 years with no attendant gamma radiation. Selenium-79 is present in spent nuclear fuel and the wastes resulting from reprocessing this fuel. The low specific activity and relatively low energy of its beta particle limits the radioactive hazards of this isotope.

Radioactive Properties of the Key Selenium Isotope

Isotope	Half-Life *(yr)*	Specific Activity *(Ci/g)*	Decay Mode	Radiation Energy *(MeV)*		
				Alpha *(α)*	Beta *(β)*	Gamma *(γ)*
Se-79	650,000	0.070	β	-	0.056	-

Ci = curie, g = gram, and MeV = million electron volts; a dash means the entry is not applicable. (See the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients for an explanation of terms and interpretation of radiation energies.) Values are given to two significant figures.

Where Does It Come From? Selenium occurs naturally as a trace element in most soils, rocks and waters, and it accompanies sulfur in volcanic effluents. Higher concentrations are present in soils near volcanoes and in minerals such as clausthalite, naumannite, tiemannite, and senenosulfur, but selenium does not occur in concentrated deposits. Although it cannot be economically recovered directly from the earth, selenium is commonly generated as a byproduct of electrolytic copper refining. Selenium-79 is produced by nuclear fission. When an atom of uranium-235 (or other fissile nuclide) fissions, it generally splits asymmetrically into two large fragments – fission products with mass numbers in the range of about 90 and 140 – and two or three neutrons. (The mass number is the sum of the number of protons and neutrons in the nucleus of the atom.) Selenium-79 is one such fission product, and it is produced with a yield of about 0.04%. That is, four atoms of selenium-79 are produced per 10,000 fissions. Selenium-79 is a component of spent nuclear fuel, high-level radioactive wastes resulting from processing spent fuel, and radioactive wastes associated with the operation of nuclear reactors and fuel reprocessing plants.

How Is It Used? Selenium has a number of industrial applications. It is used in photocells, solar cells, and exposure meters for photography and as a toner for photographic and copier uses. Selenium is also used to decolorize glass and to impart a scarlet red color to clear glass, glazes and enamels. Additional uses are in rectifiers and other electronic equipment, as a vulcanizing agent in processing of rubber, and as an additive in stainless steel and other alloys. Selenium compounds are used as an insecticide to combat insects that attack cultivated plants and also to treat various skin and scalp conditions (such as dandruff).

What's in the Environment? Selenium is naturally present in the earth's crust at an average concentration of about 0.05 milligram per kilogram (mg/kg), and its concentration in seawater is about 0.45 microgram per liter (µg/L). Trace amounts of selenium-79 are present in soil around the globe from radioactive fallout. It can also be present at certain nuclear facilities, such as reactors and facilities that process spent nuclear fuel. The highest concentrations of selenium-79 at the Hanford site are in areas that contain waste from processing irradiated fuel, such as in tanks in the central portion of the site. Selenium is generally one of the less mobile radioactive metals in soil as it preferentially adheres well to soil particles. Concentrations in sandy soil are estimated to be 150 times

higher than in interstitial water (in pore spaces between the soil particles), and it is even less mobile in clay soils where concentration ratios exceed 700. The low fission yield of selenium-79 limits its presence at DOE sites, so it is generally not a major groundwater contaminant at these sites. Its concentration in plants is typically 0.025 (or 2.5%) of that in soil, although levels are much higher in seleniferous plants. Certain foods are especially high in selenium, such as garlic.

What Happens to It in the Body? Selenium can be taken into the body by eating food, drinking water, or breathing air. Children, and to a lesser extent adults, can also be exposed by ingesting soils. Gastrointestinal absorption is the principal source of internally deposited selenium in the general population. About 80% of selenium incorporated in food and soluble inorganic compounds are absorbed from the gastrointestinal tract into the bloodstream. However, elemental selenium and selenides are relatively inactive biologically, and only about 5% of these forms are absorbed from the intestines. After reaching the blood, selenium selectively deposits in the liver (15%), kidneys (5%), spleen (1%) and pancreas (0.5%). The remainder is deposited uniformly throughout all other organs and tissues. Of the selenium deposited in any organ or tissue, 10% is retained with a biological half-life of 3 days, 40% is retained with a biological half-life of 30 days, and 50% is retained with a biological half-life of 150 days. (This information is per simplified models that do not reflect intermediate redistribution.) Selenium does not appear to be absorbed through the skin.

What Are the Primary Health Effects? Selenium is a health hazard only if it is taken into the body. External gamma exposure is not a concern because selenium-79 decays by emitting a beta particle with no gamma radiation. While inside the body, this selenium poses a health hazard from the beta particles emitted during its radioactive decay, and the main concern is associated with the increased likelihood of inducing cancer. Selenium (nonradioactive) is an essential element for humans that is required for normal enzyme function and is necessary for normal growth and metabolism. The recommended daily intakes are 55 and 70 µg for women and men, respectively. It is also an important antioxidant that helps protect cell membranes and prevent free radical generation. Some studies suggest that selenium decreases the risk of certain forms of cancer and diseases of the heart and blood vessels. Chronic exposures to moderate levels can cause selenosis, which is characterized by hair loss, brittle nails, and neurological abnormalities. A common cause of selenium poisoning in humans is overdosing on vitamins. Exposure to selenium or selenium dioxide in air can result in respiratory irritation, bronchial spasms, and coughing. Selenium can cause "blind staggers" in animals that drink water or eat plants or animals in areas of high selenium. The U.S. Environmental Protection Agency (EPA) has classified one specific form of selenium, selenium sulfide, as a probable human carcinogen. Selenium sulfide is not present in foods and is biologically different from the types of selenium compounds found in food or in the environment. Selenium may be protective against the toxic effects of mercury exposures.

What Is the Risk? Lifetime cancer mortality risk coefficients have been calculated for nearly all radionuclides, including selenium *(see box at right)*. Ingestion is the exposure route of most concern because this risk coefficient is about three times higher than that for inhalation, and ingestion is generally the most common means of radioactive selenium intake. Similar to other radionuclides, the risk coefficient for tap water is about 75% of that for dietary ingestion. The EPA toxicity value for estimating potential for non-cancer effects from oral exposure to chemicals is called a reference dose (RfD), which is an estimate of the highest dose that can be taken in every day without causing an adverse non-cancer effect. The RfD for selenium, shown at right, is based on studies of individuals with selenosis who live in areas with naturally high concentrations of selenium in soil. Those exposures were estimated to be at levels about 70 to 100 times higher than the recommended daily intake.

Radiological Risk Coefficients

This table provides selected risk coefficients for inhalation and ingestion. Recommended default absorption types were used for inhalation, and dietary values were used for ingestion. Risks are for lifetime cancer mortality per unit intake (picocurie, pCi), averaged over all ages and both genders (10^{-12} is a trillionth). Other values, including for morbidity, are also available.

Isotope	Lifetime Cancer Mortality Risk	
	Inhalation *(pCi^{-1})*	**Ingestion** *(pCi^{-1})*
Selenium-79	2.3×10^{-12}	6.7×10^{-12}

For more information, see the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients and the accompanying Table 1.

Chemical Toxicity Value	
Form	**Non-Cancer Effect: *Oral RfD***
Selenium and compounds	0.005 mg/kg-day

Strontium

What Is It? Strontium is a soft, silver-gray metal that occurs in nature as four stable isotopes. (Isotopes are different forms of an element that have the same number of protons in the nucleus but a different number of neutrons.) Strontium-88 is the most prevalent form, comprising about 83% of natural strontium. The other three stable isotopes and their relative abundance are strontium-84 (0.6%), strontium-86 (9.9%), and strontium-87 (7.0%). Strontium is present in nature chiefly as celestite ($SrSO_4$) and strontianite ($SrCO_3$), and it comprises about 0.025% of the earth's crust.

Symbol: **Sr**

Atomic Number: **38**
(protons in nucleus)

Atomic Weight: **88**
(naturally occurring)

Sixteen major radioactive isotopes of strontium exist, but only strontium-90 has a half-life sufficiently long (29 years) to warrant concern for nuclear facilities such as the U.S. Department of Energy Hanford site. The half-lives of all other strontium radionuclides are less than 65 days. Strontium-90 decays to yttrium-90 by emitting a beta particle, and yttrium-90 decays by emitting a more energetic beta particle with a half-life of 64 hours to zirconium-90. The main health concerns for strontium-90 are related to the energetic beta particle from yttrium-90.

Radioactive Properties of the Key Strontium Isotope and an Associated Radionuclide

Isotope	Half-Life	Specific Activity *(Ci/g)*	Decay Mode	Radiation Energy *(MeV)*		
				Alpha *(α)*	Beta *(β)*	Gamma *(γ)*
Sr-90	29 yr	140	β	-	0.20	-
Y-90	*64 hr*	*550,000*	*β*	-	*0.94*	<

Ci = curie, g = gram, and MeV = million electron volts; a dash means the entry is not applicable, and a "<" means the radiation energy is less than 0.001 MeV. (See the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients for an explanation of terms and interpretation of radiation energies.) Properties of yttrium-90 are included here because this radionuclide accompanies strontium decays. Values are given to two significant figures.

Where Does It Come From? While four stable isotopes of strontium occur naturally, strontium-90 is produced by nuclear fission. When an atom of uranium-235 (or other fissile nuclide) fissions, it generally splits asymmetrically into two large fragments – fission products with mass numbers in the range of about 90 and 140 – and two or three neutrons. (The mass number is the sum of the number of protons and neutrons in the nucleus of the atom.) Strontium-90 is such a fission product, and it is produced with a yield of about 6%. That is, about six atoms of strontium-90 are produced per 100 fissions. Strontium-90 is a major radionuclide in spent nuclear fuel, high-level radioactive wastes resulting from processing spent nuclear fuel, and radioactive wastes associated with the operation of reactors and fuel reprocessing plants.

How Is It Used? Strontium has a variety of commercial and research uses. It has been used in certain optical materials, and it produces the red flame color of pyrotechnic devices such as fireworks and signal flares. Strontium has also been used as an oxygen eliminator in electron tubes and to produce glass for color television tubes. In addition, strontium-90 has been used as an isotopic energy source in various governmental research applications, including in radiothermal generators to produce electricity for a variety of purposes including devices to power remote weather stations, navigational buoys, and satellites.

What's in the Environment? 

Beyond the four stable isotopes naturally present in soil, strontium-90 is also present in surface soil around the world as a result of fallout from past atmospheric nuclear weapons tests. Current strontium-90 levels in surface soil typically range from 0.01 to 1 picocurie per gram (pCi/g), reflecting various rainfall and wind patterns, elevation, and terrain; most levels fall between 0.05 and 0.5 pCi/g, with 0.1 pCi/g as a general average. Strontium-90 is relatively mobile and can move down through soil with percolating water to groundwater. Environmental transport of strontium is strongly influenced by its chemical form. Strontium preferentially adheres to soil particles, and the amount in sandy soil is typically about 15 times higher than in interstitial

water (in the pore spaces between soil particles); concentration ratios are typically higher (110) in clay soil. As a note, many years ago the U.S. Environmental Protection Agency (EPA) established a maximum contaminant level for strontium-90 in public drinking water supplies. That value based on extant dosimetry models is 8 pCi per liter (pCi/L). The value using current, improved dosimetry models would be 36 pCi/L.

What Happens to It in the Body? Strontium can be taken into the body by eating food, drinking water, or breathing air. Gastrointestinal absorption from food or water is the principal source of internally deposited strontium in the general population. On average, 30 to 40% of ingested strontium is absorbed into the bloodstream. The amount absorbed tends to decrease with age, and is higher (about 60%) in children in their first year of life. Adults on fasting and low-calcium diets can also increase intestinal absorption to these levels, as the body views strontium as a replacement for calcium. Strontium behaves similarly to calcium (although it is not homeostatically controlled, i.e., the body does not actively regulate levels within the cells), but living organisms generally use and retain it less effectively. For adults, about 31% of the activity entering the blood (plasma) from the gastrointestinal tract is retained by bone surfaces; the remainder goes to soft tissues or is excreted in urine and feces. Much of the activity initially deposited on bone surfaces is returned to plasma within a few days based on an updated biokinetic model that accounts for redistribution in the body. About 8% of the ingested activity remains in the body after 30 days, and this decreases to about 4% after 1 year. This activity is mainly in the skeleton.

What Are the Primary Health Effects? Strontium is a health hazard only if it is taken into the body. External gamma exposure is not a major concern because strontium-90 emits no gamma radiation and its decay product yttrium-90 emits only a small amount. Strontium-90 concentrates in bone surfaces and bone marrow, and its relatively long radioactive half-life (29 years) make it one of the more hazardous products of radioactive fallout. The health effects associated with strontium-90 were studied concurrent with development of the atomic bomb during World War II by the Manhattan Engineer District. Bone tumors and tumors of the blood-cell forming organs are the main health concern. These tumors are associated with the beta particles emitted during the radioactive decay of strontium-90 and yttrium-90.

What Is the Risk? Lifetime cancer mortality risk coefficients have been calculated for nearly all radionuclides, including strontium-90 *(see box at right).* Most of the risk is associated with the high-energy beta particle emitted by yttrium-90. Although the risk coefficient for ingestion is lower than for inhalation, ingestion is generally the most common way this radioisotope enters the body. Similar to other radionuclides, the risk coefficient for tap water is about 80% of that for dietary ingestion. In addition to potential radiogenic effects, strontium has been shown to inhibit calcification and cause bone deformities in animals, notably at high doses. The EPA toxicity value for estimating the potential for non-cancer effects from oral exposure is termed a reference dose (RfD), which is an estimate of the highest dose that can be taken in every day without causing an adverse non-cancer effect. The RfD for ingested strontium *(see box at right)* based on rachitic bone effects was developed by studying test animals given relatively high doses over their lifetimes, then adjusting and normalizing those results to a milligram per kilogram per day (mg/kg-day) basis for humans. A noncancer toxicity value for inhalation exposure has not been developed.

Radiological Risk Coefficients

This table provides selected risk coefficients for inhalation and ingestion. The recommended default absorption type was used for inhalation, and the dietary value was used for ingestion. These values include the contribution from the decay product yttrium-90. Risks are for lifetime cancer mortality per unit intake (pCi), averaged over all ages and both genders (10^{-9} is a billionth, and 10^{-12} is a trillionth). Other values, including for morbidity, are also available.

Isotope	Lifetime Cancer Mortality Risk	
	Inhalation (pCi^{-1})	Ingestion (pCi^{-1})
Strontium-90	1.0×10^{-10}	7.5×10^{-11}

For more information, see the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients and the accompanying Table 1.

Chemical Toxicity Value
Non-Cancer Effect: ***Oral RfD***
0.6 mg/kg-day

Technetium

What Is It? Technetium is a silvery gray metal that looks like platinum and tarnishes slowly in moist air. Essentially all the technetium on earth has been created by human activities involving fissionable materials. Taking its name from the Greek work *technetos* meaning artificial, it was first produced in 1937 by bombarding molybdenum with deuterons (a form of hydrogen with a neutron in the nucleus) in a cyclotron.

Symbol: **Tc**

Atomic Number: **43**
(protons in nucleus)

Atomic Weight: **-**
(not naturally occurring)

There are no stable, i.e., nonradioactive, isotopes of technetium. (Isotopes are different forms of an element that have the same number of protons in the nucleus but a different number of neutrons.) Of the ten major radioactive isotopes, only three – technetium-97, technetium-98 and technetium-99 – have half-lives sufficiently long to warrant concern over time. The half-lives of the other isotopes are less than 90 days. Only one of the three long-lived isotopes, technetium-99, is produced in sufficient quantities to be of concern at Department of Energy (DOE) environmental management sites such as Hanford. This fission product decays by emitting a beta particle to produce the stable isotope ruthenium-99. The very long half-life (and thus low specific activity) of technetium-99 limits its radioactive hazards.

Technetium-98 also decays by emitting a beta particle while technetium-97 decays by electron capture. These two radionuclides have very long half-lives (in excess of a million years). An additional radionuclide, technetium-99m (the "m" means metastable), is used in medical diagnostic procedures. This isotope has a half-life of about six hours and is a decay product of molybdenum-99, a radionuclide with a half-life of 66 hours that also decays by emitting a beta particle.

Radioactive Properties of Key Technetium Isotopes

Isotope	Half-Life *(yr)*	Specific Activity *(Ci/g)*	Decay Mode	Radiation Energy *(MeV)*		
				Alpha *(α)*	Beta *(β)*	Gamma *(γ)*
Tc-97	2.6 million	0.0014	EC	-	0.0056	0.011
Tc-98	4.2 million	0.00088	β	-	0.16	1.4
Tc-99	210,000	0.017	β	-	0.10	-

EC = electron capture, Ci = curie, g = gram, and MeV = million electron volts; a dash means the entry is not applicable. (See the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients for an explanation of terms and interpretation of radiation energies.) Values are given to two significant figures.

Where Does It Come From? Technetium is produced as a result of nuclear transformations, typically in a nuclear reactor. When an atom of a fissile nuclide such as uranium-235 fissions, it generally splits asymmetrically into two large fragments – fission products with mass numbers in the range of about 90 and 140 – and two or three neutrons. (The mass number is the sum of the number of protons and neutrons in the nucleus of the atom.) Technetium-99 and molybdenum-99 are two such fission products, with a relatively high yield of about 6%. That is, about six atoms of each isotope are produced per 100 fissions. Technetium-99m is a short-lived decay product of molybdenum-99. (An extremely small amount of technetium was created naturally in sustained underground nuclear reactions estimated to have occurred about 1.9 billion years ago in Gabon, Africa. This phenomenon occurred because much higher concentrations of uranium-235 were present at that time; the current uranium-235 concentration, about 0.72%, will not sustain such natural reactions.) Technetium-99 is a key radionuclide in spent nuclear fuel, high-level radioactive wastes resulting from processing spent fuel, and radioactive wastes associated with operating nuclear reactors and fuel reprocessing plants.

How Is It Used? Technetium is a very good corrosion inhibitor for steel, and protection can be achieved by adding only very small amounts during production. However, this use is limited by the

radioactive nature of technetium. Technetium-99m is commonly used in nuclear medicine as a radioactive tracer. In this application, the radionuclide is chemically attached to a drug chosen for its tendency to collect in specific organs of the body, and the solution is then injected into the patient. After a short time (its half-life is only 6 hours), an image is collected with a radiosensitive detector for analysis. This technique is very useful in identifying cancer metastases in locations distant from primary tumors.

What's in the Environment? Technetium is not a naturally occurring element. Technetium-99 is present in soil due to fallout from past atmospheric nuclear weapons tests. Estimated concentrations in surface soil are very low, on the order of 0.0001 picocuries per gram (pCi/g), due to its low specific activity. Technetium-99 is very mobile in the environment, especially under aerobic conditions (i.e., where oxygen is present). From the surface it can move rapidly downward with percolating water because most technetium compounds do not bind well to soil particles. The concentration associated with sandy soil particles is estimated at 0.1 of that in interstitial water (in the pore spaces between the soil particles), although technetium binds more tightly to clay soils (with concentration ratios 10 times higher). For this reason, technetium-99 has been found in groundwater at several DOE sites.

What Happens to It in the Body? Technetium pertechnetate (TcO_4) is readily taken up from the intestines and lungs following ingestion or inhalation, with about 50 to 80% of the amount ingested being transferred to the bloodstream. After reaching the blood, about 4% of the technetium pertechnetate deposits in the thyroid where it is retained with a biological half-life of 0.5 days; the other two organs to which this isotope preferentially distributes are the stomach wall (10%) and liver (3%). The rest of what enters the blood is uniformly distributed throughout all other organs and tissues with a short residence time. Of the amount that reaches body tissues, half is excreted in urine and half is excreted in feces. For the technetium that is distributed to organs other than the thyroid, about 75% leaves the body with a biological half-life of 1.6 days, 20% clears with a half-life of 3.7 days, and 5% clears with a half-life of 22 days. (This information is per simplified models that do not reflect intermediate redistribution.)

What Are the Primary Health Effects? Technetium-99 is a health hazard only if it is taken into the body. It does not pose an external hazard because it decays by emitting a relatively low-energy beta particle with no gamma radiation. The main concern is cancer induction from the beta particles associated with its radioactive decay. Technetium can concentrate in several organs depending on its chemical form, so there is no primary organ of concern. This is one reason why the short-lived isotope technetium-99m has such wide usage in nuclear medicine as a diagnostic tool. The low energy of the beta particle, the lack of significant gamma or X-rays, and the rapid excretion of technetium-99 from the body limit the potential for health effects.

What Is the Risk? Lifetime cancer mortality risk coefficients have been calculated for nearly all radionuclides, including technetium *(see box at right).* While the coefficients for ingestion are somewhat lower than for inhalation, ingestion is generally the most common means of entry into the body. Similar to other radionuclides, the risk coefficients for tap water are about 70% of those for dietary ingestion.

Radiological Risk Coefficients

This table provides selected risk coefficients for inhalation and ingestion. Recommended default absorption types were used for inhalation, and dietary values were used for ingestion. Risks are for lifetime cancer mortality per unit intake (pCi), averaged over all ages and both genders (10^{-12} is a trillionth). Other values, including for morbidity, are also available.

Isotope	Lifetime Cancer Mortality Risk	
	Inhalation (pCi^{-1})	Ingestion (pCi^{-1})
Technetium-97	7.6×10^{-13}	2.3×10^{-13}
Technetium-98	2.6×10^{-11}	6.0×10^{-12}
Technetium-99	1.3×10^{-11}	2.3×10^{-12}

For more information, see the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients and the accompanying Table 1.

Thorium

Symbol: **Th**

Atomic Number: **90** *(protons in nucleus)*

Atomic Weight: **232** *(naturally occurring)*

What Is It? Thorium is a radioactive element that occurs naturally in low concentrations (about 10 parts per million) in the earth's crust. It is about three times as abundant as uranium and about as abundant as lead or molybdenum. Thorium in its pure form is a silvery-white heavy metal that is about as dense as lead. In nature, almost all thorium is thorium-232, although several additional isotopes can be present in small amounts. (Isotopes are different forms of an element that have the same number of protons in the nucleus but a different number of neutrons.) Thorium is a soft, ductile metal that is pyrophoric in powdered form. When heated in air, thorium turnings ignite and burn brilliantly with a white light.

Of the 26 known isotopes of thorium, only 12 have half-lives greater than one second, and of these only 3 have half-lives sufficiently long to warrant a concern. These key isotopes decay very slowly by emitting an alpha particle. The half-lives of thorium-232 and thorium-230, the isotopes of most concern, are very long. Their low specific activity means these two isotopes are not highly radioactive. Both thorium-232 and thorium-230 are present in soil and ores in secular equilibrium with radium-228 and radium-226, respectively. The health risks for these two radium isotopes (shown in the Radium fact sheet) must be added to those shown here to estimate the total risk. Thorium-229 is not generally associated with nuclear fuel cycle activities previously performed by the Department of Energy (DOE), and hence is not a radionuclide of concern at DOE environmental management sites. The health risks associated with thorium-228, which has a half-life of 1.9 years, are commonly included with those for radium-228 because thorium-228 cannot persist for an extended period of time in the absence of radium-228. *(See the companion fact sheet for Radium.)*

Radioactive Properties of Key Thorium Isotopes and Associated Radionuclides

Isotope	Half-Life	Natural Abundance *(%)*	Specific Activity *(Ci/g)*	Decay Mode	Radiation Energy *(MeV)*		
					Alpha *(α)*	Beta *(β)*	Gamma *(γ)*
Th-232	14 billion yr	>99	0.00000011	α	4.0	0.012	0.0013
Th-230	77,000 yr	<<1	0.020	α	4.7	0.015	0.0016
Th-229	7,300 yr	<<1	0.22	α	4.9	0.12	0.096
Ra-225	*15 days*		*40,000*	*β*	-	*0.11*	*0.014*
Ac-225	*10 days*		*59,000*	*α*	*5.8*	*0.022*	*0.018*
Fr-221	*4.8 min*		*180 million*	*α*	*6.3*	*0.010*	*0.031*
At-217	*0.032 sec*		*1.6 trillion*	*α*	*7.1*	<	<
Bi-213	*46 min*		*20 million*	*α, β*	*0.13*	*0.44*	*0.13*
Po-213 (98%)	*0.0000042 sec*		*13,000 trillion*	*α*	*8.4*	-	-
Tl-209 (2%)	*2.2 min*		*410 million*	*β*	-	*0.69*	*2.0*
Pb-209	*3.3 hr*		*4.7 million*	*β*	-	*0.20*	-

Ci = curie, g = gram, and MeV = million electron volts; a "<" means the radiation energy is less than 0.001 MeV, and a dash means the entry is not applicable. (See the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients for explanation of terms and interpretation of radiation energies.) Bismuth-213 decays by both emitting an alpha particle (2%) and a beta particle (98%). Certain properties of additional radionuclides are included here because they accompany the thorium decays. Values are to two significant figures.

Where Does It Come From? Thorium is widely distributed in small amounts in the earth's crust. The chief commercial source is monazite sands in the United States (in North Carolina, South Carolina, Idaho, Colorado, Montana, and Florida) as well as in Brazil, India, Australia, and South Africa. The concentration of thorium oxide in monazite sands is about 3 to 10%. Thorium is also found in the minerals thorite (thorium silicate) and thorianite (mixed thorium and uranium oxides). The isotope thorium-230, a decay product of uranium-238, is found in uranium deposits as well as in uranium mill tailings.

How Is It Used? The principal use of thorium has been in the preparation of the Welsbach mantle for portable gas lanterns. These mantles contain thorium oxide with about 1% cerium oxide and other ingredients, and they glow with a dazzling light when heated in a gas flame. Thorium is an important alloying element in magnesium and is used to coat tungsten wire for components of electronic equipment. Thorium can also be added to ceramic items such as crucibles to make them more heat resistant, as well as to refractive glass to allow for smaller and more accurate camera lenses. In addition, thorium is used in welding rods and electric bulb filaments to improve product performance.

Thorium can also be used as a fuel in nuclear reactors. While thorium-232 itself is not fissile, it transforms into the fissile isotope uranium-233 upon absorption of a neutron. Although use of the thorium-232/uranium-233 fuel cycle has been demonstrated in pilot-scale studies, it has not been proven to be economically or technically viable for use in commercial nuclear power plants.

What's in the Environment? Thorium is naturally present in soil, rocks, surface water, groundwater, plants, and animals at low concentrations, on the order of ten parts per million. Higher levels are present in certain geological materials such as monzanite sands. Essentially all naturally occurring thorium is present as thorium-232. Thorium-230 is a radioactive decay product of uranium-238 and is found in low concentrations in uranium deposits and mill tailings. In its natural state, thorium occurs as an oxide (ThO_2), phosphate ($ThPO_4$), and silicate ($ThSiO_4$). Thorium preferentially adheres very tightly to soil particles, with concentrations in sandy soil generally more than 3,000 times higher than in interstitial water (water in the pore spaces between soil particles); it is even less mobile in clay soils, with concentration ratios over 5,000. The concentration of thorium in plants is typically about 0.0042 (or 0.42%) of that in soil. Data have not indicated that it biomagnifies in terrestrial or aquatic food chains. Because of its low solubility, thorium is not generally a major groundwater contaminant at DOE sites.

What Happens to It in the Body? Thorium can be taken into the body by eating food, drinking water, or breathing air. Most thorium that is inhaled or ingested in food and water is excreted within a few days, with only a small fraction being absorbed into the bloodstream. Gastrointestinal absorption from food or water is the principal source of internally deposited thorium in the general population. About 0.02 to 0.05% of the amount ingested is absorbed into the bloodstream through the intestines. Of the amount entering the blood, about 70% deposits in bone where it is retained with a biological half-life of about 22 years, 4% deposits in the liver where it is retained with a biological half-life of 700 days, and 16% is uniformly distributed to all other organs and tissues of the body where it is cleared with a biological half-life of 700 days. (per simplified models that do not reflect intermediate redistribution). Most of the remaining 10% is directly excreted. Thorium is predominantly deposited on the endosteal surfaces of mineral bone and only slowly redistributes throughout the bone volume.

What Are the Primary Health Effects? Thorium is generally a health hazard only if it is taken into the body. External gamma exposure is not a major concern because thorium emits only a small amount of gamma radiation. Although thorium-229 has a much higher gamma component than either thorium-232 or thorium-230, thorium-229 comprises a very small fraction of natural thorium. (Note that if significant concentrations of radium occur along with thorium, which is common, the external gamma dose associated with the radium must also be addressed.) The major means of exposure to thorium are ingestion of food and water containing thorium and inhalation of thorium-contaminated dust. Ingestion is generally the main exposure concern, unless there is a nearby source of airborne dust containing thorium such as uranium mill tailings. Thorium is taken up in the body much more readily if inhaled rather than ingested (see table below), so both exposure routes can be important. The main health concern for environmental exposures is generally bone cancer.

Most of the human data for thorium exposure comes from diagnostic studies. Colloidal thorium-232 dioxide (Thorotrast) was injected into patients as a radiographic contrast medium between 1928 and 1955. The epidemiological data from these studies show that the primary health effects of high doses of injected Thorotrast are blood disorders and liver tumors. Some evidence of increased incidence of lung, pancreatic, and hematopoietic cancers was found in workers occupationally exposed to thorium via inhalation. However, these workers were also exposed to several other toxic agents, so direct causation cannot be inferred. Few data are available regarding the health effects associated with low (e.g., environmental) levels of exposure from either inhalation or ingestion.

What Is the Risk? Lifetime cancer mortality risk coefficients have been calculated for nearly all radionuclides, including thorium *(see box at right)*. The risk coefficients for the isotopes of most concern (thorium-230 and thorium-232) are similar. While the coefficients for thorium-229 are about five to eight times higher, this isotope is generally not of concern at DOE sites. The risk coefficients for ingestion, the most common type of exposure, are much lower than those for inhalation for all three isotopes. Similar to other radionuclides, the risk coefficients for tap water are nearly 80% of those for dietary ingestion.

Radiological Risk Coefficients

This table provides selected risk coefficients for inhalation and ingestion. Recommended default absorption types were used for inhalation, and dietary values were used for ingestion. These values include the contributions from the short-lived thorium decay products. Risks are for lifetime cancer mortality per unit intake (picocurie, pCi), averaged over all ages and both genders (10^{-9} is a billionth, and 10^{-12} is a trillionth). Other values, including for morbidity, are also available.

Isotope	Lifetime Cancer Mortality Risk	
	Inhalation (pCi^{-1})	Ingestion (pCi^{-1})
Th-229	2.2×10^{-7}	4.7×10^{-10}
Th-230	2.7×10^{-8}	8.0×10^{-11}
Th-232	4.1×10^{-8}	9.1×10^{-11}

For more information, see the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients and the accompanying Table 1.

Tin

What Is It? Tin is a soft, silvery white metal with a highly crystalline structure that is malleable and ductile. When a bar of tin is bent, a crackling sound is emitted caused by the friction of the tin crystals. Tin exists in nature as ten stable isotopes. (Isotopes are different forms of an element that have the same number of protons in the nucleus, but a different number of neutrons.) These ten isotopes and their approximate abundances are tin-112 (1.0%), tin-114 (0.7%), tin-115 (0.3%), tin-116 (15%), tin-117 (7.7%), tin-118 (24%), tin-119 (8.6%), tin-120 (33%), tin-122 (4.6%), and tin-124 (5.8%).

Symbol:	**Sn**
Atomic Number: *(protons in nucleus)*	**50**
Atomic Weight: *(naturally occurring)*	**119**

Thirteen major radioactive isotopes of tin exist of which only two – tin-121m and tin-126 – have half-lives long enough to warrant concern at Department of Energy (DOE) environmental management sites such as Hanford. Tin-121m has a half-life of 55 years, and tin-126 has a half-life of 250,000 years; the half-lives of the other isotopes are less than one year. Both isotopes are fission products, with the fission yield of tin-126 being significantly larger than the yield of tin-121m. Thus, tin-126 is the more prevalent isotope at DOE sites. While tin-126 has a low specific activity that tends to limit its radioactive hazards, its short-lived daughter antimony-126 has a high gamma component, making external exposure to tin-126 a potential concern.

Radioactive Properties of Key Tin Isotopes and Associated Radionuclides

Isotope	Half-Life	Specific Activity *(Ci/g)*	Decay Mode	Radiation Energy *(MeV)*		
				Alpha *(α)*	Beta *(β)*	Gamma *(γ)*
Sn-121m	55 yr	54	β, IT	-	0.035	0.0049
Sn-121 (78%)	*27 hr*	*970,000*	*β*	-	*0.11*	-
Sn-126	250,000 yr	0.029	β	-	0.17	0.057
Sb-126	*12 d*	*85,000*	*β*	-	*0.28*	*2.8*

IT = isomeric transition, Ci = curie, g = gram, and MeV = million electron volts; a dash means the entry is not applicable. (See the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients for an explanation of terms and interpretation of radiation energies.) Tin-121m decays by two means: emitting a beta particle (22%) and isomeric transition (78%). Certain properties of tin-121 and antimony-126 are included here because these radionuclides accompany the tin decays. Values are given to two significant figures.

Where Does It Come From? The principal ore of tin is the mineral cassiterite (tin oxide, SnO_2) found either in rocks within often irregular veins or lodes, or in debris that has built up from the gradual wearing down of tin-bearing rocks to form alluvial deposits found in river beds and valleys or on the ocean floor close to shore. Most of the world's tin is produced by Malaysia, Brazil, Indonesia, Thailand, Bolivia, and Australia. The United States imports more than one-fifth of the average annual world production of tin.

Tin-121m and tin-126 are produced by nuclear fission. When an atom of uranium-235 (or other fissile nuclide) fissions, it generally splits asymmetrically into two large fragments – fission products with mass numbers in the range of about 90 and 140 – and two or three neutrons. (The mass number is the sum of the number of protons and neutrons in the nucleus of the atom.) Tin-121m and tin-126 are two such fission products; the fission yield of tin-121m is very low (about 0.00003%), while the fission yield of tin-126 is about 0.06%. That is, much less than one atom of tin-121m and six atoms of tin-126 are produced per 10,000 fissions. These two tin isotopes are components of spent nuclear fuel, high-level radioactive wastes resulting from processing spent fuel, and radioactive wastes associated with the operation of nuclear reactors and fuel reprocessing plants.

How Is It Used? Tin is used in a number of industrial processes throughout the world. Tinplate (sheet steel coated with a thin layer of tin) is the primary material used for food cans, and tin is also commonly used in bakeware and food storage containers. Tin is used to produce common alloys such as bronze (tin and copper) and solder (tin and lead). It is also used as an alloy with titanium in the aerospace industry and as an ingredient

in some insecticides. Stannic sulfide, also known as mosaic gold, is used in powdered form for bronzing articles made of plaster of paris or wood. Dental amalgam contains about 13% tin together with silver and mercury, and mercury-free alternative dental filling materials with about twice the amount of tin are under development. Tin and tin-alloy coatings are widely used in the manufacture of bearings and in many kinds of machinery and fabricated parts, for both their anti-corrosion and lubricant properties.

What's in the Environment? Tin is present in the earth's crust at a concentration of about 2.2 milligram per kilogram (mg/kg), and its concentration in seawater is about 0.01 micrograms (µg) per liter. Trace amounts of tin-121m and tin-126 are present in soil around the globe from radioactive fallout. These isotopes can also be present at certain nuclear facilities, such as reactors and spent fuel reprocessing plants. Tin is generally one of the less mobile radioactive metals in soil, although certain forms can move downward with percolating water some distance to underlying layers of soil depending on site conditions. Tin preferentially adheres quite well to soil, and the concentration associated with sandy soil particles is estimated to be about 130 times higher than in interstitial water (the water in the pore spaces between the soil particles), with even higher concentration ratios in loam and clay. Thus, tin is generally not a major contaminant in groundwater at DOE sites.

What Happens to It in the Body? Tin can be taken into the body by eating food, drinking water, or breathing air. Gastrointestinal absorption from food or water is the principal source of internally deposited tin in the general population. Gastrointestinal absorption is generally quite low, with only about 2% of the amount ingested being transferred to the bloodstream. Thirty-five percent of tin that reaches the blood is deposited in mineral bone, 15% is distributed throughout all other organs and tissues of the body, and the remaining 50% is excreted. Of the tin deposited in any organ or tissue, 20% is retained with a biological half-life of 4 days, 20% is retained with a biological half-life of 25 days, and 60% is retained with a biological half-life of 400 days. (This information is per simplified models that do not reflect intermediate redistribution.)

What Are the Primary Health Effects? Tin poses an external as well as an internal health hazard. The strong gamma radiation associated with tin-126 makes external exposure to this isotope a concern. The main means of internal exposure are ingestion of food and water containing tin isotopes. While in the body, tin poses a health hazard from both the beta particles and gamma rays, and the main health concern is associated with the increased likelihood of inducing cancer in bone and other organs and tissues in which it may be deposited.

What Is the Risk? Lifetime cancer mortality risk coefficients have been calculated for nearly all radionuclides, including tin *(see box at right).* While the coefficients for ingestion are lower than for inhalation, ingestion is generally the most common means of entry into the body. Similar to other radionuclides, the risk coefficients for tap water are about 70% of those for dietary ingestion.

In addition to risks from internal exposures, there is an external gamma exposure risk associated with tin-126. To estimate a lifetime cancer mortality risk, if it is assumed that 10,000 people were continuously exposed to a thick layer of soil with an initial average concentration of 1 pCi/g tin-126, then 6 of these 10,000 people would be predicted to incur a fatal cancer. (This is in comparison to the 2,000 people from this group predicted to die of cancer from all other causes per the U.S. average.) This risk is largely associated with the gamma ray emitted by its short-lived decay product antimony-126.

Radiological Risk Coefficients

This table provides selected risk coefficients for inhalation and ingestion. Maximum values are given for inhalation since no default absorption types were provided, and dietary values were used for ingestion. These values include the contributions from the short-lived tin decay products. Risks are for lifetime cancer mortality per unit intake (picocurie, pCi), averaged over all ages and both genders (10^{-9} is a billionth, and 10^{-12} is a trillionth). Other values, including for morbidity, are also available.

Isotope	Lifetime Cancer Mortality Risk	
	Inhalation (pCi^{-1})	Ingestion (pCi^{-1})
Tin-121m	4.1×10^{-11}	2.9×10^{-12}
Tin-126	3.9×10^{-10}	3.0×10^{-11}

For more information, see the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients and accompanying Table 1.

Tritium (Hydrogen-3)

Symbol: **H** (H-3)

Atomic Number: **1**
(protons in nucleus)

Atomic Weight: **1**
(naturally occurring H)

What Is It? Tritium is the only radioactive isotope of hydrogen. (An isotope is a different form of an element that has the same number of protons in the nucleus but a different number of neutrons.) The nucleus of a tritium atom consists of a proton and two neutrons. This contrasts with the nucleus of an ordinary hydrogen atom (which consists solely of a proton) and a deuterium atom (which consists of one proton and one neutron). Ordinary hydrogen comprises over 99.9% of all naturally occurring hydrogen. Deuterium comprises about 0.02%, and tritium comprises about a billionth of a billionth (10^{-16} percent) of natural hydrogen.

Radioactive Properties of Tritium

Isotope	Half-Life *(yr)*	Natural Abundance *(%)*	Specific Activity *(Ci/g)*	Decay Mode	Radiation Energy *(MeV)*		
					Alpha *(α)*	Beta *(β)*	Gamma *(γ)*
H-3	12	<<1	9,800	β	-	0.0057	-

Ci = curie, g = gram, and MeV = million electron volts; a dash means the entry is not applicable. (See the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients for an explanation of terms and interpretation of radiation energies.) Values are given to two significant figures.

The most common forms of tritium are tritium gas (HT) and tritium oxide, also called "tritiated water." In tritiated water, a tritium atom replaces one of the hydrogen atoms so the chemical form is HTO rather than H_2O. The chemical properties of tritium are essentially the same as those of ordinary hydrogen. It decays with a half-life of 12 years by emitting a beta particle to produce helium-3. Tritium has a relatively high specific activity and is generated by both natural and artificial processes. It is of concern at Department of Energy (DOE) sites that operated tritium production facilities, such as Hanford.

Where Does It Come From? Tritium is naturally present as a very small percentage of ordinary hydrogen in water, both liquid and vapor. This tritium is produced as a result of the interaction of cosmic radiation with gases in the upper atmosphere, and the natural steady-state global inventory is about 7.3 kilograms (kg). (About five times this amount remains from past atmospheric nuclear weapons tests.) After being produced in the atmosphere, it is readily incorporated into water and falls to earth as rain, thus entering the natural hydrological cycle. Tritium is also produced as a fission product in nuclear weapons tests and in nuclear power reactors, with a yield of about 0.01%. That is, about one atom of tritium is produced per 10,000 fissions. Each year a large commercial nuclear power reactor produces about 20,000 curies (2 grams) of tritium, which is generally incorporated in the nuclear fuel and cladding.

Because little tritium is naturally present, it must be produced artificially for use on a practical scale. Tritium can be made in production nuclear reactors, i.e., reactors designed to optimize the generation of tritium and special nuclear materials such as plutonium-239. Tritium is produced by neutron absorption of a lithium-6 atom. The lithium-6 atom, with three protons and three neutrons, and the absorbed neutron combine to form a lithium-7 atom with three protons and four neutrons, which instantaneously splits to form an atom of tritium (one proton and two neutrons) and an atom of helium-4 (two protons and two neutrons). The United States has recovered an estimated 225 kg of tritium, of which 150 kg has decayed into helium-3, leaving a current inventory of approximately 75 kg. While tritium can also be produced in accelerators by bombarding helium-3 with neutrons, this approach has not been proven on a large scale.

How Is It Used? Tritium is used as a component in nuclear weapons to boost the yield of both fission and thermonuclear (or fusion) warheads. Tritium is also used as a tracer in biological and environmental

studies, and as an agent in luminous paints such as those used to make building exit signs, airport runway lights, and watch dials.

What's in the Environment? Tritium is present in water (liquid and vapor) as a result of natural processes in the atmosphere, as well as from fallout from past atmospheric nuclear weapons tests and the operation of nuclear reactors and fuel reprocessing plants. The form of most concern, tritium oxide (HTO), is generally indistinguishable from normal water and can move rapidly through the environment in the same manner as water. Tritium is naturally present in surface waters at about 10 to 30 picocuries per liter (pCi/L). The maximum contaminant level developed by the Environmental Protection Agency for tritium in drinking water supplies is 20,000 pCi/L or 0.02 microcuries per liter (a picocurie is a millionth of a microcurie). Higher concentrations can be present in water at facilities that produce and utilize tritium, including certain DOE sites.

What Happens to It in the Body? Tritium can be taken into the body by drinking water, eating food, or breathing air. It can also be taken in through the skin. Nearly all (up to 99%) inhaled tritium oxide can be taken into the body from the lungs, and circulating blood then distributes it to all tissues. Ingested tritium oxide is also almost completely absorbed, moving quickly from the gastrointestinal tract to the bloodstream. Within minutes it is found in varying concentrations in body fluids, organs, and other tissues. Skin absorption of airborne tritium oxide can also be a significant route of uptake, especially for exposure to high concentrations of tritiated water vapor, as could occur under conditions of high humidity during hot weather, because of the normal movement of water through the skin. For someone immersed in a cloud of airborne tritium oxide (HTO), the uptake by absorption through the skin would be about half that associated with inhalation. No matter how it is taken into the body, tritium is uniformly distributed through all biological fluids within one to two hours. Tritium is eliminated from the body with a biological half-life of 10 days, the same as for water. During the time it is in the body, a small fraction of the tritium is incorporated into easily exchanged hydrogen sites in organic molecules.

What Is the Primary Health Effect? Tritium poses a health hazard only if it is taken into the body, because tritium decays by emitting a low-energy beta particle with no gamma radiation. This beta particle cannot penetrate deeply into tissue or travel far in air. The most likely form of uptake is as tritium oxide (or tritiated water), as the uptake of tritium gas is typically very low (less than 1%). Tritiated water behaves the same as ordinary water, both in the environment and in the human body. Hence, a significant fraction of the inhaled and ingested tritium is directly absorbed into the bloodstream. The health hazard of tritium is associated with cell damage caused by the ionizing radiation that results from radioactive decay, with the potential for subsequent cancer induction.

What Is the Risk? Lifetime cancer mortality risk coefficients have been calculated for nearly all radionuclides, including tritium *(see box at right).* The values given here are for tritiated water; additional values are available, including for inhalation and ingestion of organically bound tritium and inhalation of tritium on particulates. As for other nuclides, the risk coefficient for tap water is about 80% of that for dietary ingestion.

Radiological Risk Coefficients

This table provides selected risk coefficients for inhalation and dietary ingestion of tritiated water. Risks are for lifetime cancer mortality per unit intake (pCi), averaged over all ages and both genders (10^{-12} is a trillionth). Other values, including for morbidity, are also available.

Isotope	Lifetime Cancer Mortality Risk	
	Inhalation (pCi^{-1})	Ingestion (pCi^{-1})
Tritium (H-3)	3.9×10^{-14}	4.4×10^{-14}

For more information, see the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients and the accompanying Table 1.

Uranium

Symbol: **U**

Atomic Number: **92**
(protons in nucleus)

Atomic Weight: **238**
(naturally occurring)

What Is It? Uranium is a radioactive element that occurs naturally in low concentrations (a few parts per million, ppm) in soil, rock, surface water, and groundwater. It is the heaviest naturally occurring element, with an atomic number of 92. Uranium in its pure form is a silver-colored heavy metal that is nearly twice as dense as lead. In nature, uranium exists as several isotopes: primarily uranium-238, uranium-235, and a very small amount of uranium-234. (Isotopes are different forms of an element that have the same number of protons in the nucleus but a different number of neutrons.) In a typical sample of natural uranium, almost all the mass (99.27%) consists of atoms of uranium-238. Less than 1% (about 0.72%) of the mass consists of atoms of uranium-235, and a very small amount (0.0055% by mass) is uranium-234.

Uranium decays very slowly by emitting an alpha particle. The half-life of uranium-238 is 4.5 billion years, which means it is not very radioactive as indicated by its low specific activity. The very long half-lives of these isotopes are the reason why uranium still exists on earth. Three additional isotopes (uranium-232, uranium-233, and uranium-236) are not naturally present but can be produced by nuclear transformations. These three isotopes also decay by emitting an alpha particle.

Radioactive Properties of Key Uranium Isotopes and Associated Radionuclides

Isotope	Half-Life	Natural Abundance *(%)*	Specific Activity *(Ci/g)*	Decay Mode	Radiation Energy *(MeV)*		
					Alpha *(α)*	Beta *(β)*	Gamma *(γ)*
U-232	72 yr	0	22	α	5.3	0.017	0.0022
U-233	160,000 yr	0	0.0098	α	4.8	0.0061	0.0013
U-234	240,000 yr	0.0055	0.0063	α	4.8	0.013	0.0017
U-235	700 million yr	0.72	0.0000022	α	4.4	0.049	0.16
Th-231	*26 hr*		*540,000*	*β*	-	*0.17*	*0.026*
U-236	23 million yr	0	0.000065	α	4.5	0.011	0.0016
U-238	4.5 billion yr	>99	0.00000034	α	4.2	0.010	0.0014
Th-234	*24 days*		*23,000*	*β*	-	*0.060*	*0.0093*
Pa-234m	*1.2 min*		*690 million*	*β*	-	*0.82*	*0.012*

Ci = curie, g = gram, and MeV = million electron volts; a dash means the entry is not applicable. (See the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients for an explanation of terms and interpretation of radiation energies.) Properties of thorium-231, thorium-234, and protactinium-234m are included here because these radionuclides accompany the uranium decays. Values are given to two significant figures.

Where Does It Come From? While small amounts of natural uranium are found almost everywhere in soil, rock, and water, uranium ores are found in just a few places – usually in hard rock or sandstone, in deposits normally covered with earth and vegetation. Uranium has been mined in the southwest United States, Canada, Australia, parts of Europe, the former Soviet Union, Namibia, South Africa, Niger, and elsewhere. It is a contaminant at many U.S. Department of Energy sites (including Hanford) and other facilities that used natural uranium, including mining, milling, and production facilities.

How Is It Used? For many years, uranium was used to color ceramic glazes, producing colors that ranged from orange-red to lemon yellow. It was also used for tinting in early photography. The radioactive properties of uranium were not recognized until 1896, and its potential for use as an energy source was not realized until the middle of the 20th century. In nuclear reactors, uranium serves as both a source of neutrons (via the fission process) and a target material for producing plutonium. (Plutonium-239 is produced when uranium-238 absorbs a neutron.) Today, its primary use is as fuel in nuclear power reactors to generate electricity. Uranium is also used in small nuclear reactors to produce isotopes for medical and industrial purposes around the world. Natural uranium must be enriched in the isotope uranium-235 for use as a nuclear fuel in light-water reactors, and this enrichment has generally been achieved by gaseous diffusion techniques. Highly enriched uranium is a primary component of certain nuclear weapons. A byproduct of the enrichment process is depleted uranium, i.e., uranium depleted in the isotope 235. *(See the companion fact sheet for Depleted Uranium.)*

What's in the Environment? Uranium is naturally present in all environmental media at very low concentrations (a few parts per million). Higher levels are present in certain areas, including those with natural uranium ores such as in the southwestern United States. In its natural state, uranium occurs as an oxide ore, U_3O_8. Additional compounds that may be present include other oxides (UO_2, UO_3) as well as fluorides, carbides or carbonates, silicates, vanadates, and phosphates. In addition to the three naturally occurring isotopes, uranium-232, uranium-233, and uranium-236 are present at Hanford. At that site, uranium-233 was produced in targets and disposed of in the 300 Area; uranium-236 measurements in

groundwater there have been used to distinguish the presence of natural uranium from uranium associated with reprocessed nuclear fuel. The environmental transport of uranium is strongly influenced by its chemical form. It is generally one of the more mobile radioactive metals and can move down through soil with percolating water to underlying groundwater. Uranium preferentially adheres to soil particles, with a soil concentration typically about 35 times higher than that in the interstitial water (the water between the soil particles); concentration ratios are usually much higher for clay soils (e.g., 1,600). Uranium can bioconcentrate in certain food crops and in terrestrial and aquatic organisms. However, data do not indicate that it biomagnifies in terrestrial or aquatic food chains. The U.S. Environmental Protection Agency (EPA) established a maximum contaminant level (MCL) for uranium in drinking water of 0.030 milligram per liter (mg/L). This equates to about 27 picocuries (pCi) per liter considering the ratio of isotopes typically present in drinking water sources.

What Happens to It in the Body? Uranium can be taken into the body by eating food, drinking water, or breathing air. Gastrointestinal absorption from food or water is the main source of internally deposited uranium in the general population. After ingestion, most uranium is excreted within a few days and never enters the bloodstream. The small fraction (0.2 to 5%) that is absorbed into the bloodstream is deposited preferentially in bone (about 22%) and kidneys (about 12%), with the rest being distributed throughout the body (12%) and excreted. Most of what goes to the kidneys leaves within a few days (in urine), while that deposited in bone can remain for many years. After inhalation, generally only a small fraction penetrates to the lung's alveolar region, where it can remain for years and from which it can also enter the bloodstream.

What Are the Primary Health Effects? Uranium is a health hazard only if it is taken into the body. External exposure is generally not a major concern because uranium emits only a small amount of low-energy gamma radiation. While uranium-235 has a much higher gamma component than either uranium-234 or uranium-238, uranium-235 only comprises about 2% of the total activity of natural uranium. The primary means of exposure are ingestion of food and water containing uranium isotopes and inhalation of uranium-contaminated dust. Ingestion is usually the exposure of concern unless there is a nearby source of airborne dust, such as a uranium mine or mill. Because uranium is absorbed much more readily if inhaled rather than ingested, both exposure routes can be important. The major health concern is kidney damage caused by the chemical toxicity of soluble uranium compounds. That effect can be reversible depending on the level of exposure. (Uranium has also been implicated in reproductive effects in laboratory animals and developmental effects in young animals, but it is not known if these problems exist for humans.) A second concern is for uranium deposited in bone, which can lead to bone cancer as a result of the ionizing radiation associated with its radioactive decay products.

What Is the Risk? Lifetime cancer mortality risk coefficients have been calculated for nearly all radionuclides, including uranium *(see box at right).* Although ingestion is generally the common means of entry, these risk coefficients are much lower than those for inhalation so both exposure routes need to be considered. Similar to other radionuclides, the risk coefficients for tap water are about 75% of those for dietary ingestion. On an activity (curie) basis, the risk coefficients are essentially the same for all uranium isotopes (although the factor for ingesting uranium-232 is somewhat higher), so the risk is essentially independent of the ratio of various isotopes in a compound. For this reason, the risk from exposure to depleted uranium is essentially the same as for enriched uranium on an activity basis. Uranium-235 also poses an external gamma exposure risk. To estimate a lifetime cancer mortality risk, if it is assumed that 100,000 people were continuously exposed to a thick layer of soil with an initial concentration of 1 pCi/g uranium-235, then 3 of those 100,000 people would be predicted to incur a fatal cancer. (This is in comparison to about 20,000 people from the group predicted to die of cancer from all other causes per the U.S. average.) Uranium can also kidney damage due to its chemical toxicity. The toxicity value used to estimate the potential for non-cancer effects following ingestion is a reference dose (RfD), which is an estimate of the highest dose that can be taken in every day over a lifetime without causing an adverse health effect. In addition to the on-line RfD shown above, EPA more recently derived a value of 0.0006 mg/kg-day to support the drinking water MCL. These values were developed by analyzing the biological effects of test animals given relatively large amounts of uranium, then adjusting and normalizing the results to a mg/kg-day basis for humans.

Radiological Risk Coefficients

This table provides selected risk coefficients for inhalation and ingestion. Recommended default absorption types were used for inhalation, and dietary values were used for ingestion. These values include contributions from short-lived uranium decay products. Risks are for lifetime cancer mortality per unit intake (pCi), averaged over all ages and both genders (10^{-9} is a billionth, and 10^{-12} is a trillionth). Other values, including for morbidity, are also available.

Isotope	Lifetime Cancer Mortality Risk	
	Inhalation (pCi^{-1})	Ingestion (pCi^{-1})
Uranium-232	1.8×10^{-8}	2.7×10^{-10}
Uranium-233	1.1×10^{-8}	6.3×10^{-11}
Uranium-234	1.1×10^{-8}	6.1×10^{-11}
Uranium-235	9.5×10^{-9}	6.2×10^{-11}
Uranium-236	9.9×10^{-9}	5.8×10^{-11}
Uranium-238	8.8×10^{-9}	7.5×10^{-11}

For more information, see the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients and the accompanying Table 1.

Chemical Toxicity Value
Non-Cancer Effect: ***Oral RfD*** *(soluble salts)*
0.003 mg/kg-day

Depleted Uranium

(This complements the general fact sheet for uranium.)

What Is Depleted Uranium? Depleted uranium is created as a byproduct during the processing of natural uranium to make it suitable for use as fuel in nuclear power plants or as a component in nuclear weapons. In natural uranium, 99.27% of the mass consists of atoms of uranium-238. About 0.72% of the mass consists of atoms of uranium-235, and a very small amount (0.0055% by mass) is uranium-234. Although uranium-235 is the rarer of the two major uranium isotopes, it is the one that most readily undergoes nuclear fission and is thus the most useful for common nuclear applications. For most of these applications, the proportion of the uranium-235 isotope found in natural uranium must be increased through a process called enrichment.

The uranium enrichment process results in the production of "enriched" uranium (containing >0.72% uranium-235) and "depleted" uranium (containing <0.72% uranium-235). The uranium enrichment process also removes much of the uranium-234 from the depleted uranium. Most depleted uranium in the United States contains between 0.2 and 0.4% uranium-235, with the remainder being uranium-238 and a slight amount of uranium-234. Depleted uranium is less radioactive than natural uranium because some of the uranium-235 and most of the uranium-234 have been removed. Depleted uranium will not undergo a nuclear chain reaction.

Specific Activity of Enriched and Depleted Uranium
(For additional information, see the Uranium fact sheet.)

The specific activity (SA) of a uranium compound depends on its isotopic composition. The SA of natural uranium (containing 0.72% uranium-235) is 6.77×10^{-7} curies per gram (Ci/g). The SA for other mixtures of uranium-238, uranium-235, and uranium-234 can be estimated using the approach developed by the U.S. Nuclear Regulatory Commission (see 10 CFR 20):

For depleted uranium (uranium-235 containing less than 0.72%):
$SA = 3.6 \times 10^{-7}$ Ci/g

For enriched uranium (uranium-235 containing more than 0.72%):
$SA = \{0.4 + 0.38(\text{enrichment}) + 0.0034(\text{enrichment})^2\}\times10^{-6}$ Ci/g, where enrichment is the percent uranium-235.

Thus, the SA of depleted uranium is approximately half that of natural uranium. (To express SA in standard international units, multiply the value in Ci/g by 3.7×10^{10} becquerels [Bq]/Ci.)

How Is It Produced? In the United States, uranium is enriched by the gaseous diffusion process in which the compound uranium hexafluoride (UF_6) is heated and converted from a solid to a gas. The UF_6 gas is forced through a long series of compressors and converters with porous barriers. Because uranium-235 has a slightly lighter isotopic mass than uranium-238, UF_6 molecules made with uranium-235 diffuse through the barriers at a slightly higher rate than do the molecules containing uranium-238 (the uranium-234 molecules diffuse through the barriers at a rate even higher than that of the uranium-235 molecules).

At the end of the process there are two UF_6 streams, with one having a higher concentration of uranium-235 than the other. The stream with the higher uranium-235 concentration is referred to as enriched UF_6, while the stream that is reduced in its concentration of uranium-235 is referred to as depleted UF_6. The depleted UF_6 can be converted to other chemical forms, such as depleted uranium oxide or depleted uranium metal. Other methods can be used to enrich uranium, and they too produce depleted uranium as a byproduct. The most common enrichment process used outside of the United States is gas centrifuge enrichment. Laser-based enrichment processes have also been investigated but not commercially developed.

How Is It Used? Although the vast majority of depleted uranium is stored as a byproduct of the enrichment process at the gaseous diffusion plant sites, several current and potential uses exist. Because of its high density, depleted uranium is currently used for radiation shielding. Depleted uranium metal was previously used on large commercial aircraft as counterweights in the wings. Military applications of depleted uranium include use as tank armor, armor-piercing projectiles (antitank weapons), and counterweights in missiles and high-performance aircraft. In a potential future use, depleted uranium could be mixed with highly enriched uranium from retired nuclear weapons to produce nuclear reactor fuel. This process is called blending, and to date only natural or slightly enriched uranium has been considered for this application. Limited amounts of depleted uranium can also be used for the fabrication of mixed uranium and plutonium oxide (MOX) fuel, which is currently being evaluated for use in commercial nuclear power plants.

What Are Depleted Uranium Munitions? Research, tests, and evaluations were conducted during the 1970s to develop improved armor-penetrating munitions capable of defeating a heavily armored target. High-density materials such as tungsten and depleted uranium were considered candidates for this type of munitions; depleted uranium was ultimately selected because of its very high density, availability, noncompetitive uses, and pyrophoricity (spontaneous combustion upon exposure to air).

The two main antitank weapons made of depleted uranium are the 120-millimeter (mm) cannon shell used by the Army's M1 Abrams tank, and the 30-mm munitions used by A-10 antitank aircraft. Depleted uranium weapons are also used in Britain's Challenger tank, and the U.S. and British Navy Phalanx gun systems. The M1 Abrams tank fires a 120-mm shell that contains a kinetic energy penetrator, a solid rod with a pointed tip. The penetrator is made from about 5 kilograms (kg) (11 pounds [lb]) of depleted uranium metal alloyed with less than 1% titanium. The shell is a sabot round, meaning that the penetrator is contained within a carrier as it is fired from the barrel. The carrier separates from the penetrator after exiting the gun barrel. In addition, many M1 Abrams tanks incorporate depleted uranium metal as part of the external armor.

120-mm antitank kinetic energy rounds fired by the M1 Abrams tank

Depleted uranium rounds are also fired by a 30-mm, seven-barrel gatling gun mounted in the nose of the A-10 Thunderbolt aircraft, the only U.S. military plane that employs depleted uranium rounds. Depleted uranium is the primary munition for the A-10 Thunderbolt for combat. Each 30-mm depleted uranium projectile contains approximately 0.3 kg (0.66 lb) of extruded depleted uranium metal alloyed with 0.75% titanium. The projectile is encased in a 0.8-mm-thick aluminum shell as the final depleted uranium round.

Depleted uranium penetrators have a “sharpening effect” upon impact that allows greater penetration through armor. Weapon testing shows that when a depleted uranium round penetrates an armored vehicle, it may pass completely through the vehicle or ricochet around and fragment inside the vehicle. Metal fragments from the penetrator and the vehicle’s hull can scatter inside the vehicle, killing and injuring personnel, destroying equipment, and causing secondary explosions and fires. As much as 70% of a depleted uranium penetrator can be aerosolized when it strikes a tank. Aerosols containing uranium oxides may contaminate the area downwind. Uranium metal and oxide fragments may also contaminate the soil around the struck vehicle. Tests of depleted uranium penetrators striking depleted uranium armored vehicles have shown that most of the contamination will occur within 5 to 7 meters (16 to 23 feet) of the vehicle.

For Whom Is Exposure a Concern? Soldiers can be exposed to depleted uranium when intact rounds are in storage or uploaded in armored vehicles, or on the battlefield following the use of depleted uranium munitions. Studies have shown that exposure to intact depleted uranium weapons systems, both munitions and armor, pose very little risk from external radiation. Uranium and its decay products are primarily alpha emitters, emitting only very low levels of gamma radiation. However, individuals involved in handling or processing depleted uranium generally wear heavy gloves to protect against the high-energy beta particle from protactinium-234m, which is only a concern for skin exposure.

On the battlefield, exposure to depleted uranium can occur several ways. After impact with an armored vehicle, depleted uranium in the form of oxide and metal fragments will contaminate the struck vehicle and the surrounding area, especially if an explosion or fire occurs. Depleted uranium oxide can be aerosolized, presenting an inhalation hazard to soldiers in the vicinity or downwind of the struck vehicle. In addition, depleted uranium dust can be resuspended in the air from soil contamination around the vehicle. Soil contamination also can be ingested inadvertently from hand to mouth contact. In another example of battlefield exposure, at least 36 soldiers in Operation Desert Storm were reported to have wounds involving imbedded depleted uranium fragments. (Many of these fragments were not removed because the risk of

surgery was judged to be too great.) Following combat, maintenance and recovery personnel can be exposed to equipment contaminated with depleted uranium. Dust containing depleted uranium can be resuspended during recovery operations, posing an inhalation hazard. Spent depleted uranium rounds and penetrators that were fired but missed their targets may be scattered on the battlefield and can thus potentially lead to additional exposures.

What Happens to It in the Body? After it is ingested, most uranium is excreted from the body within a few days and never enters the bloodstream. The small fraction that is absorbed into the bloodstream (0.2 to 5%) is deposited preferentially in bone and kidneys. Most of what goes to the kidneys leaves within a few days (in urine), while that deposited in bone can remain for many years. After inhalation, generally only a small fraction penetrates to the lung's alveolar region, where it can remain for years and can also enter the bloodstream.

What Are the Primary Health Effects? Uranium is a health hazard only if it is taken into the body. External gamma exposure is generally not a major concern because uranium emits only a small amount of low-energy gamma radiation, and beta exposure is only of concern for direct handling operations (due to potential skin effects). The main means of exposure are ingestion of food and water containing uranium isotopes and inhalation of uranium-contaminated dust. Ingestion is usually the exposure route of concern unless there is a nearby source of airborne dust. Because uranium is taken up in the body much more readily if inhaled than if ingested, both exposure routes can be important. The major health concern is kidney damage caused by the chemical toxicity of soluble uranium compounds; these effects can be reversible depending on the level of exposure. Uranium is not considered a chemical carcinogen. A second concern is for uranium deposited in bone, which can lead to bone cancer as a result of the ionizing radiation associated with the radioactive decay products. Uranium has caused reproductive problems in laboratory animals and developmental problems in young animals, but it is not known if these problems exist for humans.

What Are the Risks? Lifetime cancer mortality risk coefficients have been calculated for nearly all radionuclides, including uranium *(see the companion fact sheet for Uranium).* On an activity (curie) basis, these risk coefficients are essentially the same for all three natural uranium isotopes. Hence, the risk is essentially independent of the ratio of the various isotopes in a uranium compound. For this reason, the risk of a fatal cancer from exposure to depleted uranium is essentially the same as for enriched uranium on an activity basis. However, because of the difference in specific activity, equal activities of enriched and depleted uranium have significantly different masses.

The decay of uranium isotopes in depleted uranium results in the generation of radioactive decay products. For example, thorium-234 and protactinium-234m (the "m" meaning metastable) are produced from the decay of uranium-238. The recycling of irradiated uranium may also result in some radioactive impurities in depleted uranium. Some of these decay products and impurities could potentially also exhibit chemical toxicity. The decay products and impurities contribute to the radiation emitted by depleted uranium; however, the concentrations are generally too small to be of significance when assessing the chemical health effects from exposures to depleted uranium.

The extent of chemical damage from exposure to a depleted uranium compound depends on its solubility and the route of exposure. In most assessments, only inhalation and ingestion are considered because although dermal absorption of some soluble compounds (e.g., uranyl nitrate) is possible, these exposures generally are not significant in association with industrial emissions or environmental exposures. When soluble or moderately soluble compounds such as uranyl fluoride (UO_2F_2) or uranium tetrafluoride (UF_4) are inhaled or ingested, some of the uranium enters the bloodstream and reaches the kidney and other internal organs; thus, chemical toxicity is of primary importance.

When insoluble compounds such as UO_2 and U_3O_8 are inhaled, the uranium is generally deposited in the lungs and can remain there for long periods of time (months or years). The main concern from exposure to these insoluble compounds is increased cancer risk from the internal exposure to radioactivity. Ingested insoluble compounds are poorly absorbed from the gastrointestinal tract, and so generally have low toxicity.

Available health effect criteria for acute and chronic uranium exposures are provided in Tables 1 and 2, respectively, derived from animal studies. Only limited data exist for human exposures.

TABLE 1. Reference Levels for Chemical Effects of Acute Uranium Exposures

Health Endpoint	Estimated Intake	Source	Air Concentration of Various Compounds [a]
Potential irreversible adverse effects	30 mg soluble uranium	NRC (1994) Fisher et al. (1994)	UO_2F_2, UF_4: 26 mg/m^3 U_3O_8: 590 mg/m^3 UO_2: 570 mg/m^3
Potential adverse effects	10 mg soluble uranium	McGuire (1991)	UO_2F_2, UF_4: 9 mg/m^3 U_3O_8: 200 mg/m^3 UO_2: 190 mg/m^3

[a] *Assumes 1-hour exposure at an inhalation rate of 1.5 m^3 per hour; assuming a higher inhalation rate for a shorter exposure duration may be appropriate (ICRP 1994) and will result in lower air concentrations. A factor of 25 decrease in systemic absorption of insoluble compounds (i.e., U_3O_8 and UO_2) was also assumed. These concentrations may not be protective against the adverse radiological effects of insoluble uranium compounds.*

TABLE 2. Reference Levels for Chemical Effects of Chronic Uranium Exposures

Route	Reference Level[a]	Source	Notes
Ingestion	Soluble compounds		
	(1) 0.003 mg/kg/d	EPA (2004); Maynard and Hodge (1949)	Based on a lowest observed adverse effect level (LOAEL) of 3 mg/kg-day and an uncertainty factor of 1,000.
	(2) 0.002 mg/kg/d	ATSDR, 1999; Gilman et al., 1998	Based on an intermediate-duration (91 days) oral exposure of rabbits to a soluble uranium compound.
	Insoluble compounds		
	(1) 0.003 mg/kg/d	EPA (2004); Maynard and Hodge (1949)	Same as for soluble compounds; no correction for decreased absorption.
	(2) Compound-specific based on absorption data	ATSDR, 1999; Gilman et al., 1998	
Inhalation	Soluble compounds		
	(1) 0.011 mg/m^3	Derived	Based on route-to-route extrapolation of ingestion value (EPA 1996).
	(2) 0.0012 mg/m^3	Derived	Based on adjustments to occupational exposure limit (EPA 1992).
	(3) 0.0004 or 0.0003 mg/m^3	ATSDR (1999)	Based on intermediate duration (5 weeks) and chronic (1 year) inhalation exposures of dogs to soluble uranium compounds.
	Insoluble compounds		
	(1) 0.011 mg/m^3	Derived	Same as for soluble compounds; no correction for decreased absorption.
	(2) 0.28 mg/m^3	Derived	Assume absorption is decreased by a factor of 25 (ICRP 1979).
	(3) 0.008 mg/m^3	ATSDR (1999)	Based on intermediate duration (5 weeks) inhalation exposures of dogs to insoluble uranium dioxide.
	(4) 0.006 mg/m^3	Derived	Based on adjustments made to occupational exposure limit (EPA 1992).

[a] *Soluble compounds include uranyl fluoride (UO_2F_2), uranyl nitrate hexahydrate ($UO_2[NO_3]_2 \cdot 6H_2O$), and uranium tetrachloride (UCl_4). Some insoluble compounds are uranium metal, uranium dioxide (UO_2), uranium trioxide (UO_3), and triuranium octaoxide (U_3O_8). Uranium hexafluoride (UF_6) decomposes to UO_2F_2 and hydrogen fluoride (HF) in the presence of moisture. Uranium tetrafluoride (UF_4) is very slightly soluble. Source: ATSDR (1999).*

Zirconium

Symbol:	**Zr**
Atomic Number: *(protons in nucleus)*	**40**
Atomic Weight: *(naturally occurring)*	**91**

What Is It? Zirconium is a lustrous, grayish-white, corrosion-resistant metal. When it exists in a finely divided form, zirconium can spontaneously ignite in air, especially at high temperatures; the solid metal is much more difficult to ignite. Zirconium occurs in nature as five stable isotopes. (Isotopes are different forms of an element that have the same number of protons in the nucleus but a different number of neutrons.) Zirconium-90 is the most prevalent form, comprising slightly more than half of natural zirconium. The other four stable isotopes and their relative abundances are zirconium-91 (11%), zirconium-92 (17%), zirconium-94 (17%), and zirconium-96 (2.8%). Zirconium occurs widely in the earth's crust, but not in concentrated deposits.

Of the six major radioactive zirconium isotopes, only one – zirconium-93 – has a half-life long enough to warrant potential concern at Department of Energy (DOE) environmental management sites such as Hanford. The half-lives of all other isotopes are less than three months. Zirconium-93 decays by emitting a beta particle with a half-life of 1.5 million years to niobium-93m (the "m" means metastable), which in turn decays by isomeric transition with a half-life of 14 years. Zirconium-93 is present in spent nuclear fuel and the wastes resulting from reprocessing this fuel. The low-specific activity and low energy of its radiations limit the radioactive hazards of this isotope.

Radioactive Properties of the Key Zirconium Isotope and an Associated Radionuclide

Isotope	Half-Life *(yr)*	Specific Activity *(Ci/g)*	Decay Mode	Radiation Energy *(MeV)*		
				Alpha *(α)*	Beta *(β)*	Gamma *(γ)*
Zr-93	1.5 million	0.0025	β	-	0.020	-
Nb-93m	*14*	*290*	*IT*	-	*0.028*	*0.0019*

IT =isomeric transition, Ci = curie, g = gram, and MeV = million electron volts; a dash means the entry is not applicable. (See the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients for an explanation of terms and interpretation of radiation energies.) Certain properties of niobium-93m are included here because this radionuclide can accompany zirconium-93 decays. Values are given to two significant figures.

Where Does It Come From? Zirconium is naturally present in a number of minerals, but it does not generally occur in concentrated deposits. The mineral zircon (zirconium orthosilicate), which is found in alluvial deposits in streambeds, ocean beaches, or old lake beds, is the only commercial source of zirconium. Commercial grade zirconium contains from 1 to 3% hafnium. The leading producers of zirconium are Australia and South Africa. Other producers include Ukraine, the United States, India, Brazil, and China. Annual worldwide production of zirconium is currently about 900,000 metric tons (MT) of which about 60 MT is produced domestically.

Zirconium-93 is produced by neutron activation of components in nuclear reactors and by nuclear fission. When a fissile nuclide such as an atom of uranium-235 fissions, it generally splits asymmetrically into two large fragments – fission products with mass numbers in the range of about 90 and 140 – and two or three neutrons. (The mass number is the sum of the number of protons and neutrons in the nucleus of the atom.) These neutrons can cause additional fissions (producing a chain reaction), escape from the reactor, or irradiate nearby materials. Zirconium is used in the cladding of nuclear fuel elements, and neutron irradiation of the cladding can produce zirconium-93. Zirconium-93 is also a fission product with a relatively high yield of about 6%. That is, about six atoms of zirconium-93 are produced per 100 fissions. Zirconium-93 is present in spent nuclear fuel, high-level radioactive wastes resulting from processing spent nuclear fuel, and radioactive wastes associated with nuclear reactors and fuel reprocessing plants.

How Is It Used? Zirconium is very resistant to corrosion that occurs in the presence of many common acids and alkalis as well as seawater. For this reason it is used extensively by the chemical industry

where corrosive agents are employed. Zirconium is used in vacuum tubes, as an alloying agent in steel, in surgical appliances, photoflash bulbs, explosive primers, and lamp filaments. It is also used in poison ivy lotions in the form of the carbonate. The mineral zircon has a high index of refraction and is used as a gem material. The impure oxide zirconia is used for laboratory crucibles that can withstand heat shock, for the lining of metallurgical furnaces, and by the glass and ceramic industries as a refractory material. Zircaloy, an alloy of zirconium containing small amounts of tin, iron, chromium, and nickel, is used as a cladding material for nuclear fuel elements. Zirconium has a low absorption cross-section for neutrons, which makes it an ideal material for use in nuclear reactor applications. Its use in commercial nuclear power generation now accounts for as much as 90% of the zirconium metal that is produced.

What's in the Environment? Zirconium is present in the earth's crust at a concentration of about 130 milligrams per kilogram (mg/kg), and its concentration in seawater is about 0.026 micrograms (µg)/liter. Trace amounts of zirconium-93 are present in soil around the globe from radioactive fallout. It can also be present at certain nuclear facilities, such as reactors and spent fuel reprocessing plants. Zirconium is generally one of the less mobile radioactive metals in soil, although certain forms can move downward some distance to underlying layers with percolating water. Zirconium preferentially adheres quite well to soil, and the concentration associated with sandy soil particles is typically about 600 times higher than in interstitial water (water in the pore space between soil particles), with higher concentration ratios (over 2,000) in loam and clay soils. Thus, zirconium is generally not a major contaminant in groundwater at DOE sites.

What Happens to It in the Body? Zirconium can be taken into the body by eating food, drinking water, or breathing air. Gastrointestinal absorption from food or water is the principal source of internally deposited zirconium in the general population. Zirconium is not well absorbed into the body, with only about 0.2% of the amount ingested being absorbed into the bloodstream through the intestines. Of the zirconium that reaches the blood, half deposits in the skeleton with a biological half-life of 8,000 days and the other half deposits in all other organs and tissues of the body where it is retained with a biological half-life of 7 days (per simplified models that do not reflect intermediate redistribution). Since zirconium is not a major constituent of mineral bone, the amount deposited in the skeleton is assumed to remain on the bone surfaces and not be absorbed into the volume of bone.

What Are the Primary Health Effects? Zirconium is a health hazard only if it is taken into the body. External gamma exposure is not a concern because zirconium-93 decays by emitting a beta particle with no gamma radiation, and niobium-93m decays by isomeric transition, in which only low-energy gamma radiation is emitted. While inside the body, zirconium presents a health hazard from the beta particles and gamma radiation, and the main concern is associated with the increased likelihood of inducing cancer.

What Is the Risk? Lifetime cancer mortality risk coefficients have been calculated for nearly all radionuclides, including zirconium *(see box at right)*. While the coefficients for ingestion are somewhat lower than for inhalation, ingestion is generally the most common means of entry into the body. The risks shown here include the contribution from niobium-93m, even though it may not be in radioactive equilibrium with zirconium-93 (about ten niobium half-lives or 140 years are needed to establish equilibrium). Similar to other radionuclides, the risk coefficients for tap water are about 75% of those for dietary ingestion.

Radiological Risk Coefficients

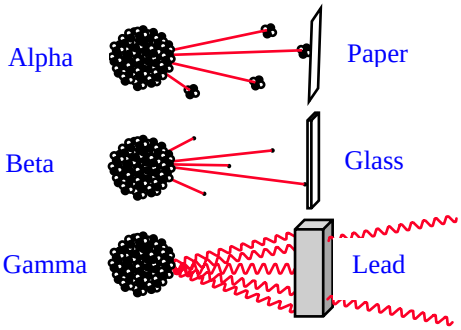
This table provides selected risk coefficients for inhalation and ingestion. Recommended default absorption types were used for inhalation, and dietary values were used for ingestion. These values include the contribution from niobium-93m. Risks are for lifetime cancer mortality per unit intake (picocurie, pCi), averaged over all ages and both genders (10^{-12} is a trillionth). Other values, including for morbidity, are also available.

Isotope	Lifetime Cancer Mortality Risk	
	Inhalation (pCi^{-1})	Ingestion (pCi^{-1})
Zirconium-93	8.4×10^{-12}	1.7×10^{-12}

For more information, see the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients and the accompanying Table 1.

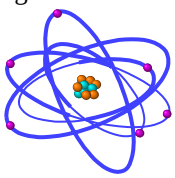
Ionizing Radiation

What Is It? Ionizing radiation is energy that is carried by any of several types of particles and rays (electromagnetic radiation) given off by radioactive material, X-ray machines, and nuclear reactions. This energy can knock electrons out of molecules with which they interact, thus creating ions. Non-ionizing radiation, such as that emitted by a laser, is different because it does not create ions when it interacts with matter but dissipates energy generally in the form of heat. The three main types of ionizing radiation are alpha particles, beta particles, and gamma rays.



An alpha particle consists of two protons and two neutrons and is identical to the nucleus of a helium atom. Because of its relatively large mass and charge, an alpha particle produces ions in a very localized area. An alpha particle loses some of its energy each time it produces an ion (its positive charge pulls electrons away from atoms in its path), finally acquiring two electrons from an atom at the end of its path to become a complete helium atom. An alpha particle has a short range (several centimeters) in air and cannot penetrate the outer layer of skin.

Beta particles can be either negative (negatron) or positive (positron). Negatrons are identical to electrons and originate in the nucleus of an atom that undergoes radioactive decay by changing a neutron into a proton. The only difference between a negative beta particle (negatron) and an electron is the ancestry. A beta particle originates in the nucleus whereas an electron is external to the nucleus. Unless otherwise specified, the term "beta particle" generally refers to a negatron. A positron is emitted from an atom that decays by changing a proton into a neutron. Beta particles are smaller and more penetrating than alpha particles, but their range in tissue is still quite limited. When its energy is spent, a negatron attaches itself to an atom and becomes an ordinary electron, while a positron collides with an ambient electron and the two particles annihilate each other, producing two gamma rays. When a negatron passes close to the nucleus of an atom, the strong attractive Coulomb force causes the beta particle to deviate sharply and lose energy at a rate proportional to the square of the acceleration. This energy manifests itself as photons termed Bremsstrahlung. The amount of beta energy converted into photons is directly proportional to the energy of the beta particle. This effect is only significant for high-energy beta particles generally passing through very dense materials such as lead, i.e., those with higher atomic numbers and so more protons in the nucleus.



Gamma rays are electromagnetic radiation given off by an atom as a means of releasing excess energy. They are bundles (quanta) of energy that have no charge or mass and can travel long distances through air (up to several hundred meters), body tissue, and other materials. A gamma ray can pass through a body without hitting anything, or it may hit an atom and give that atom all or part of its energy. This normally knocks an electron out of the atom, ionizing it. This electron then uses the energy it receives from the gamma ray to create additional ions by knocking electrons out of other atoms. Because a gamma ray is pure energy, it no longer exists once it loses all its energy. The capability of a gamma ray to do damage is a function of its energy, where the distance between ionizing events is large on the scale of the nucleus of a cell.

Additional forms of ionizing radiation beyond the three types shown in the figure above include neutrons, protons, neutrinos, muons, pions, heavy charged particles, X-rays and others. Essentially all radioactive materials at the Hanford Site originated from neutron interactions with uranium fuel to produce plutonium. Byproducts of this process include fission products (most of which are in the high-level waste currently in on-site storage), activation products in the containment and reactor coolant materials, and various radioactive wastes. However, the radioactive hazards that remain at the Hanford Site are largely those associated with the three general types of radiation shown above, so the discussion here is limited to these three.

Where Does It Come From? All organisms are being exposed to ionizing radiation from natural sources all the time. Radiation doses are typically given in units of rem – an acronym for Roentgen equivalent man – or millirem (mrem), which is one one-thousandth of a rem. This unit was developed to allow for the consistent reporting of hazards associated with the various types and energies of radiation on the human body. The rem is the product of the absorbed dose in rads (i.e., the amount of energy imparted to tissue by the radiation, where 1 rad equals 0.01 joules/kg) and factors for the relative biological effectiveness (RBE) of the radiation. The RBE is directly related to the linear energy transfer (LET) or distance over which the radiation energy is imparted to the absorbing medium and is accounted for by a quality factor. For example, alpha particles are 20 times more hazardous than beta particles for the same energy deposition and hence have a quality factor of 20, whereas the quality factor for beta particles is one. The International Commission on Radiological Protection (ICRP) has developed a methodology for reporting the effective dose equivalent. This is the product of the dose (in rem or mrem) to individual tissues and the tissue-specific weighting factors (fractional values less than one) that indicate the relative risk of cancer induction or hereditary defects from irradiation of that tissue, summed over all relevant tissues. By use of the effective dose equivalent, it is possible to compare the relative radiation hazards from various types of radiation that impact different organs of the body. The doses discussed below are effective dose equivalents.

More than 80% of the total dose received by individuals in the United States comes from natural sources; 55% comes from radon gas decay products, 8% from cosmic radiation, 8% from terrestrial radiation, and 11% from internal sources such as potassium-40. The rest comes from man-made sources that include medical X-rays (11%), nuclear medicine (4%), consumer products (3%), radioactive fallout, and nuclear power plants (<1%). These percentages are representative, and not all people are exposed to the same sources to the same degree. The National Council on Radiation Protection and Measurements (NCRP) estimates an average radiation dose of 360 mrem/yr from natural and man-made sources, with about 300 mrem/yr from natural sources and 60 mrem/yr from man-made sources (mostly for medical procedures). For reference, a person receives a dose of about 5 mrem on a flight from New York City to Los Angeles, and a typical chest X-ray produces a dose of about 10 mrem.

How Is It Used? Radioactive materials and other sources of ionizing radiation are widely used to diagnose and treat diseases in human and veterinary medicine. Medical and dental X-rays are used to detect problems such as broken bones and dental cavities. Sealed radiation sources are used to deliver very high, localized radiation doses to treat certain types of cancers. Ionizing radiation also has a number of industrial and commercial uses. Radioactive sources are used in consumer products such as smoke detectors and to sterilize food products. Ionizing radiation is also used to test materials, inspect welds, generate heat and electricity for space travel, determine soil moisture content, and track the movement of various elements in the environment and human body (through use of radioactive tracers). Additional uses continue to be identified.

What's in the Environment? Exposure to background radiation and naturally occurring radioactive materials results in an annual dose of about 300 mrem/yr. Of this total, about one-third is due to external ionizing radiation, the main contributors being cosmic rays and terrestrial gamma rays (29 mrem/yr each), and radionuclides within the body (40 mrem/yr). Cosmic rays are produced when subatomic particles originating outside the solar system interact with particles in the upper atmosphere to produce gamma rays, neutrons and leptons that can reach and penetrate the earth's surface. It is these secondary particles and rays that produce the dose from cosmic radiation. Naturally occurring radioactive elements such as uranium, thorium and radium are present in soil, rock, water, and all other environmental media, and certain of these radionuclides (and their radioactive decay products) give off gamma rays as they undergo radioactive decay. The principal contributor to the dose from radionuclides in the body is potassium-40, which decays by emitting an energetic beta particle and gamma rays. About two-thirds of the background radiation dose (of 300 mrem/yr) is due to intake of radionuclides into the body. The largest contributor is inhalation of radon-220 and radon-222 gases and their short-lived radioactive decay products, which are charged particles that readily attach to airborne dust particles. In fact, inhalation of radon gas contributes nearly all of the 200 mrem/yr attributable to radionuclide intake, and most is due to the short-lived radon-222 decay products polonium-218 and polonium-214. Ingestion of food and water containing naturally occurring radionuclides accounts for only a few mrem/yr.

What Are the Primary Health Effects? High doses of ionizing radiation can lead to effects such as skin burns, hair loss, birth defects, illness, cancer, and death, depending on the dose and the period of time over which it is received. Acute doses (such as from a serious accident involving nuclear materials) can result in damage to the blood-forming organs, gastrointestinal tract, and central nervous system. Very high doses, e.g., on the order of 500 rem (or 500,000 mrem) or more, can cause death in many (but not all) individuals, depending on the degree of medical intervention. The main health concern associated with radiation exposure is the induction of various cancers. Additional effects may include genetic mutations (although none have been observed in humans) and teratogenic effects such as mental retardation. The U.S. Environmental Protection Agency (EPA) has indicated that for radioactively contaminated Superfund sites, the risk from cancer is limiting and should be used as the sole basis for assessing radiation-related human health risks. The EPA noted that on average, about 50% of all cancers that can be induced by radiation (they can also be caused by other agents) are fatal, ranging from about 10% for thyroid cancer to 100% for liver cancer. Other EPA estimates for specific isotopes indicate the average may be higher, e.g., 60 to 70% or more.

What Happens to It in the Body? Radioactive materials can enter the body by inhalation, ingestion, or dermal absorption. In addition, gamma radiation external to the body can penetrate the skin and produce a dose in various tissues. Inhalation is the primary exposure mode for gaseous radionuclides (such as radon), and particulates near the source of an airborne release. A fractional amount of inhaled radionuclides is transferred from the lungs to the blood, where it distributes to other organs. The extent of absorption is strongly dependent on the radionuclide and its chemical form. Ingestion is the primary uptake mode for radionuclides in soil, water, and food, including those naturally occurring (such as radium and uranium in soil and groundwater) and man-made (such as plutonium from radioactive fallout). A fractional amount of ingested radionuclides is absorbed from the gastrointestinal tract into the blood while the rest clears the body through normal biological processes via urine and feces. As with inhalation, the extent of uptake depends on the radionuclide and its chemical form. The skin is generally an effective barrier against absorption of radionuclides, so dermal absorption is a very minor route of exposure. An exception to this is dermal absorption of tritiated water, i.e., water containing some amount of tritium (hydrogen-3) in place of a normal hydrogen atom in the water molecule, which is absorbed through the skin in the same manner as ordinary water.

What Is the Risk? While the EPA has developed lifetime cancer mortality risk coefficients for nearly all radionuclides, the agency has not developed a risk coefficient for ionizing radiation as a general category. A nominal mortality value of 5×10^{-7} incremental cancer risk per mrem has been identified for low-LET radiation delivered at a low dose and dose rate.

Natural Decay Series: Uranium, Radium, and Thorium

Uranium, radium, and thorium occur in three natural decay series, headed by uranium-238, thorium-232, and uranium-235, respectively. In nature, the radionuclides in these three series are approximately in a state of secular equilibrium, in which the activities of all radionuclides within each series are nearly equal.

Two conditions are necessary for secular equilibrium. First, the parent radionuclide must have a half-life much longer than that of any other radionuclide in the series. Second, a sufficiently long period of time must have elapsed, for example ten half-lives of the decay product having the longest half-life, to allow for ingrowth of the decay products *(see the companion fact sheet on Ionizing Radiation)*. Under secular equilibrium, the activity of the parent radionuclide undergoes no appreciable changes during many half-lives of its decay products.

The radionuclides of the uranium-238, thorium-232, and uranium-235 decay series are shown in Figures N.1, N.2, and N.3, along with the major mode of radioactive decay for each. Radioactive decay occurs when an unstable (radioactive) isotope transforms to a more stable isotope, generally by emitting a subatomic particle such as an alpha or beta particle. Radionuclides that give rise to alpha and beta particles are shown in these figures, as are those that emit significant gamma radiation.

Gamma radiation is not a mode of radioactive decay (such as alpha and beta decay). Rather, it is a mechanism by which excess energy is emitted from certain radionuclides, i.e., as highly energetic electromagnetic radiation emitted from the nucleus of the atom. For simplicity, only significant gamma emissions associated with the major decay modes are shown in Figures N.1 through N.3; that is, radionuclides listed are those for which the radiation dose associated with gamma rays may pose a health concern. The gamma component is not shown for those radionuclides whose gamma emissions do not generally represent a concern.

Of the two conditions noted above for secular equilibrium, the first is generally met for the uranium-238, thorium-232 and uranium-235 decay series in naturally occurring ores. While the second condition may not be met for all ores or other deposits of uranium and thorium (given the extremely long half-lives for the radionuclides involved and the geological changes that occur over similar time scales), it is reasonable to assume secular equilibrium for naturally occurring ores to estimate the concentrations of the various daughter radionuclides that accompany the parent. The state of secular equilibrium in natural uranium and thorium ores is significantly altered when they are processed to extract specific radionuclides.

After processing, radionuclides with half-lives less than one year will reestablish equilibrium conditions with their longer-lived parent radionuclides within several years. For this reason, at processing sites what was once a single, long decay series (for example the series for uranium-238) may be present as several smaller decay series headed by the longer-lived decay products of the original series (that is, headed by uranium-238, uranium-234, thorium-230, radium-226, and lead-210 in the case of uranium-238). Each of these sub-series can be considered to represent a new, separate decay series. Understanding the physical and chemical processes associated with materials containing uranium, thorium, and radium is important when addressing associated radiological risks.

In the fact sheets developed for uranium, radium, and thorium, the contributions of radionuclides having half-lives less than one year were included in the risk coefficients. (Each fact sheet identifies which radionuclides are included in these coefficients.) In some situations, it may be necessary to add the radiological risk identified for a given radionuclide to that of its parent radionuclide to properly represent the total risk. For example, the radiological risk for thorium-232 is comprised of the risk for thorium-232 plus the risk for radium-228. Decay series information should be used together with the information in these fact sheets to ensure that the radiological risks associated with uranium, radium, and thorium are properly estimated and represented.

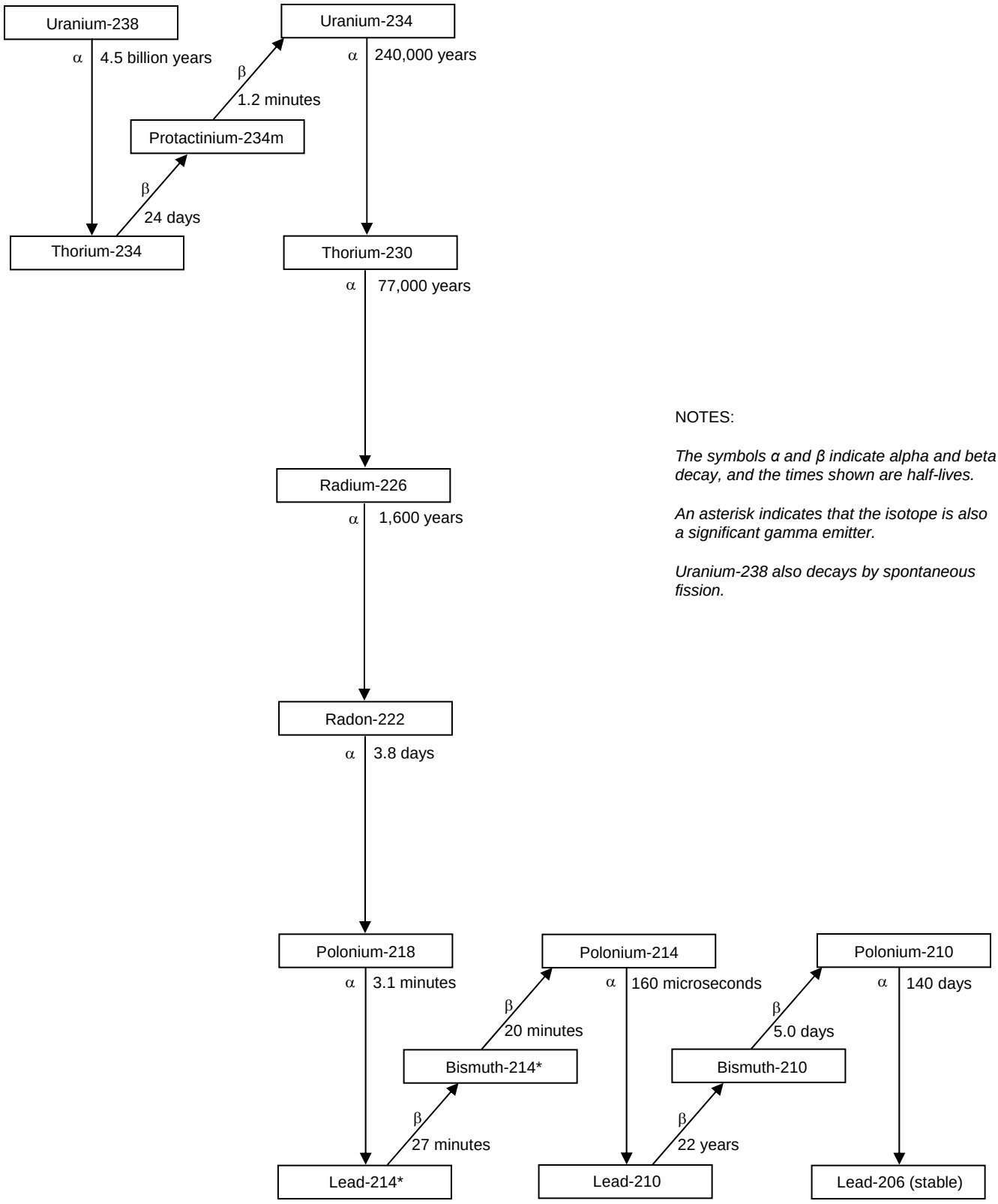


FIGURE N.1 Natural Decay Series: Uranium-238

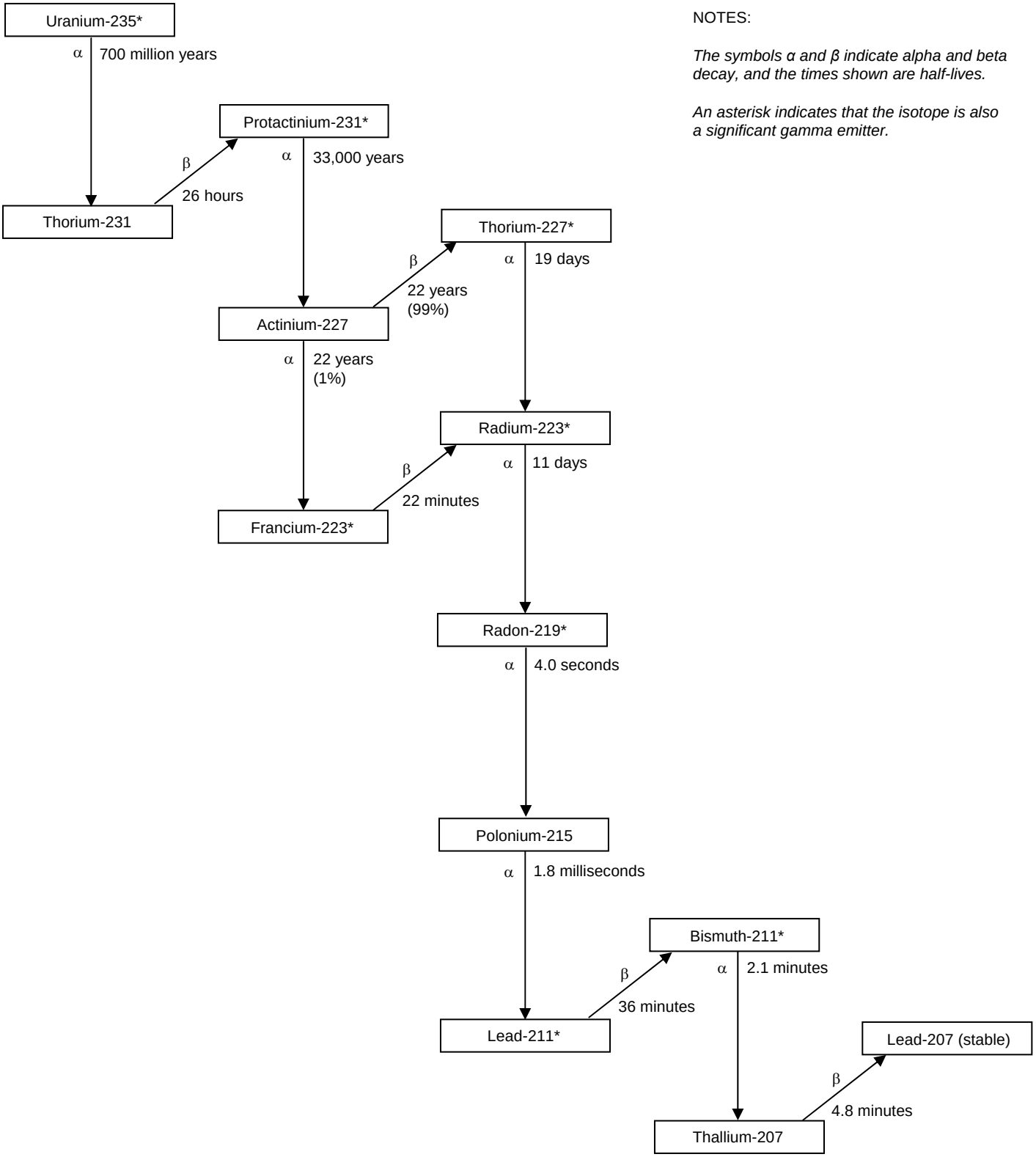


FIGURE N.2 Natural Decay Series: Uranium-235

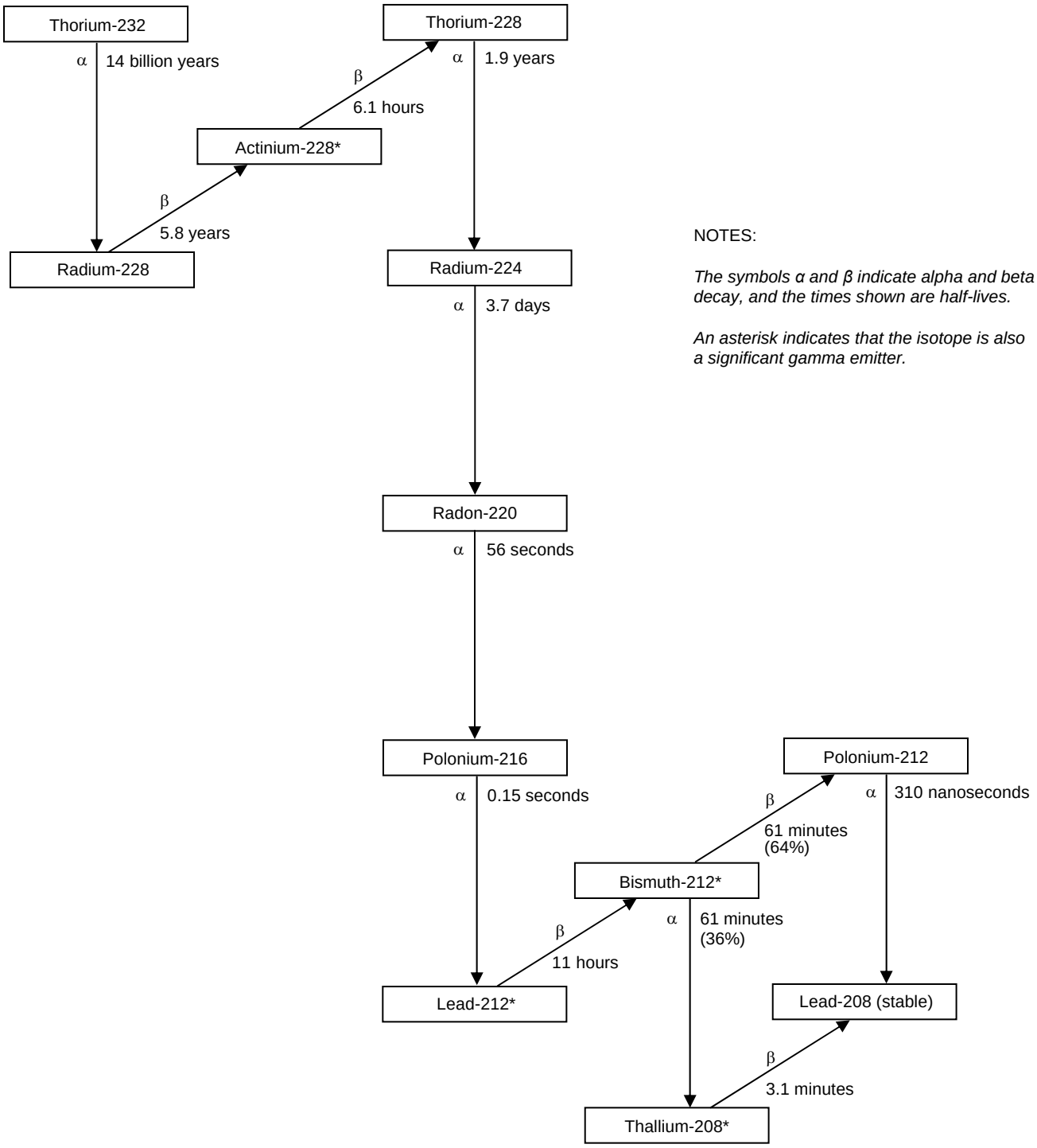


FIGURE N.3 Natural Decay Series: Thorium-232

Transuranic Radionuclides

What Are They? Taking their name from being *trans*- or beyond uranium, transuranic radionuclides have atomic numbers greater than that of uranium, which is 92. All transuranic isotopes are radioactive. (Isotopes are different forms of an element that have the same number of protons in the nucleus but a different number of neutrons.) Those associated with americium, californium, curium, neptunium, and plutonium are generally of concern for U.S. Department of Energy (DOE) environmental management sites such as Hanford. More information on these five radionuclides and their decay products is presented in the separate radionuclide-specific fact sheets. An additional transuranic radionuclide that can be found at DOE sites is berkelium-247; summary radioactive properties for this transuranic isotope is given in the green box on the back of this page.

Where Do They Come From? Most transuranic radionuclides at DOE sites were produced in nuclear reactors by neutron capture, with the remainder being produced in particle accelerators. Because multiple neutron capture reactions are usually needed to produce higher-numbered isotopes for a given element, the relative number of individual isotopes decreases with increasing atomic number. For example, after nuclear fuel is discharged from a plutonium production reactor, more plutonium-238 and plutonium-239 would generally be present than the higher-numbered isotopes such as plutonium-242. Transuranic radionuclides typically do not occur naturally in the environment, although very minute amounts can be present with some uranium ores. The underground, natural sustained nuclear reactions estimated to have occurred about 1.9 billion years ago in Gabon, Africa, also produced some transuranic radionuclides, including neptunium and plutonium.

Like certain other radioactive isotopes such as those of naturally occurring uranium, radium, and thorium, the transuranic radionuclides undergo radioactive decay to create typically long chains of decay products. Radioactive decay occurs when an unstable atom transforms to a more stable isotope by emitting an alpha or beta particle, although some isotopes also decay by spontaneous fission. During spontaneous fission, the atom self-disintegrates into two smaller atoms accompanied by a release of energy. The radioactive decay series for several key transuranic radionuclides are shown in Figures T.1 through T.3 at the end of this fact sheet. These figures illustrate the relationships among isotopes in terms of radioactive decay and ingrowth. They also identify the major decay modes and show which isotopes emit significant gamma radiation. A number of these transuranic decay series eventually transition into the decay series for the naturally occurring radionuclides, which are shown separately in Figures N.1 through N.3 within the companion fact sheet: *Natural Decay Series: Uranium, Radium, and Thorium*. Many concepts relevant to decay series, such as secular equilibrium, are discussed in that companion fact sheet and are therefore not repeated here. Interested readers are encouraged to refer to that sheet for more information.

What Are Transuranic Wastes? Radioactive wastes containing more than 100 nanocuries per gram (nCi/g) of alpha-emitting transuranic radionuclides with half-lives greater than 20 years are termed "transuranic wastes." (100 nCi/g is one tenth of a microcurie or one ten-millionth of a curie per gram.) This term technically applies to radioactive wastes generated after 1970. Prior to 1970, no specific considerations were identified for radioactive wastes containing transuranic radionuclides. At that time, these wastes were managed in the same manner as other low-level radioactive wastes regardless of the concentration of transuranic radionuclides, and all these wastes were generally disposed of by shallow land burial. Today, transuranic wastes are segregated from other low-level radioactive wastes and managed separately because the hazards associated with the higher concentrations of long-lived alpha-emitting radionuclides are believed to warrant more stringent handling and disposal considerations.

Radioactive wastes that would meet today's definition of transuranic wastes but were disposed of before 1970 by shallow land burial and other similar techniques at DOE sites are often referred to as "buried transuranic wastes." The DOE is currently evaluating the most appropriate manner to manage these wastes over the long term on a site-specific basis. Transuranic (or TRU) wastes generated by more recent defense-related activities, including a substantial inventory in controlled storage, are currently being sent to the Waste Isolation Pilot Plant located near Carlsbad, New Mexico, for deep underground disposal. This facility began accepting wastes in 1999 and has a permitted capacity of about 175,600 m^3.

The following table summarizes key properties of selected transuranic radionuclides. (For americium, californium, curium, neptunium, and plutonium, this information is also given in the radionuclide-specific fact sheets and the supporting Table 2.) The table below includes radionuclides beyond those that define transuranic wastes, i.e., those with half-lives <20 years or that decay by means other than alpha emission. (These are indicated by regular green shading, while those that define TRU wastes are in yellow-green.)

Radioactive Properties of Selected Transuranic Radionuclides

Isotope	Half-Life	Specific Activity *(Ci/g)*	Decay Mode	Radiation Energy *(MeV)*		
				Alpha *(α)*	Beta *(β)*	Gamma *(γ)*
Americium-241	430 yr	3.5	α	5.5	0.052	0.033
Americium-242m	150 yr	9.8	IT	0.025	0.044	0.0051
Americium-242	*16 hr*	*820,000*	*β, EC*	-	*0.18*	*0.018*
Americium-243	7,400 yr	0.20	α	5.3	0.022	0.056
Neptunium-239	*2.4 days*	*230,000*	*β*	-	*0.26*	*0.17*
Berkelium-247	1,400 yr	1.1	α	5.6	0.061	0.11
Californium-248	330 days	1,600	α	6.3	0.0060	0.0013
Californium-249	350 yr	4.1	α	5.8	0.043	0.33
Californium-250	13 yr	110	α	6.0	0.0057	0.0012
Californium-251	900 yr	1.6	α	5.8	0.020	0.13
Californium-252	2.6 yr	540	α	5.9	0.0056	0.0012
Curium-242	160 days	3,400	α	6.1	0.010	0.0018
Curium-243	29 yr	52	α	5.8	0.14	0.13
Curium-244	18 yr	82	α	5.8	0.086	0.0017
Curium-245	8,500 yr	0.17	α	5.4	0.065	0.096
Curium-246	4,700 yr	0.31	α	5.4	0.0080	0.0015
Curium-247	16 million yr	0.000094	α	4.9	0.021	0.32
Curium-248	340,000 yr	0.0043	α	4.7	0.0060	0.0012
Curium-250	6,900 yr	0.21	α, β	1.3	0.0016	-
Plutonium-246 (25%)	*11 days*	*49,000*	*β*	-	*0.13*	*0.14*
Berkelium-250 (14%)	*3.2 hr*	*3.9 million*	*β*	-	*0.29*	*0.89*
Americium-246 (25%)	*39 min*	*20 million*	*β*	-	*0.66*	*0.70*
Neptunium-235	1.1 yr	1,400	EC	<0.001	0.010	0.0071
Neptunium-236	120,000 yr	0.013	β, EC	-	0.21	0.14
Plutonium-236 (9%)	*2.9 yr*	*540*	*α*	*5.8*	*0.013*	*0.0021*
Neptunium-237	2.1 million yr	0.00071	α	4.8	0.070	0.035
Protactinium-233	*27 days*	*21,000*	*β*	-	*0.20*	*0.20*
Plutonium-238	88 yr	17	α	5.5	0.011	0.0018
Plutonium-239	24,000 yr	0.063	α	5.1	0.0067	<0.001
Plutonium-240	6,500 yr	0.23	α	5.2	0.011	0.0017
Plutonium-241	14 yr	100	β	<0.001	0.0052	<0.001
Plutonium-242	380,000 yr	0.0040	α	4.9	0.0087	0.0014
Plutonium-244	83 million yr	0.000018	α	4.6	0.0071	0.0012

Decay products are in italics. Values are given to two significant figures. EC = electron capture, IT = isomeric transition, Ci = curie, g = gram, and MeV = million electron volts. A dash indicates that the entry is not applicable. (See radionuclide-specific fact sheets and the companion fact sheet on Radioactive Properties, Internal Distribution, and Risk Coefficients for further information, including on terminology and interpretation of radiation energies.)

What Are the Health Risks? Transuranic wastes pose a concern for environmental management because of the relatively long half-lives of the transuranic radionuclides, combined with their emission of alpha particles (and to a lesser degree beta particles). Because the alpha particles cannot penetrate the outer layer of skin, these radionuclides pose a hazard primarily if they are taken into the body. Some isotopes also emit gamma radiation, which results in an additional hazard from external exposure. The main concern is cancer induction due to energy deposited in tissues during radioactive decay. Lifetime cancer mortality risk coefficients have been calculated for nearly all radionuclides, including transuranic

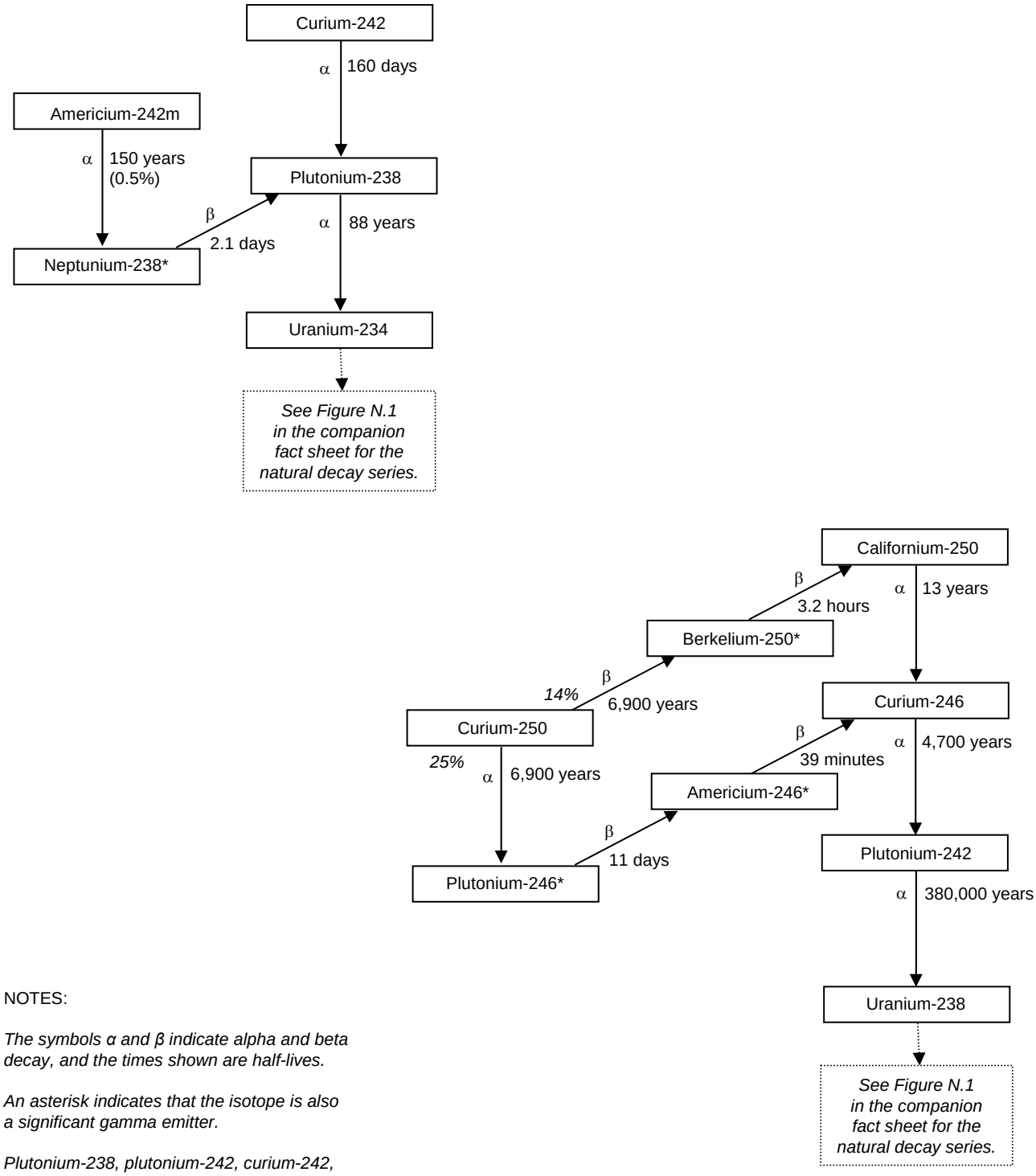
isotopes. Selected radiological risk coefficients are summarized in the yellow box below. This summary includes information presented on the radionuclide-specific fact sheets for americium, californium, curium, neptunium, and plutonium and the supporting Table 1. Information for berkelium-247 is taken from the same source reference used for that table. Shown below are selected risk coefficients of various transuranic radionuclides for inhalation and dietary ingestion, and for external gamma irradiation where that entry is appropriate. The mortality risk represents the lifetime risk of incurring a fatal cancer, and the morbidity risk represents the risk of incurring both fatal and non-fatal cancers.

Mortality and Morbidity Risk Coefficients for Selected Transuranic Radionuclides

Isotope	Lifetime Cancer Risk					
	Mortality			Morbidity		
	Inhalation	Ingestion	External	Inhalation	Ingestion	External
Americium-241	2.4×10^{-8}	9.5×10^{-11}	1.9×10^{-8}	2.8×10^{-8}	1.3×10^{-10}	2.8×10^{-8}
Americium-242m	1.3×10^{-8}	6.8×10^{-11}	-	1.6×10^{-8}	9.0×10^{-11}	-
Americium-243	2.3×10^{-8}	9.8×10^{-11}	4.3×10^{-7}	2.7×10^{-8}	1.4×10^{-10}	6.4×10^{-7}
Berkelium-247	4.0×10^{-8}	1.2×10^{-10}	2.1×10^{-7}	4.8×10^{-8}	1.6×10^{-10}	3.1×10^{-7}
Californium-248	2.4×10^{-8}	3.8×10^{-11}	-	2.6×10^{-8}	6.2×10^{-11}	-
Californium-249	4.0×10^{-8}	1.2×10^{-10}	9.3×10^{-7}	4.8×10^{-8}	1.6×10^{-10}	1.4×10^{-6}
Californium-250	3.5×10^{-8}	8.0×10^{-11}	-	3.7×10^{-8}	1.1×10^{-10}	-
Californium-251	4.1×10^{-8}	1.3×10^{-10}	2.6×10^{-7}	4.9×10^{-8}	1.7×10^{-10}	3.8×10^{-7}
Californium-252	2.1×10^{-8}	4.1×10^{-11}	-	2.2×10^{-8}	5.7×10^{-11}	-
Curium-242	1.4×10^{-8}	3.2×10^{-11}	-	1.5×10^{-8}	5.5×10^{-11}	-
Curium-243	2.4×10^{-8}	8.5×10^{-11}	2.9×10^{-7}	2.7×10^{-8}	1.2×10^{-10}	4.2×10^{-7}
Curium-244	2.3×10^{-8}	7.5×10^{-11}	-	2.5×10^{-8}	1.1×10^{-10}	-
Curium-245	2.4×10^{-8}	9.5×10^{-11}	1.6×10^{-7}	2.8×10^{-8}	1.3×10^{-10}	2.4×10^{-7}
Curium-246	2.4×10^{-8}	9.3×10^{-11}	-	2.8×10^{-8}	1.3×10^{-10}	-
Curium-247	2.2×10^{-8}	9.0×10^{-11}	8.9×10^{-7}	2.5×10^{-8}	1.3×10^{-11}	1.3×10^{-6}
Curium-248	8.8×10^{-8}	3.4×10^{-10}	-	1.0×10^{-7}	4.8×10^{-10}	-
Curium-250	5.0×10^{-7}	2.0×10^{-9}	9.7×10^{-7}	5.8×10^{-7}	2.8×10^{-9}	1.4×10^{-6}
Neptunium-235	1.0×10^{-12}	2.8×10^{-13}	-	1.2×10^{-12}	5.1×10^{-13}	-
Neptunium-236	2.6×10^{-9}	1.5×10^{-11}	2.2×10^{-7}	3.0×10^{-9}	2.3×10^{-11}	3.2×10^{-7}
Neptunium-237	1.5×10^{-8}	5.8×10^{-11}	5.4×10^{-7}	1.8×10^{-8}	9.1×10^{-11}	8.0×10^{-7}
Plutonium-238	3.0×10^{-8}	1.3×10^{-10}	-	3.4×10^{-8}	1.7×10^{-10}	-
Plutonium-239	2.9×10^{-8}	1.3×10^{-10}	-	3.3×10^{-8}	1.7×10^{-10}	-
Plutonium-240	2.9×10^{-8}	1.3×10^{-10}	-	3.3×10^{-8}	1.7×10^{-10}	-
Plutonium-241	2.8×10^{-10}	1.9×10^{-12}	-	3.3×10^{-10}	2.3×10^{-12}	-
Plutonium-242	2.8×10^{-8}	1.3×10^{-10}	-	3.1×10^{-8}	1.7×10^{-10}	-
Plutonium-244	2.7×10^{-8}	1.3×10^{-10}	-	3.1×10^{-8}	1.6×10^{-10}	-

Source: *Cancer Risk Coefficients for Environmental Exposure to Radionuclides*, Federal Guidance Report 13, U.S. Environmental Protection Agency, EPA 402-R-99-001, September 1999. Values are averaged over all ages and both genders and include the contributions from short-lived decay products, i.e., those with half-lives less than one year. (For context, 10^{-9} is a billionth, 10^{-12} is a trillionth, and a pCi is a picocurie, or a trillionth of a curie.) To convert to standard international units, multiply by 27 pCi per becquerel (Bq). For ingestion and inhalation, units are risk per pCi. For inhalation, the values corresponding to the recommended default absorption type for particulates are shown; the maximum value is given if no absorption type was recommended. For ingestion, the dietary values shown are the highest for ingestion exposures; values for tap water ingestion are typically 70 to 80% of those for diet. For external exposure, risk coefficients are given for those radionuclides with gamma-ray energies >0.03 MeV per decay, accounting for the fraction of time that the radioactive decay results in the emission of gamma rays. A dash indicates the radionuclide or its decay products does not emit significant gamma radiation (see the fact sheet for Radioactive Properties, Internal Distribution, and Risk Coefficients). Units for external gamma risk coefficients shown in the table are risk per pCi/g soil for one year of exposure. Risk coefficients do not exist for curium-248, curium-250, or plutonium-244, so values shown have been derived (see the *Curium* and *Plutonium* fact sheets for derivation approach); the external value for curium-250 is attributable to its short-lived decay products.

Two parent radionuclides are shown here.



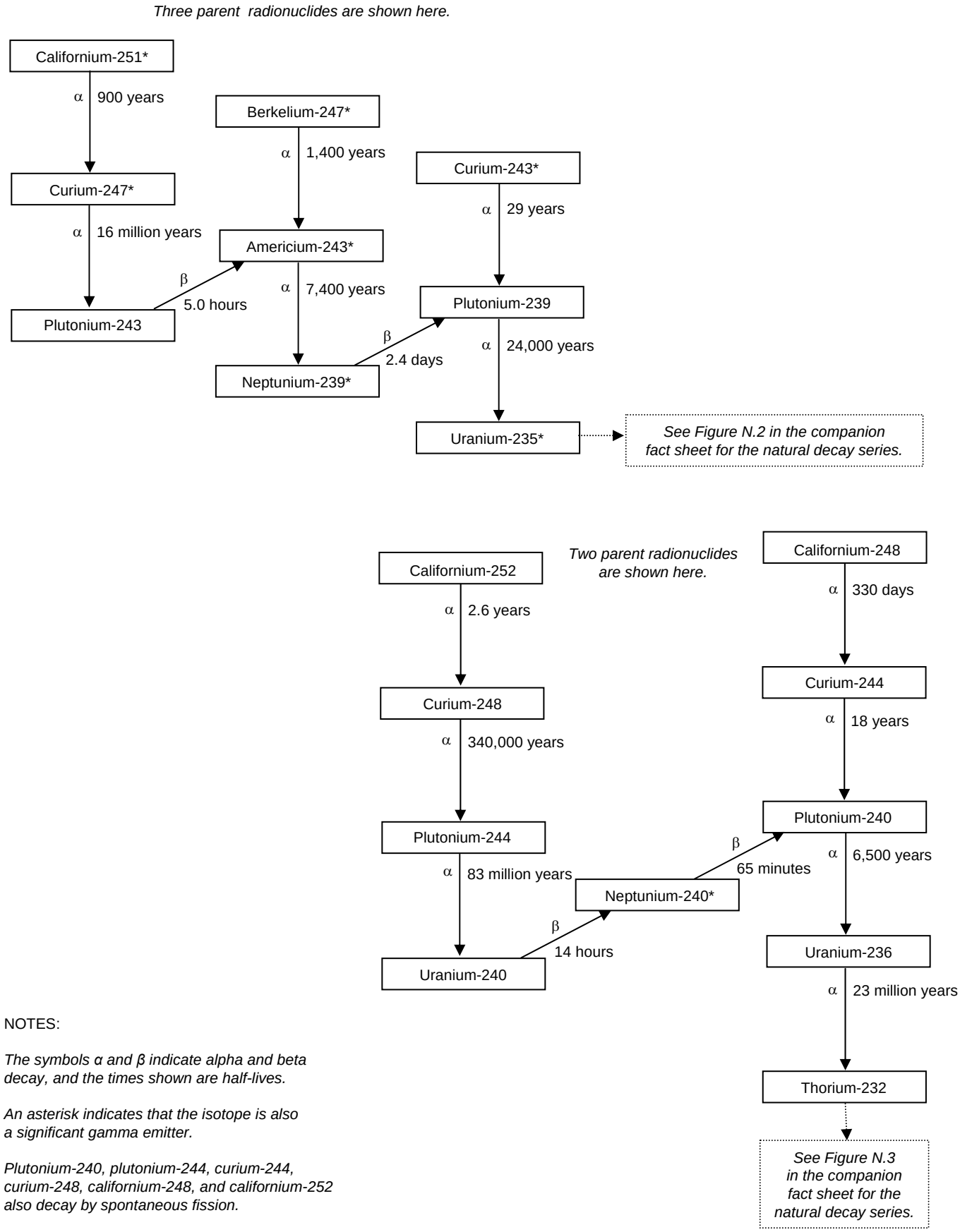
NOTES:

The symbols α and β indicate alpha and beta decay, and the times shown are half-lives.

An asterisk indicates that the isotope is also a significant gamma emitter.

Plutonium-238, plutonium-242, curium-242, curium-246, curium-250, and californium-250 also decay by spontaneous fission.

FIGURE T.1 Transuranic Decay Series: Americium-242m and Curium-242, and Curium-250



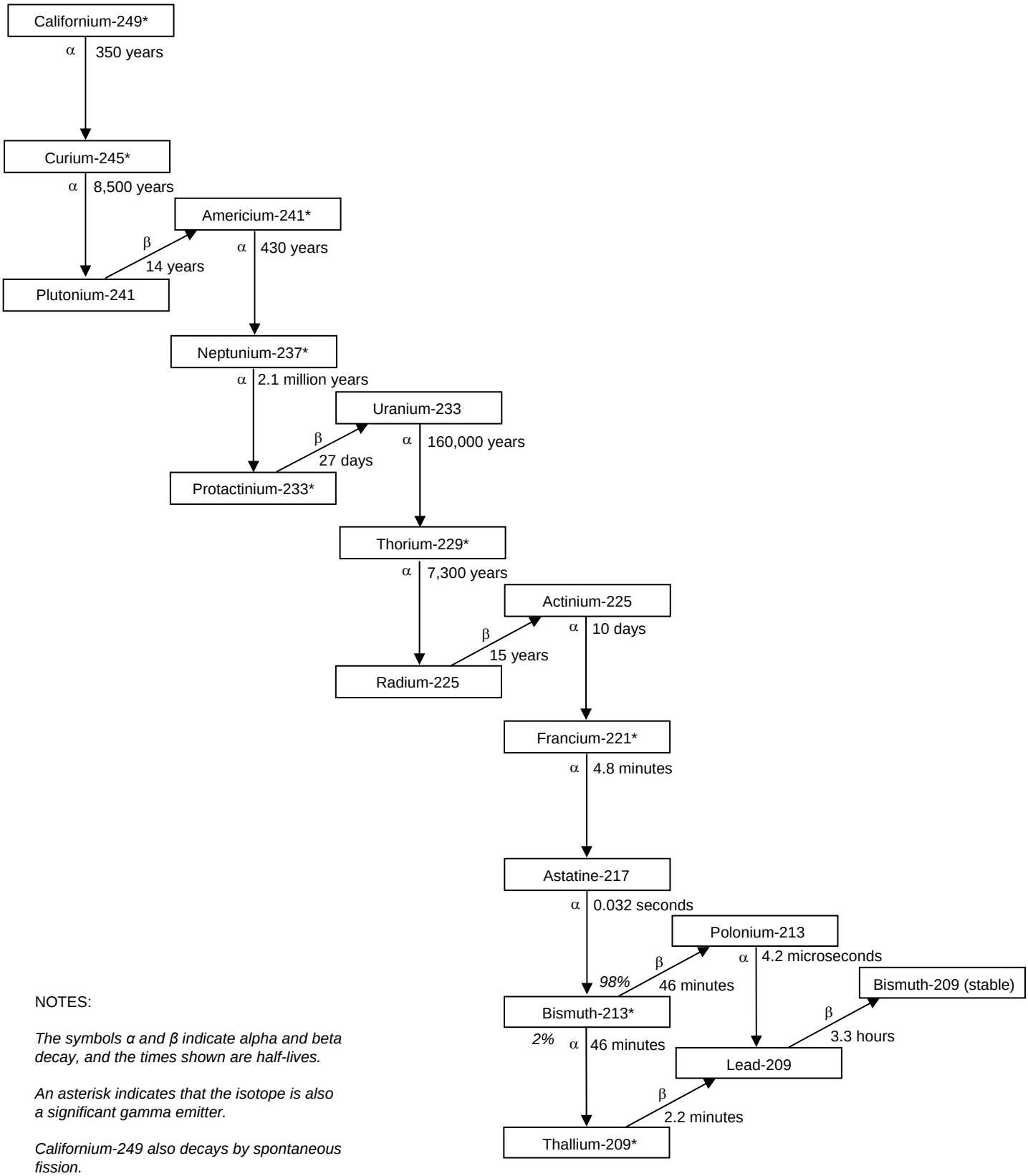
NOTES:

The symbols α and β indicate alpha and beta decay, and the times shown are half-lives.

An asterisk indicates that the isotope is also a significant gamma emitter.

Plutonium-240, plutonium-244, curium-244, curium-248, californium-248, and californium-252 also decay by spontaneous fission.

FIGURE T.2 Transuranic Decay Series: Californium-251, Berkelium-247, and Curium-243; Californium-252 and Californium-248



NOTES:

The symbols α and β indicate alpha and beta decay, and the times shown are half-lives.

An asterisk indicates that the isotope is also a significant gamma emitter.

Californium-249 also decays by spontaneous fission.

FIGURE T.3 Transuranic Decay Series: Californium-249

Radioactive Properties, Internal Distribution, and Risk Coefficients

The fact sheets for the individual radionuclides contain summary-level information on the radioactive properties of the major isotopes of concern at Department of Energy (DOE) environmental management sites such as Hanford, as well as information on the potential health risks associated with exposure to these radionuclides. This information was developed using standard references and publications as identified in Table 3. The fact sheets are intentionally brief and it is not possible to include all relevant information associated with the highlights summarized in these sheets. This companion fact sheet has been prepared to provide additional context to assist the reader in understanding the basis of the information presented in the individual fact sheets and to allow for proper interpretation of the radionuclide-specific data.

Radioactive Properties. Each radionuclide fact sheet contains a table that summarizes the key properties of the various radioactive isotopes of that element. The information provided in that table includes the radioactive half-life, specific activity, radioactive decay modes, and the average energy of the emitted radiations. To simplify the presentation, all values have been rounded to two significant figures. Much of the information was obtained from Appendix G of Federal Guidance Report (FGR) Number 13 of the Environmental Protection Agency (EPA), *Cancer Risk Coefficients for Environmental Exposure to Radionuclides*, 402-R-99-001 (September 1999). These data were also checked with the more detailed decay information in International Commission on Radiological Protection (ICRP) Report 38, *Radionuclide Transformations, Energy and Intensity of Emissions* (1983). The ICRP report is a major source of the information given in Appendix G of FGR Number 13.

Half-Life. The radioactive half-life is the length of time for a given amount of radioactive material to decrease to one half its initial amount by radioactive decay. Half-lives are constant for each radionuclide and can range from less than a second to billions of years. Only those radionuclides with half-lives longer than about one year are of concern for DOE environmental management sites, as shorter-lived radionuclides will have already decayed away to innocuous levels because production activities involving radioactive materials at major sites such as Hanford ceased more than ten years ago.

Specific Activity. The specific activity is the activity per mass and is given in units of curies (Ci) per gram in the individual fact sheets. For reference, the specific activity of radium-226 is about 1 Ci per gram, and for context 1 gram of material is about 0.035 ounce. The specific activities in the fact sheets (in units of curies per gram) were calculated using the following equation given in the Health Physics and Radiological Health Handbook (1992, p. 264). Specific Activity = $A_{Ra\text{-}226} \times T_{Ra\text{-}226} / A_i \times T_i$ where $A_{Ra\text{-}22a} = 226$, the atomic weight of radium-226; $T_{Ra\text{-}226} = 1{,}600$, the value used for the half-life of radium-226, in years (to two significant figures); A_i = the atomic weight of the isotope; and T_i = half-life of the isotope in years. The specific activity can be expressed in international units by multiplying the value in the fact sheet by 3.7×10^{10} becquerels (Bq) per Ci.

Decay Mode. The radioactive decay modes identified in the fact sheets include beta-particle emission, alpha-particle emission, isomeric transition (IT), electron capture (EC), and spontaneous fission (SF). The companion fact sheet on *Ionizing Radiation* contains additional information on the first two decay modes. The IT decay mode is a process whereby a nucleus in an elevated energy state (typically a metastable isotope designated by the letter "m") releases excess energy by emitting a gamma ray. The product of the decay is not a new isotope, but rather the same isotope in a reduced (more stable) energy configuration. The EC decay mode is a process in which an inner-shell electron orbiting the nucleus of an atom is "captured" by the nucleus where it combines with a proton to become a neutron, and excess energy is given off in the form of gamma rays. An outer-shell electron fills the "hole" left in the inner shell, and the excess energy associated with the movement of an outer-shell electron to an inner shell is given off as X-rays. The SF decay mode is a process in which an unstable nucleus splits (fissions) into two smaller products without needing additional neutrons to initiate the process, i.e., it is spontaneous. For simplicity, only the major decay modes are shown in the fact sheets; decay modes that occur less than 1% of the time are not included. Report 38 of the ICRP includes a very detailed accounting of all decay modes for each radionuclide and can be consulted for additional information.

In addition to the radioactive decay modes discussed above, there are additional mechanisms by which unstable atoms release energy. Internal conversion is a process in which the excess energy of a nucleus in an excited state is transferred to an electron orbiting the nucleus, which results in the electron being emitted from the atom. This process competes with gamma-ray emission as a mechanism for releasing excess energy from the nucleus, such as occurs during IT decay. Both internal conversion and EC result in a "hole" in the inner shell of orbital electrons, which is filled by an outer-shell electron with excess energy given off in the form of X-rays. These X-rays can interact with other orbital electrons, transferring sufficient energy to them to result in the emission of additional electrons. Such emitted electrons generated by interactions with X-rays are termed Auger electrons and have very little kinetic energy.

Decay Energy. The average energy reported for individual isotopes in the fact sheets represents the energy of the indicated radiation multiplied by the fractional yield for the given decay mode. That is, the energy reported for the various types of radiation represents the average energy per decay of the radionuclide. The energy of the radiation is given in units of million electron volts (MeV). One MeV is equal to 0.16 trillionth of a joule. The following two examples are provided to illustrate how this information should be interpreted.

Consider a radionuclide that decays by emitting an alpha particle with two different energies: half the time the energy is 5 MeV and the other half of the time the energy is 6 MeV. The energy reported for the alpha particle decay in this case would be 5.5 MeV. Consider a second example in which a radionuclide decays half the time by emitting a beta particle with an average energy of 0.5 MeV, and the other half of the time it decays by emitting an alpha particle with an energy of 6 MeV. The energy of the beta particle would be reported as 0.25 MeV (half of 0.5 MeV), and the energy of the alpha particle would be given as 3 MeV (half of 6 MeV). As a note, the average energy of a beta particle is typically about one-third the maximum energy (which is often the energy reported in radionuclide charts), or about 30% of the maximum for negatrons and 40% for positrons. (See the companion fact sheet on *Ionizing Radiation* for a discussion of negatrons and positrons.) The summary-level tables in the individual radionuclide fact sheets include the contributions of all primary (alpha and beta particles and gamma rays) and secondary (X-rays and Auger electrons) radiations.

The average energy reported for gamma rays in the fact sheets includes the contributions of X-rays and has been adjusted to account for the fractional yield. The only difference between these two types of electromagnetic radiation is their origin, and hence energy. Gamma rays originate in the nucleus as a means of releasing excess energy from the atom, while X-rays are emitted when electrons outside the nucleus move from higher to lower energy states. Radionuclides having gamma-ray energies less than 0.03 MeV per decay, considering the fractional yield (as described above), generally do not present a health concern from external gamma exposure. The average energy per decay reported for beta particles includes the contributions of all electrons and positrons regardless of their origin (internal or external to the nucleus). The average energy per decay reported for alpha particles does not include the contribution of the recoiling atom (which is typically quite small, e.g., a few percent of the total energy associated with the alpha-decay process).

Some radionuclides decay into short-lived daughters that always accompany the parent. (The term *parent* is used to describe the original isotope, and *daughter* is used for the decay product.) For example, strontium-90 decays to yttrium-90 by emitting a beta particle with a 29-year half-life. The daughter yttrium-90 quickly decays by emitting a beta particle, with a half-life of 64 hours. So for all practical purposes, each decay of strontium-90 can be considered to yield two beta particles, one for strontium-90 and one for yttrium-90. Short-lived decay products need to be considered when estimating the potential health effects of exposures to radionuclides. To facilitate this consideration, the radioactive properties of both the parent and its short-lived daughter(s) are presented in the individual radionuclide fact sheets.

Internal Distribution. To estimate the human health risks associated with radionuclides, it is necessary to follow the movement of the isotopes from intake through excretion. These isotopes constantly emit radiation at a rate proportional to their specific activity as they pass through the body irradiating various organs. Some radionuclides very quickly deposit in one or two organs; others deposit more slowly throughout the entire body. Various models and computer codes have been developed by the ICRP, EPA, and other national and international organizations to estimate internal radiation doses and risks from intake of radionuclides. These models are based on extensive animal and human data and can be quite complex. A number of codes and models were considered by the EPA in developing the risk coefficients presented in FGR Number 13, as illustrated by the references in that document. The risk coefficients were calculated used the DCAL (Dose and Risk Calculation) software developed by Oak Ridge National Laboratory for the EPA. The DCAL is a comprehensive system for calculating radiation dose and risk coefficients using age-dependent models that incorporate information developed by the ICRP and other organizations on the distribution and retention of radionuclides by various organs in the body. The primary distribution of selected radionuclides in the body is shown in Figure 1 of the accompanying distribution fact sheet.

Risk Coefficients. The EPA has developed mortality risk coefficients for nearly all radionuclides to estimate the lifetime risk of incurring a fatal cancer from environmental exposures using the DCAL software as described in FGR Number 13. These coefficients have been calculated by state-of-the-art methods and computer models that take into account age and gender dependence of intake, metabolism, dosimetry, and radiogenic risk, as well as competing causes of death, to estimate health risks from internal and external exposures. The values are given per unit uptake (picocurie, pCi) averaged over all ages and both genders. (For context, 10^{-9} is a billionth, 10^{-12} is a trillionth, and a pCi is a trillionth of a Ci.) To convert to standard international units, the given values should be multiplied by 27 pCi/Bq.

Each radionuclide fact sheet contains a table with selected mortality risk coefficients for inhalation and ingestion, which are also summarized in Table 1. These values include the contributions from short-lived decay products, as identified on the radioactive properties summary table described above (e.g., the value for strontium-90 includes the contribution from yttrium-90). For inhalation, the values correspond to the recommended default absorption type for particulates, except as otherwise noted (e.g., the tritium values are for tritiated water). For ingestion, the dietary values shown are the highest for ingestion exposures; the values for tap water ingestion vary by radionuclide and are typically 70 to 80% of those for dietary intake. Coefficients are also available to estimate the risk of incurring all types of cancer (morbidity risk coefficients), and these values also vary by radionuclide. For most radionuclides, the ingestion mortality coefficients are on the order of 60 to 80% of the morbidity values, with iodine an exception at about 10%. For inhalation the percentages are a bit higher, ranging from 70% (for cesium and tritium) to nearly 100% (for uranium-234 and uranium-238), with the mortality coefficient for iodine again much lower at about 10%. For major gamma-emitting radionuclides, risk coefficients are shown in Table 1 and risk text is included in the fact sheet.

Radiological Dispersal Device (RDD)

What Is an RDD? A *radiological dispersal device (RDD)* is an unconventional weapon that a terrorist might use to destabilize a community, as described at right. Although often used to represent a dirty bomb, the radioactivity in an RDD could also be distributed passively (nonexplosively), such as through spraying or spreading by hand. Alternately, a *radiological exposure device (RED)* might be used, which would simply involve placing a radioactive source in a public area to expose people passing by.

Radiological Dispersal Device:

Any method used to deliberately disperse radioactive material to create terror or harm. A dirty bomb is an example of an RDD. It is made by packaging explosives (like dynamite) with radioactive material to be dispersed when the bomb goes off.

Where Would the Radioactive Material Come From? Radionuclides are used in a variety of industry, medicine, and scientific research applications, as illustrated by the examples below. Many of these are in sealed sources, used in civil engineering (in flow gauges and to test soil moisture and material thickness/integrity for construction), in petroleum engineering (in well logging for oil exploration), in the airline industry (in fuel gauges and to check welds and structural integrity), in medicine (cancer treatment, pacemakers, and diagnostics), in homes (smoke detectors), and to make electricity (in radiothermal generators or RTGs, that generate power in remote areas ranging from lighthouses to outer space).

Examples of Radionuclides in Common Use

Medicine		**Industry/Commerce**				**Science**
Diagnosis	***Treatment***	***Energy, Defense***	***Testing, Production***	***Food, Agriculture***	***Home***	***Research***
Tracer, flow *(Tc-99m, I-131)*	Gamma knife, blood/tissue sterilization *(Cs-137, Co-60)*	Commercial electricity *(U, Pu)*	Nondestructive test of structural integrity, radiographic imaging *(Co-60, Ir-192)*	Food product sterilization *(Co-60)*	Smoke detector *(Am-241)*	High-energy physics *(Cf-252, U-235)*
Tissue scan for clot, mass *(Ga-67)*	Needle, seed implants *(Cs-137, Ir-192, Ra-226)*	Remote power *(Sr-90)*	Density, moisture gauges *(Am-241, Cs-137)*	Pest (fruit fly) sterilization *(Cs-137, Co-60)*	Luminescent watch/clock dial *(H-3)*	Biokinetics *(Pu, Sr-90, others)*
X-ray *(Cs-137, Co-60)*	Pacemaker *(Pu-238)*	Defense/weapons *(Pu, H-3, U and depleted U)*	Material thickness, flow, conveyor, level gauges *(Am-241, Cs-137, Co-60, Kr-85)*	Seed, spice sterilization *(Cs-137, Co-60)*	Gas camping lantern *(Th-232)*	Biological tracer, protein/synthesis *(C-14, H-3, N-15 P-32, S-35)*

Am-americium, C-carbon, Cf-californium, Co-cobalt, Cs-cesium, Ga-gallium, H-3-tritium, I-iodine, Ir-iridium, K-potassium, Kr-krypton, N-nitrogen, P-phosphorous, Pu-plutonium, Ra-radium, S- sulfur, Sr-strontium, Tc- technetium, Th- thorium, U-uranium.

Radioactive sources can be portable or fixed, and most are quite small, ranging from tiny brachytherapy needles or seeds (implanted for localized cancer treatment) to thimble-sized plugs sealed within secure capsules for industrial gauges. Even the larger sources are fairly small; for example, the radioactive component of an RTG can range from the size of a roll of duct tape to a small wastebasket, although the outer housing can more than double the overall size. Most sources are encapsulated or sealed in housings of stainless steel, titanium, platinum, or other metal, and gamma emitters are encased in dense shielding (such as lead) to attenuate external gamma irradiation.

Only some of the materials identified above are considered likely RDD candidates, based on portability coupled with relatively high levels of radioactivity. Not of concern are those with minute amounts of radioactivity, e.g., smoke detectors, camping lanterns, or brachytherapy needles; these would not constitute a dispersal or exposure issue even if thousands were collected to extract their radioactive material. (Key radionuclides of concern for RDDs are described in the next section.) Radioactive waste from the nuclear power industry or legacy weapons facilities is also considered a possible source, with its attractiveness depending on the specific radionuclides in that waste, their physical and chemical forms, and levels of radioactivity. High-activity wastes (e.g., from nuclear energy reactors) are well controlled, and the largest volumes of radioactive waste typically contain relatively low concentrations, so these materials are generally considered a secondary concern for RDDs.

Which Radionuclides Are of Most Concern? Although dozens of radionuclides are used across various sealed sources (selected devices and associated sources are shown at right), only a small number are in concentrated amounts or are widely available. Nine isotopes of interest for RDDs are:

- Americium-241 *(Am-241)*
- Californium-252 *(Cf-252)*
- Cesium-137 *(Cs-137)*
- Cobalt-60 *(Co-60)*
- Iridium-192 *(Ir-192)*
- Plutonium-238 *(Pu-238)*
- Polonium-210 *(Po-210)*
- Radium-226 *(Ra-226)*
- Strontium-90 *(Sr-90)*

Source for medical teletherapy machine (1-in.diameter, within stainless steel housing) *(Cs-137, Co-69)*

Teletherapy machine

Source for industrial radiography device (like for medical unit) *(Co-60, Ir-192)*

Industrial radiography device

Source inside capsule at end of flexible cable *(Co-60, Ir-192; well logs also use Am-241, Cs-137)*

Well logging devices with source at tip

Russian radiothermal generators (RTGs) *(Sr-90)*

Basic radiological properties for these nine isotopes are summarized below. (Note: radium-226 exists in nature, as does a small amount of polonium-210; the rest are man made.)

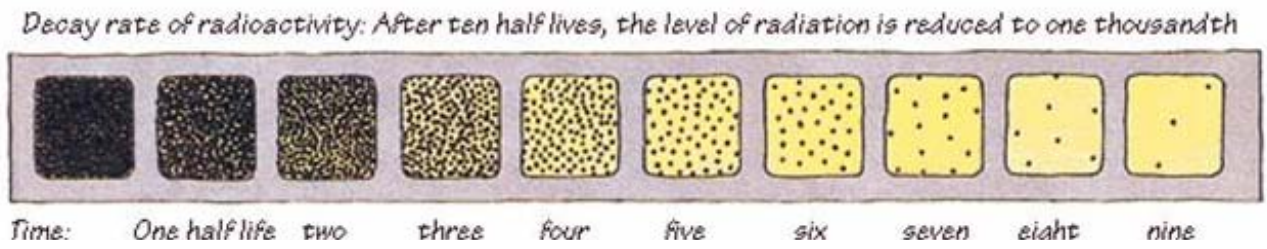
The specific activity of a radionuclide is inversely proportional to its half-life, as curies per gram (Ci/g). Unique to each isotope, it provides an indication of the rate at which that given radionuclide decays. Note that although iridium-192 has the highest specific activity among the nine, it decays to a stable isotope much more quickly than the others because its half-life is only 2.5 months. As a general rule of thumb, 7 to 10 half-lives can indicate how long an isotope could be expected to remain radioactive. (Less than 1% of the original amount remains after seven half-lives.) For example, if iridium-192 were dispersed, it would probably be gone within 2 years, while levels of cobalt-60 would drop to 1 percent within 40 years, but it would take cesium-137 more than 200 years to be reduced to the same level.

Basic Radiological Properties of Nine Key Radionuclides for RDDs

Isotope	*Half-Life (years)*	*Specific Activity (Ci/g)*	*Decay Mode*	*Radiation Energy (MeV)*		
				Alpha (α)	*Beta (β)*	*Gamma (γ)*
Americium-241	430	3.5	α	5.5	0.052	0.033
Californium-252	2.6	540	α (SF, EC)	5.9	0.0056	0.0012
Cesium-137	30	88	β, IT	-	0.19, 0.065	0.60
Cobalt-60	5.3	1,100	β	-	0.097	2.5
Iridium-192	0.2 *(74 d)*	9,200	β, EC	-	0.22	0.82
Plutonium-238	88	17	α	5.5	0.011	0.0018
Polonium-210	0.4 *(140 d)*	4,500	α	5.3	-	-
Radium-226	1,600	1.0	α	4.8	0.0036	0.0067
Strontium-90	29	140	β	-	0.20, 0.94	-

SF = spontaneous fission; IT = isomeric transition; EC = electron capture. A hyphen means not applicable. The radiation energies for cesium-137 include the contributions of barium-137 metastable (Ba-137m), and those for strontium-90 include the contributions of yttrium-90.

This concept of half life is illustrated below (from the Uranium Information Center, Melbourne, Australia, www.uic.com.au/ral.htm):



Three of the nine isotopes considered candidates for an RDD are strong gamma-ray emitters: Cs-137 (from Ba-137m), Co-60, and Ir-192. These three could pose an external hazard to individuals who handle them (e.g., potential terrorists) if their protective shielding was removed or not used. In fact, it is precisely their gamma radiation that makes these three isotopes valuable for commercial and medical applications. Gamma emitters are used to sterilize food and equipment, irradiate tumors, nondestructively evaluate high-integrity welds and castings (industrial radiography), and in industrial gauges.

A fourth, Sr-90, emits beta particles and has limited but notable commercial uses. Like alpha emitters, beta emitters primarily represent an internal health hazard if ingested or inhaled. The major use of Sr-90 is in RTGs, and many of these were produced by the former Soviet Union to generate electricity in remote locations for applications such as lighthouses. The poor accountability for Soviet-era RTGs has been widely publicized, and they pose a considerable threat because they may contain tens of thousands of curies of Sr-90. Radioactive decay has substantially reduced initial levels of older RTGs, but levels in newer units could be much higher than 10,000 Ci. Summary information for these four isotopes in typical sealed sources is highlighted at left.

Selected Highlights for Typical Commercial Sources

Radionuclide	*Typical Form*	*Initial Activity (Ci)*	
		Medical	*Industrial*
Cesium-137	Cesium chloride	0.01-10	10-1,000
Cobalt-60	Metallic cobalt or alloy	0.01-10	10-1,000
Iridium-192	Metallic iridium	0.01-10	10-1,000
Strontium-90	Strontium chloride, fluoride, titanate	1-10	1-10,000

The remaining isotopes are primarily alpha emitters: Am-241, Cf-252, Po-210, Pu-238, and Ra-226. Alpha particles are easily shielded with only minimal amounts of material, so they do not pose a significant external health hazard. Rather, their significance relates to health concerns if ingested or inhaled. In addition, Am-241 is commonly mixed with beryllium to produce a neutron-emitting source. Similarly, Cf-252 emits neutrons through spontaneous fission. Neutron emitters represent both an external and internal health hazard. Among other applications, alpha or neutron emitters have been used in soil moisture/density gauges, medical pacemakers, and well logging gauges used in the petroleum industry.

How Dispersible Would these Radionuclides Be? Dispersibility will depend on the physical and chemical properties of the radioactive material used in an RDD. Metallic forms would be difficult to disperse while a powder could be dispersed fairly readily. Common forms of radionuclides in sealed sources are shown on the next page. Cobalt, iridium, and polonium generally exist as solid metals and would not be readily dispersible. Several of the others, including americium, californium, and plutonium, are typically oxides that could exist as a powder. Cesium is typically found as cesium chloride, which is also a powder and is quite soluble in water. Radium and strontium are used in various forms; strontium fluoride in certain sealed sources is sintered such that it is essentially insoluble and nondispersible. Even considering the forms in current sources, the specific physical and chemical characteristics of radioactive materials that could be in an RDD is uncertain because the original material could be chemically or physically altered (weaponized) to enhance dispersal. If the dispersal method is explosion via a dirty bomb, that would also likely physically and chemically alter the materials to produce a mixture that could include oxides as well as nitrates (from the explosives) over a range of particle sizes.

Chemical Forms of Radioactive Materials Often Found in Sealed Sources	
Radionuclide	***Form***
Americium-241	Americium oxide; americium-beryllium (AmBe) neutron sources are typically compressed powders
Californium-252	Californium oxide
Cesium-137	Cesium chloride
Cobalt-60	Metallic cobalt, or cobalt-nickel alloy
Iridium-192	Metallic iridium
Plutonium-238	Plutonium dioxide, generally pressed into a ceramic-like material
Polonium-210	Metallic foil
Radium-226	Radium bromide or radium chloride
Strontium-90	Metallic strontium, strontium chloride, strontium-fluoride, strontium-titanate

What Would the Response to an RDD Involve? The response to an RDD event would consist of several phases. The first phase would involve immediate life-saving measures, such as treating blast victims and evacuating areas as indicated (e.g., based on radioactivity levels). The second phase would involve evaluating the extent of contamination and taking measures to control further contamination and minimize human exposures. The last phase would involve recovery and cleanup efforts, including decontamination and remediation of contaminated property.

As background on potential health effects, evidence linking radiation exposure to observable biological effects has only been found at relatively high doses, i.e., acute doses exceeding 25 rads (see below). (For context, natural background radiation translates to an average annual dose of about 0.3 rem, which is far below the threshold for acute effects and corresponds to a lifetime risk of about 1 in 100.) On average, about half of all cancers that can be induced by radiation are fatal; this ranges from about 10% for thyroid cancer to essentially 100% for liver cancer. An RDD would most likely result in relatively small radiation exposures, which overall might not substantially differ from an annual background dose. But in the unlikely event someone was highly exposed, chelation therapy (to enhance excretion) and other medical interventions could be pursued, including to limit internal deposition.

Threshold Doses for Prodromal Effects	
***Dose** (rads)*	***Indicator Effects***
50	Blood count changes
100	Nausea, vomiting, appetite loss, malaise, and fatigue
200	Diarrhea or bloody diarrhea
300	Epilation (hair loss)
500	Erythema (skin reddening)

The degree of decontamination for a given area would depend on conditions specific to that setting. Federal, state and local officials are developing emergency response plans, but a common set of numerical standards for RDD cleanup has not been established. Although various radiation regulations would be used as guides, none are directly applicable to RDD scenarios. More than 10 years ago, in its manual of protective actions guides (PAGs), EPA identified nonbinding recommendations for responses in the early and intermediate stages of a radiological emergency. However, these PAGs were developed for accidents at nuclear power plants and not for incidents of radiological terrorism. Specifically the PAGs do not contain guidance for the long-term phase, final cleanup.

The Department of Homeland Security has formed an interagency working group that includes representatives from eight Federal departments and agencies to develop new guidance for cleaning up after any RDD attack. Recommendations are expected to include use of the EPA PAG values for radiation exposure in the early and intermediate stages of a radiological terrorist attack. For the cleanup of areas contaminated by an RDD, rather than using a single numeric guideline that would not be able to account for all settings, it is expected that local stakeholders and decision makers would follow a process to develop cleanup plans tailored to the specific characteristics of the given situation, considering

optimization approaches. Thus, residual radionuclide concentrations would be expected to vary from case to case, and there will not likely be a uniform, generic cleanup level.

Illustrative Case Study: 1987 Radiological Accident in Goiania, Brazil

In September 1987, a hospital in Goiania, Brazil, moved to a new location and left its radiation cancer therapy unit behind. Found by scrap metal hunters, it was dismantled and the cesium chloride source containing 1,400 Ci of cesium-137 was removed. Pieces were distributed to family and friends, and several who were intrigued by the glow spread it across their skin. Eleven days later, alert hospital staff recognized symptoms of acute radiation syndrome in a number of victims.

The ensuing panic caused more than 112,000 people – 10% of the population – to request radiation surveys to determine whether they had been exposed. At a makeshift facility in the city's Olympic Stadium, 250 people were found to be contaminated. 28 had sustained radiation-induced skin injuries (burns), while 50 had ingested cesium, so for them the internal deposition translated to an increased risk of cancer over their lifetime. Tragically, 2 men, 1 woman, and 1 child died from acute radiation exposure to the very high levels of gamma radiation from the breached source.

In addition to the human toll, contamination had been tracked over roughly 40 city blocks. Of the 85 homes found to be significantly contaminated, 41 were evacuated and 7 were demolished. It was also discovered that through routine travels, within that short time people had cross-contaminated houses nearly 100 miles away. Cleanup generated 3,500 m^3 radioactive waste at a cost of $20 million.

The impacts of this incident continued beyond the health and physical damage to profound psychological effects including fear and depression for a large fraction of the city's inhabitants. Further, frightened by the specter of radioactive contamination, neighboring provinces isolated Goiania and boycotted its products. The price of their manufactured goods dropped 40% and stayed low for more than a month. Tourism, a primary industry, collapsed and recent population gains were reversed by business regression. Total economic losses were estimated at hundreds of millions of dollars. A key lesson learned from this incident is the importance of enhancing the broader understanding of radiation. This fact sheet is intended to help support that objective.

(For additional information see: International Atomic Energy Agency (IAEA), 1988, *The Radiological Accident in Goiania*, Vienna, Austria.)




Where Can I Find More Information about RDDs? In the last several years, a number of reports, studies, articles, and books have been published that discuss issues related to RDDs. An introduction to selected resources follows; this list is not intended to be comprehensive.

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Health-Based Radionuclide Concentrations in Drinking Water and Air

To address radionuclide releases to water (notably drinking water) or air, health-based concentrations can be developed to support environmental responses by using standard dose conversion factors and risk estimators from the U.S. Environmental Protection Agency (EPA), as presented in Federal Guidance Reports 11 and 13 (EPA 1988 and EPA 1999). Dose-based concentrations for 15 radionuclides that are of interest to various environmental programs are tabulated below.

Drinking Water and Air Concentrations for Selected Radionuclides[a]

Radionuclide	Specific Activity *(Ci/g)*	Drinking Water Maximum Contaminant Level (MCL)-Equivalents		National Emissions Standard for Hazardous Air Pollutants (NESHAPs) Equivalents	
		(pCi/L)	*(µg/L)*	*(pCi/m^3)*	*($µg/m^3$)*
Americium-241	3.5	1.5	4.3×10^{-7}	0.0031	8.8×10^{-10}
Californium-252	540	5.1	9.4×10^{-9}	0.0087	1.6×10^{-11}
Cesium-137	88	110	1.2×10^{-6}	43	4.9×10^{-7}
Cobalt-60	1,100	200	1.9×10^{-7}	6.3	5.7×10^{-9}
Europium-154	270	570	2.1×10^{-6}	4.8	1.8×10^{-8}
Hydrogen-3 *(tritium)*	9,800	86,000	8.7×10^{-6}	21,000	2.2×10^{-6}
Iodine-129	0.00018	20	0.11	7.9	0.045
Iridium-192	9,200	950	1.0×10^{-7}	49	5.3×10^{-9}
Plutonium-238	17	1.7	1.0×10^{-7}	0.0035	2.1×10^{-10}
Radium-226	1.0	5	5.0×10^{-6}	0.16	1.6×10^{-7}
Ruthenium-103	32,000	1,800	5.6×10^{-8}	150	4.8×10^{-9}
Ruthenium-106	3,400	200	5.9×10^{-8}	2.9	8.4×10^{-10}
Strontium-90	140	8	5.7×10^{-8}	1.0	7.5×10^{-9}
Technetium-99	0.017	3,700	0.22	1,400	0.079
Uranium-238	0.00000034	10	30	0.12	0.35

[a] Ci = curie; g = gram ; L = liter; m^3 = cubic meters ; pCi = picocurie (10^{-12} curie); µg = microgram (10^{-6} gram); calculated values are given to two significant figures. Shading indicates the five isotopes with short-lived decay products: cesium-137 (barium-137m, 3-min half life); radium-226 (radon-222, 3.8-d half life, and its daughters); ruthenium-106 (rhodium-106, 30-s half life); strontium-90 (yttrium-90, 64-h half life); and uranium-238 (thorium-234, 24-d half life; to protactinium-234m, 1.2-min half life). The strontium-90 MCL was established years ago using old dosimetry models; updated dosimetry gives 36 pCi/L.

Because these concentrations are derived from EPA dose limits for drinking water and air, they are calculated using the current dose conversion factors from Federal Guidance Report 11 (EPA 1988) with the standard EPA intake assumptions of 2 L drinking water per day (2 L/d) for ingestion and 20 m^3 air/d for inhalation. In this table, the MCL (for strontium) and the MCL and NESHAP equivalents include the contributions of short-lived decay products. For the five radionuclides with short-lived daughters (shaded above), these daughters have half-lives of less than a month. This approach considers that beyond internal ingrowth after intake, the short-lived decay products would also be present at the same concentrations as the parents (i.e., in secular equilibrium) in the air or water to which someone is exposed.

Longer-lived decay products are not included in the values above; notably the contributions of lead-210 (which has a 22-year half-life) and its two short-lived decay products are not included in the value for radium-226 because it takes many years for them to occur by radionuclide ingrowth. By this approach, information in Table 1 can be used to assess introduced contaminants to which exposures would be finite

rather than continuous, given that interest in discrete releases has increased within various environmental protection and preparedness programs. To also support analyses of long-term scenarios, such as chronic exposures to continuing airborne releases or to groundwater contaminated by past industrial releases, the contributions of lead-210 and its short-lived decay products to the radium-226 values for drinking water and air are provided in the following discussion.

For radium-226 in air, incorporating the contributions of lead-210 and its two decay products lowers the value from 0.16 to 0.043 pCi/m^3. For drinking water, EPA has established MCLs for radium-226, strontium-90, and uranium. The MCL for radium-226 and radium-228 combined is 5 pCi/L, which is shown here as the level for radium-226. Because radium-228 originates from a different decay chain and has a much shorter half-life of 5.8 years, it would not be expected to coexist with introduced radium-226, so no adjustment is warranted for the combined value. Accounting for all decay products over time (to stable lead-206), the concentration for radium-226 that gives an annual dose of 4 mrem (as the 50-yr committed effective dose equivalent, or CEDE) would be 0.64 pCi/L; excluding the ingrowth of lead-210 and its two short-lived decay products (which occurs over the longer term), the value would be 4.1 pCi/L.

The MCL for strontium-90 was established many years ago based on extant dosimetry models. The value using updated dosimetry with the 4 mrem/yr annual dose limit is 36 pCi/L. The MCL for total uranium is 30 µg/L. Applying the specific activity of uranium-238 to this MCL produces the value of 10 pCi/L for that isotope alone. Uranium-234 (and a very small amount of uranium-235) would be expected to accompany uranium-238 in an introduced source, so the total uranium activity would be about 20 pCi/L for uranium in its natural isotopic ratios at the MCL concentration of 30 µg/L. If someone drank 2 L every day for a year with this total uranium level, the resulting estimated dose would be 4 mrem (on the order of 1% of average background). For the other nine isotopes, an MCL equivalent has been calculated from the EPA annual dose limit for drinking water of 4 mrem assuming an ingestion rate of 2 L/d. Although this dose limit is specifically for radionuclides that emit beta particles and photons, it was also used here to derive concentrations for alpha emitters for which isotope-specific MCLs do not exist (americium-241, californium-252, and plutonium-238); note this aligns with the dose comparison for uranium above. The MCL for these isotopes would be 15 pCi/L as gross alpha, so levels estimated from the 4 mrem/yr dose are below this MCL – at 1.5, 5.1, and 1.7 pCi/L, respectively.

For inhalation, EPA has identified an annual dose limit of 10 mrem for the air pathway in NESHAPs. Indicated as the effective dose equivalent (EDE) (in 40 CFR 61, Subpart H), the standard NESHAPs compliance code CAP88-PC integrates internal EDEs for 50 years, which results in the 50-year CEDE. The NESHAP-equivalent values in Table 1 correspond to this annual dose limit as CEDEs, assuming an inhalation rate of 20 m^3/d. This approach is also consistent with the CEDE basis of the MCL equivalents. As further context for the radium-226 decay product, radon-222 in air, the EPA limit for indoor radon is 4 pCi/L, or 4,000 pCi/m^3.

In addition to these dose-based calculations that use the dose conversion factors in Federal Guidance Report 11, radiological risk estimators (as risk per pCi) in the more recent Federal Guidance Report 13 can also be used to back-calculate isotope-specific concentrations for a given target risk level. To illustrate for two isotopes, concentrations corresponding to a 10^{-4} risk level for cancer incidence from tap water ingestion for one year are 4,500 pCi/L for cesium-137 and 1,900 pCi/L for strontium-90. For longer exposures, these numbers are divided by the number of years to obtain a duration-specific value.

References

EPA, 1988, *Limiting Values of Radionuclide Intake and Air Concentration and Dose Conversion Factors for Inhalation, Submersion, and Ingestion*, Federal Guidance Report No. 11, EPA-520/1-88-020, Office of Radiation Programs, Washington, DC (September).

EPA, 1999, *Cancer Risk Coefficients for Environmental Exposure to Radionuclides*, Federal Guidance Report No. 13, EPA 402-R-99-001, Office of Radiation and Indoor Air, Washington, DC (September).

Arsenic

Symbol:	**As**
Atomic Number: *(protons in nucleus)*	**33**
Atomic Weight:	**75**

What Is It? Inorganic and organic arsenic occur naturally in the environment, with inorganic forms being most abundant. Inorganic arsenic is associated with other metals in igneous and sedimentary rocks, and it also occurs in combination with many other elements, especially oxygen, chlorine, and sulfur. Organic arsenic contains carbon and hydrogen. Both inorganic and organic forms exist naturally in soils, plants, animals, and humans. Most pure, inorganic arsenic compounds are white or colorless powders with no specific smell or taste. Because it is an element, arsenic does not degrade nor can it be destroyed.

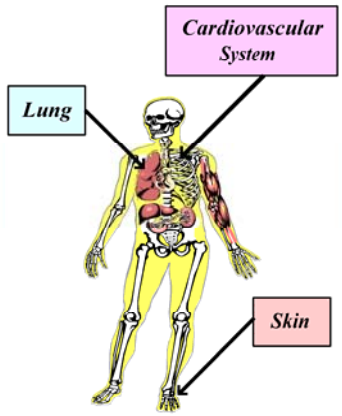
How Is It Used? Arsenic has been recognized as a poison since ancient times. In past centuries it was used to treat syphilis, and decades ago it was a common active ingredient in pesticides and was also a common wood preservative. Today, about 90% of arsenic produced is used as a wood preservative (chromated copper arsenate). Although organic arsenicals continue to be used as pesticides, primarily on cotton, inorganic compounds can no longer be used. Arsenic is also used as a feed additive for poultry and swine and in cattle and sheep dips to control lice and ticks. In addition, arsenic is used in alloys (primarily in lead-acid batteries for automobiles) and in semiconductors and light-emitting diodes.

What's in the Environment? Arsenic occurs everywhere in the environment. Weathering of rock is the major natural source of inorganic arsenic, and it is also released by human activities. For example, arsenic is emitted as a fine dust when arsenic-containing ores are heated at smelters to process copper or lead. The concentration of arsenic in the earth's crust ranges from 2 to 5 milligrams per kilogram (mg/kg), or parts per million (ppm). The mean natural soil concentration is 5 mg/kg, and it ranges from about 1 to 40 mg/kg. Water-soluble arsenites (the trivalent form, As III) and arsenates (the pentavalent form, As V) are the most common forms. Arsenites especially can be relatively mobile, with a typical concentration associated with soil particles estimated to be 10 to 200 times higher than in the interstitial water (water in the pore spaces between the soil particles). Levels in drinking water are commonly 2 to 20 parts per billion (ppb). Bacteria, fungi, and some plants methylate inorganic arsenic, converting it to organic compounds. Many methylated forms are volatile, such as dimethylarsine. Some organisms (notably in aquatic systems) can accumulate nontoxic, organic forms of arsenic; for example, levels of arsenobetaine in shrimp are often high. However, the typical ratio of the arsenic concentration in plants to that in soil is low, estimated at 0.006 (or 0.6%).

What Happens to It in the Body? Arsenic can be taken in by eating food, drinking water, or breathing air, and to a limited degree via skin contact. Children, and to a lesser extent adults, can also be exposed by ingesting soil. When ingested, dissolved arsenic compounds are readily absorbed (80-90%) through the gastrointestinal tract and distributed in the blood to the liver, kidney, lung, spleen, aorta, and skin. Two processes are involved in arsenic metabolism: (1) oxidation/reduction reactions that interconvert arsenate and arsenite, and (2) methylation of arsenite to form monomethyl arsenic acid and dimethyl arsenic acid. The methylated forms are less toxic and more easily excreted in the urine. Most arsenic is eliminated in the urine within a week (75-90%, depending on the compound), especially from the liver, kidney, and spleen, while that in the skin, brain, skeleton, and especially hair and nails, remains somewhat longer. When arsenic is inhaled and deposited in the lungs, about 80% is absorbed into the bloodstream and distributes throughout the body as above. Arsenic in soil or dissolved in water does not readily penetrate the skin (only 3% is estimated to be absorbed), so dermal exposures are not typically a concern.

What Are the Primary Health Effects? Depending on the amount ingested, arsenic can be beneficial (animal studies suggest that low levels of arsenic in the diet are essential) or adverse (high levels can be toxic). The acute lethal dose to humans can be about 2 to 20 mg/kg body weight per day (mg/kg-day). Ingesting high doses of arsenic irritates the stomach and intestines, with symptoms including nausea, vomiting, diarrhea and liver swelling. However, wide recognition of its toxicity makes arsenic poisoning today very rare. Ingesting small amounts over time produces chronic effects such as skin darkening and formation of corns, damage to peripheral nerves, cardiovascular system effects, hair and appetite loss, and mental disorders. Effects from inhaling arsenic dust

include respiratory irritation, rhinitis, pharyngitis, laryngitis, and sometimes nasal perforation. Skin contact with inorganic arsenic dusts can cause dermatitis, allergic hypersensitivity, and conjunctivitis. Occupational exposure studies show a correlation between chronic arsenic exposure and lung cancer. Arsenic can also cause reproductive/developmental effects, including spontaneous abortions and reduced birth weights. Epidemiological studies indicate an association between arsenic concentrations in drinking water and increased incidences of skin, liver, kidney, lung, and bladder cancers. Studies also show an association between inhaling arsenic and lung cancer. From these sets of data, the U.S. Environmental Protection Agency (EPA) has classified inorganic arsenic as a known human carcinogen. Limited information is available on the joint toxicity of arsenic with other chemicals. For neurological effects, the predicted direction of joint toxicity of arsenic and lead is greater than additive, whereas the joint toxicity of these metals is predicted to be less than additive for the kidney and hematopoietic (blood-forming) system. The joint toxicity of arsenic and cadmium on the kidney, hematopoietic system, and male reproductive system is predicted to be less than additive. Additional information on joint toxicity is provided in the companion chemical mixtures fact sheet.



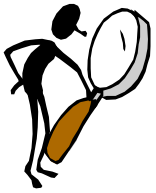
Primary organs affected when arsenic is inhaled or ingested.

What Is the Risk? The EPA has developed toxicity values *(see box below)* to estimate the risk of getting cancer or other adverse health effects as a result of inhaling or ingesting inorganic arsenic. These toxicity values have been developed based on studies of workers exposed to arsenic in occupational settings, workers applying arsenical pesticides, and populations consuming drinking water containing high concentrations of arsenic. The toxicity value for estimating the risk of getting cancer following oral exposure is called a slope factor (SF), and the value for estimating risk following inhalation exposure is called an inhalation unit risk (UR). An SF is an estimate of the chance that a person exposed to the chemical will get cancer from ingesting 1 mg/kg-day for a lifetime. The UR is an estimate of the chance that a person will get cancer from continuous exposure to a chemical in air at a unit concentration of 1 mg per cubic meter (m^3). Using the oral SF, the EPA estimates that a person would have a one-in-a-million chance of developing cancer if exposed daily to 2 liters (L) of drinking water containing 0.02 microgram (µg)/L arsenic. Similarly, using the inhalation UR, EPA estimates that a person would have a one-in-a-million chance of developing cancer if exposed daily over a lifetime to air containing 0.0002 µg/m^3. The toxicity value for non-cancer effects from oral exposure is called a reference dose (RfD), which is an estimate of the highest dose that can be taken in every day without causing adverse non-cancer health effects. To illustrate how the RfD is applied, a 150-pound (lb) person could safely ingest 0.02 mg arsenic every day without expecting any adverse effects (2.2 lb = 1 kg, or 1,000 g, or 1 million mg). A toxicity value for non-cancer effects following inhalation exposure to arsenic is not available.

Chemical Toxicity Values		
Cancer Risk		**Non-Cancer Effect**
Inhalation UR	***Oral SF***	***Oral RfD***
4.3 per mg/m^3	1.5 per mg/kg-day	0.0003 mg/kg-day

What Are Current Limits for Environmental Releases and Human Exposure? To help track facility releases to the environment, the Superfund amendments addressing emergency planning and community right-to-know require immediate reporting of a release of 1 lb (0.454 kg) or more of any arsenic compound that occurs in a 24-hour period, and also require normal releases to be reported annually and entered into a nationwide Toxics Release Inventory. For drinking water, EPA revised its maximum contaminant level of 0.05 mg/L to 0.01 mg/L in 2001; public water systems are to comply with this limit by January 2006. For air, the Occupational Safety and Health Administration has established limits of 0.01 mg/m^3 for inorganic and organic arsenic compounds.

Where Can I Find More Information? More information on arsenic can be found in the primary information source for this overview, the Toxicological Profile for Arsenic, prepared by the Agency for Toxic Substances and Disease Registry (ATSDR) and available at http://www.atsdr.cdc.gov/toxpro2.html. Other web-based sources of information include the ATSDR ToxFAQs (http://www.atsdr.cdc.gov/toxfaq.html), the EPA's Integrated Risk Information System (http://www.epa.gov/iris/subst/index.html), and the National Library of Medicine Hazardous Substances Data Bank (http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB).



Beryllium

What Is It? Beryllium is a hard, grayish metal that occurs naturally as a component of certain rocks, soil, coal, oil, and volcanic dust. Beryllium minerals have been known since ancient times as the gemstones emerald, aquamarine, and beryl. Compounds of beryllium are either white or colorless and do not have a particular smell. Because it is an element, beryllium does not degrade nor can it be destroyed.

Symbol:	**Be**
Atomic Number: *(protons in nucleus)*	**4**
Atomic Weight:	**9**

How Is It Used? Beryllium is used primarily in metal alloys (mainly with copper) for instruments, aircraft parts, springs, electrical connectors, and other industrial components. It is also incorporated into ceramics used in electrical insulators, microwave ovens, and rocket nozzles. Pure beryllium metal is used in missile and rocket parts, aircraft, heat shields, mirrors, and nuclear weapons.

What's in the Environment? The concentration of beryllium in the earth's crust generally ranges from 1 to 15 milligrams per kilogram (mg/kg), or parts per million (ppm). The average concentration of naturally occurring beryllium in U.S. soils is 0.6 ppm, and levels typically range from 0.1 to 40 ppm. Concentrations in sandy soil are estimated to be up to 250 times higher than in interstitial water (the water in the pore space between the soil particles), with much higher concentration ratios in loam and clay soils.

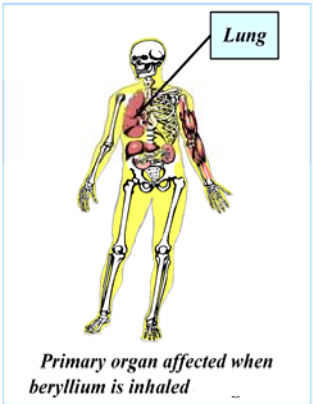
Beryllium naturally enters waterways through the weathering of rocks and soils that contain this metal. It can also be released to surface waters from industrial waste discharges. Beryllium levels in drinking water range from 0.01 to 0.7 parts per billion (ppb). It is also naturally present in various foods, with a median concentration of 22.5 µg/kg reported across 38 different food types, ranging from less than 0.1 µg/kg to 2,200 µg/kg (in kidney beans). One cigarette contains about 0.5 to 0.7 µg beryllium, with about 5 to 10% escaping into sidestream smoke.

The major source of environmental releases from human activities is combustion of coal and fuel oil. Air concentrations of beryllium are typically less than 0.0005 microgram per cubic meter ($\mu g/m^3$). The form usually released to the atmosphere is beryllium oxide, which eventually falls on land or water either in rain and snow or as dry particles. This oxide does not dissolve easily and does not move readily in soil or water, and it is considered unlikely to accumulate in plants and animals. The typical ratio of the beryllium concentration in plants to that in soil estimated to be low, at 0.0015 (or 0.15%).

What Happens to It in the Body? Beryllium can enter the body by eating food, breathing air, or smoking a cigarette. Children, and to a lesser extent adults, can also be exposed to a limited extent by ingesting soil. The internal fate of beryllium depends on the form that enters the body. Most beryllium compounds do not dissolve easily and are not well absorbed (less than 1%) from the gastrointestinal tract, so they are generally excreted in the feces. Dermal absorption is also expected to be very low, although information is limited. The beryllium that is absorbed is excreted very slowly, and it tends to accumulate in bone as well as other tissues and organs. Dusts and fumes containing beryllium can be inhaled and deposited in the lungs. Some deposited particles can clear from the lungs slowly, and soluble beryllium compounds can be converted to less soluble compounds. The biological half-life of inhaled, soluble beryllium compounds is about 2 to 8 weeks. Inhaled beryllium is excreted mainly in the urine.

What Are the Primary Health Effects? Inhalation of beryllium can result in two types of respiratory disease, acute beryllium disease and chronic beryllium disease (also referred to as berylliosis). Both forms can be fatal. The acute disease usually occurs after exposure to high levels (more than 1 mg/m^3) of the relatively soluble forms of beryllium, with symptoms ranging from inflammation of the nasal passages to severe chemical pneumonia. Some people can get chronic beryllium disease from breathing low levels, occurring in less than 15% of those exposed to more than 0.0005 mg/m^3. This disease is a type of immune

response only observed in sensitized individuals, and it involves the formation of granuloma and development of fibrosis of the lung. There can be a protracted latency period (up to 25 years) before the onset of any symptoms following exposure. In contrast, ingesting beryllium has generally not been reported to cause effects in humans because very little is absorbed into the body. Contact dermatitis is the most common effect of beryllium on the skin, and contact with scraped or cut skin can cause rashes or ulcers. In its current narrative for the cancer weight of evidence, the U.S. Environmental Protection Agency (EPA) describes beryllium as a likely human carcinogen for the inhalation pathway and states that the carcinogenic potential of ingested beryllium cannot be determined. Under the previous 1996 cancer guidelines, EPA classified beryllium as a probable human carcinogen.



Primary organ affected when beryllium is inhaled

What Is the Risk? The EPA has developed toxicity values (see box below) to estimate the risk of getting cancer or other adverse health effects as a result of inhaling or ingesting beryllium. The toxicity value for estimating the risk of getting cancer following inhalation exposure is called an inhalation unit risk (UR), which is an estimate of the chance that a person will get cancer from continuous exposure to a chemical in air at a unit concentration of 1 mg/m^3. The toxicity value for non-cancer effects from inhalation exposure is called a reference concentration (RfC), which is an estimate of the highest concentration in air that could be breathed every day without causing an adverse effect. The value for evaluating the possibility of non-cancer effects from oral exposure is the reference dose (RfD). The UR and RfC for beryllium are based on studies of humans exposed to beryllium in the workplace, and the RfD was developed by studying test animals given relatively high doses over their lifetimes, then adjusting and normalizing those results to a mg/kg-day basis for humans. To illustrate how the UR is applied, the EPA estimates that a person would have a one-in-a-million chance of developing cancer if exposed to air containing beryllium at a concentration of 0.0004 $\mu g/m^3$ every day over a lifetime. (A microgram is one millionth of a gram.) Using the RfD, it is estimated that 150-pound (lb) person could safely ingest 0.14 mg of beryllium every day without experiencing any adverse effects (2.2 lb = 1 kg, or 1,000 g, or 1 million mg).

Chemical Toxicity Values		
Cancer Risk	**Non-Cancer Effect**	
Inhalation UR	***Oral RfD***	***Inhalation RfC***
2.4 per mg/m^3	0.002 mg/kg-day	0.00002 mg/m^3

What Are Current Limits for Environmental Releases and Human Exposure? To help track facility releases to the environment, the Superfund amendments that address emergency planning and community right-to-know require immediate reporting of a release of 10 lb (4.54 kg) or more of any beryllium compound that occurs within a 24-hour period, and also require normal releases to be reported annually and entered into a nationwide Toxics Release Inventory. For drinking water supplies, the EPA has established a maximum beryllium level of 4 µg/liter. For workers exposed over regular work days, the National Institute for Occupational Safety and Health (NIOSH) and the Occupational Safety and Health Administration (OSHA) have established protective levels of 0.0005 and 0.002 mg/m^3 of beryllium and beryllium compounds, respectively. In addition, the EPA restricts industrial releases of beryllium to 10 g in a 24-hour period or an amount that would result in atmospheric levels of 0.01 $\mu g/m^3$ or less averaged over 30 days.

Where Can I Find More Information? More information on beryllium can be found in the primary information source for this overview, the Toxicological Profile for Beryllium, prepared by the Agency for Toxic Substances and Disease Registry (ATSDR) and available on the Internet at http://www.atsdr.cdc.gov/toxpro2.html. Other web-based sources of information include the ATSDR ToxFAQs (http://www.atsdr.cdc.gov/toxfaq.html), EPA's Integrated Risk Information System Database (http://www.epa.gov/iris/subst/index.html), and the Hazardous Substances Data Bank (http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB).

Chromium

What Is It? Chromium is found naturally in rocks, soil, plants, and animals, including people. It occurs in combination with other elements as chromium salts, some of which are soluble in water. The pure metallic form rarely occurs naturally. Chromium does not evaporate, but it can be present in air as particles. Because it is an element, chromium does not degrade nor can it be destroyed.

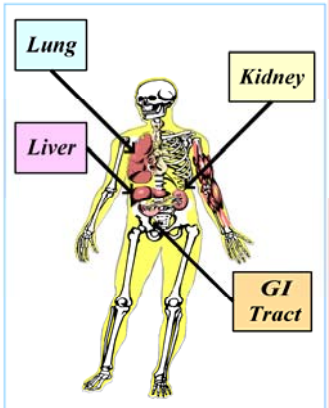
Symbol:	**Cr**
Atomic Number: *(protons in nucleus)*	**24**
Atomic Weight:	**52**

How Is It Used? Chromium is used to make steel and other alloys, for chrome plating, and as an additive to limit corrosion. Named for its colored compounds, chromium has also been used to make dyes and pigments for paints, and to make bricks in furnaces, tan leather, and preserve wood.

What's in the Environment? Chromium is present everywhere and can be found in three forms: metal ore, trivalent chromium (Cr III), and hexavalent chromium (Cr VI). The trivalent form occurs naturally in many fresh vegetables and fruits, meat, grains, and yeast. Relatively insoluble, it is the most prevalent form in surface soils where oxidation processes (which convert chromium from the hexavalent to trivalent form) are most common. Hexavalent chromium also occurs naturally, notably in water-saturated (reducing) conditions, and it is an indicator of human pollution. This form is relatively soluble and can move more readily through soil to groundwater. The concentration of naturally occurring chromium in U.S. soil ranges from 1 to 2,000 parts per million (ppm), with an average of 54 ppm. The concentration in sandy soil particles is estimated to be 70 times higher than in interstitial water (the water in the pore spaces between the particles), and concentration ratios are higher (e.g., 1,500) for clay soil. The typical ratio of chromium in plants to chromium in soil is estimated at 0.0045 (or 0.45%).

Chromium concentrations in air and water are very low. In air, concentrations generally range between 0.01 and 0.03 microgram per cubic meter ($\mu g/m^3$), and drinking water levels are generally less than 2 parts per billion (ppb). Absent information on the specific form, a general assumption often made for chromium that has been present in surface soil over time is that 10 to 17% of the total is in the hexavalent form. Groundwater can be contaminated at certain industrial facilities where sodium dichromate solutions (hexavalent) were used to prevent corrosion in piping. For groundwater, it is often assumed that most is hexavalent.

What Happens to It in the Body? Chromium can be taken in by breathing air, drinking water, or eating food. Children, and to a lesser extent adults, can also be exposed by ingesting soil. It can also be absorbed through the skin to a limited extent. Hexavalent chromium is more readily absorbed than trivalent chromium, regardless of the route of exposure. When air containing chromium is inhaled, chromium particles can be deposited in the lungs. Those deposited in the upper part of the lungs are usually coughed up and swallowed. Some that deposit deep in the lungs can dissolve, which allows chromium to pass through the lining of the lungs and enter the bloodstream. The finding of toxic effects following dermal exposure suggests that chromium is absorbed through the skin, although information on the percent absorbed is limited. Once in the bloodstream, chromium moves to all parts of the body. It is not metabolized, but hexavalent chromium is reduced by enzymatic reactions to trivalent chromium in the body. Inhaled chromium is excreted both in the urine and the feces. Ingestion of food is the major source of chromium exposure for most people in the United States. On average, adults take in an estimated 60 µg of trivalent chromium every day with their food. If taken in as hexavalent chromium, it is rapidly converted to the trivalent form after entering the stomach. When swallowed, most chromium leaves the body within a few days through the feces. A small amount – about 0.5% of Cr III and 10% of Cr VI – will pass through the lining of the intestines and enter the bloodstream. From there, chromium is distributed to all parts of the body. It then passes through the kidneys and is eliminated in the urine in a few days. The trivalent form in food can attach to other compounds that make it easier for chromium to be absorbed and enter the bloodstream from the stomach and intestines.



Primary organs affected when chromium is taken in through food, water, or air.

What Are the Primary Health Effects? The trivalent form of chromium is an essential nutrient in our diet and is needed for many important functions, including lipid, protein, and fat metabolism. Even at levels above those required to maintain health, it exhibits very low toxicity and it is not known to cause cancer. In contrast, hexavalent chromium can be toxic, including causing cancer if it is inhaled; the lethal dose is estimated at about 7- milligrams hexavalent chromium per kilogram (mg/kg) body weight. When inhaled, hexavalent chromium can damage the lining of the nose and throat, and irritate the lungs as well as the gastrointestinal tract. Nasal irritation has been observed following acute exposure at levels less than 0.01 mg/m^3. When swallowed, it can upset the stomach and damage the liver and kidneys. Some people have an allergic skin reaction after touching material containing chromium. Hexavalent chromium is one of a small set of chemicals the U.S. Environmental Protection Agency (EPA) has classified as a known human carcinogen, based on studies of workers in chromium processing factories who developed lung cancer after chronic inhalation exposures. However, hexavalent chromium does not cause cancer when ingested, most likely because it is rapidly converted to the trivalent form after entering the stomach. Information on joint toxicity with other chemicals is provided in the companion chemical mixtures fact sheet.

What Is the Risk? The EPA has developed toxicity values *(see box below)* to estimate the risk of getting cancer or experiencing other adverse health effects as a result of inhaling or ingesting chromium. The toxicity value for estimating the risk of getting cancer following inhalation exposure is called an inhalation unit risk (UR), which is an estimate of the chance that a person will get cancer from continuous exposure to a chemical in air at a unit concentration of 1 mg/m^3. A reference dose (RfD) is an estimate of the highest dose that can be taken in every day without causing an adverse non-cancer effect, and a reference concentration (RfC) is an estimate of the highest concentration in air that could be breathed every day without causing an adverse effect. These toxicity values have been developed by studying test animals given relatively high doses over their lifetimes, then adjusting those results to a mg/kg-day basis for humans, or directly from studies of humans exposed to chromium in the workplace. To illustrate, a 150-pound (lb) person could safely ingest 100 mg (about three ounces) of trivalent chromium or 0.2 mg of hexavalent chromium every day without expecting any adverse effects. Using the UR, EPA estimates that a person would have a one-in-a-million chance of developing cancer if continuously exposed for a lifetime to air containing 0.00008 $\mu g/m^3$ hexavalent chromium, as particulates.

Chemical Toxicity Values

Form of Chromium	Cancer Effect	Non-Cancer Effect	
	Inhalation UR	*Oral RfD*	*Inhalation RfC*
Cr III	None established	1.5 mg/kg-day	None established
Cr VI	12 per mg/m^3	0.003 mg/kg-day	0.0001 mg/m^3 *(as particulates)* 0.000008 mg/m^3 *(as chromic acid mists and dissolved Cr VI aerosols)*

What Are Current Limits for Environmental Releases and Human Exposures? To help track facility releases to the environment, the Superfund amendments that address emergency planning and community right-to-know require the immediate reporting of releases of 10 lb (4.54 kg) or more of chromic acid and 1,000 lb (454 kg) or more for all other regulated chromium compounds that occur within a 24-hour period, and also require normal releases to be reported annually and entered into a nationwide Toxics Release Inventory. The EPA has established a maximum contaminant level in drinking water of 0.1 ppm. The Occupational Safety and Health Administration has established protective levels of 0.5 and 1 mg/m^3 in air for water-soluble and -insoluble trivalent compounds, and 0.1 mg/m^3 for hexavalent compounds.

Where Can I Find More Information? More details can be found in the primary information source for this overview, the Toxicological Profile for Chromium, prepared by the Agency for Toxic Substances and Disease Registry (ATSDR) and available at http://www.atsdr.cdc.gov/toxpro2.html. Other web-based sources of information include the ATSDR ToxFAQs (http://www.atsdr.cdc.gov/toxfaq.html), EPA's Integrated Risk Information System (http://www.epa.gov/iris/subst/index.html), and the National Library of Medicine Hazardous Substances Data Bank (http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB).

Copper

Symbol:	**Cu**
Atomic Number: *(protons in nucleus)*	**29**
Atomic Weight:	**64**

What Is It? Copper is a reddish metal that can be easily molded or shaped. It is an essential element for all living things, including humans. Because copper is an element, it does not degrade nor can it be destroyed.

How Is It Used? Copper is extensively mined and processed in the United States. It is primarily used as the metal or alloy in electrical wiring, sheet metal, pipes, and other metal products, including pennies (which are made with copper and zinc). Copper compounds are also commonly used in farming to treat plant diseases like mildew. In addition, they are used to treat water to eliminate algae, and as a preservative for wood, leather, and fabrics.

What's in the Environment? Copper is very common in the environment. It occurs naturally in rock, soil, water, sediment, and air, as well as in plants and animals. Its average concentration in the earth's crust is about 50 parts per million (ppm). Soil concentrations commonly range from 2 to 100 ppm and up to 250 ppm, but concentrations as high as 7,000 ppm have been found near copper production facilities. High soil concentrations also occur in areas where waste from sewage treatment plants or mining and other copper industries are disposed of on the soil. Considerable data indicate that copper does not biomagnify in the food chain. The typical ratio of the concentration of copper in plants to that in soil is estimated at 0.25 (or 25%). Most plants contain less than 10 ppm copper on a dry-weight basis, and concentrations in animal foods are 2 to 4 ppm, with dairy products containing less than 1 ppm.

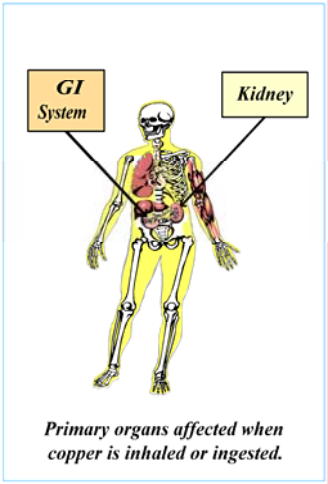
Copper is prevalent in surface water throughout the United States, with an average concentration in lakes and rivers of 4 parts per billion (ppb). Copper in aquatic systems can strongly attach to suspended particles and sediment, and it also converts to forms that are not easily absorbed after uptake by organisms. The average concentration in groundwater is similar to that in surface water, with higher levels in areas with higher naturally occurring copper in geologic materials. Although it can leach through somewhat acidic, sandy soil, copper is relatively immobile in most soils. It binds more strongly to soil particles than do other divalent cations, and it precipitates out of soil water by forming solids with ions such as carbonate and hydroxide. Concentrations of copper in soil particles can range between 35 and 100 or more times higher than concentrations in water in the pore spaces between the particles. Copper is also present in drinking water, with U.S. concentrations generally ranging from 20 to 75 ppb. However, many households with older plumbing have concentrations above 1,000 ppb (or 1 ppm) because copper in the pipes and brass faucets dissolves into the water as it sits overnight.

What Happens to It in the Body? Exposure to copper can occur by breathing air, drinking water, eating food, and to a limited degree, by skin contact with soil or water. Children, and to a lesser extent adults, can also be exposed by ingesting soil. Absorption of copper following ingestion is normally regulated by homeostatic mechanisms so that the balance between copper intake and excretion is controlled. Studies of humans indicate that, in general, about 50% of ingested copper is absorbed into the bloodstream, although reported values range from 15 to 97%. Water-soluble forms of copper are more readily absorbed than insoluble forms. Zinc, molybdenum, and some other metals can decrease dietary copper absorption. Most absorbed copper is transported to the liver, with minor amounts going to bone and other tissues. Although it is known that copper can enter the body through the lungs or skin, information on the amount absorbed is limited. Most copper is excreted in the bile and feces, with only 2 to 4% of absorbed copper excreted in the urine. It may take several days for copper to leave the body, although studies have shown that injected copper has a half-life in the body of about 4 weeks.

What Are the Primary Health Effects? In humans, copper is necessary for overall good health. People in the United States are estimated to take in 1 to 10 milligrams (mg) or more each day in their diets. The National Academy of Sciences has recommended 2 to 3 mg of copper as a safe and adequate daily intake for

adults. Copper has been shown to have a protective effect against cadmium poisoning, and people who do not have enough copper in their diet can be more susceptible to adverse effects from lead.

Drinking water with concentrations of 30 ppm or greater can cause vomiting, diarrhea, stomach cramps, and nausea. Large intakes can cause liver or kidney damage, or even death in cases of extreme exposure. People with Wilson's disease have a genetic defect that results in the accumulation of copper in tissues, including the liver, kidney, and cornea. The excess copper in these people can cause damage to the kidney, liver, and brain; hemolytic anemia; and other effects.



Primary organs affected when copper is inhaled or ingested.

Acute exposure via inhalation of copper dusts or fumes can cause a condition called "metal fume fever," characterized by chills, fever, dry throat and aching muscles. Long-term exposure to copper dust in air can irritate the nose, mouth, and eyes, and cause headaches, dizziness, nausea, and diarrhea. Adverse effects on the lungs of animals have been reported at concentrations of 0.1 to 3 ppm in air. In some people, skin contact with copper can result in an allergic reaction that is expressed as a skin irritation or rash. No data exist indicating that copper can cause cancer, and the International Agency for Research on Cancer (IARC) has stated that copper is not a human carcinogen. Similarly, data do not exist to indicate that copper can cause birth defects in humans.

What Is the Risk? The U.S. Environmental Protection Agency (EPA) develops toxicity values to estimate the risk of non-cancer health effects from ingesting chemicals. The toxicity value used to estimate a non-cancer effect is called a reference dose (RfD), which is an estimate of the highest dose that can be taken in every day without causing an adverse non-cancer effect. For copper, no formal RfD has been developed. (And as explained above, there is no indication of cancer risk from copper exposures; thus no cancer slope factor has been defined for this metal.) For comparison, a reference value could be derived from the maximum contaminant level goal (MCLG) identified for drinking water (see next section), although this level is not based on health effects. To illustrate how this could be applied, if it is assumed that a 150-lb person drinks 2 liters of water every day containing a given amount of copper, the amount corresponding to the MCLG would be 2.5 mg (2.2 lb = 1 kg, or 1,000 g).

What Are Current Limits for Environmental Releases and Human Exposure? To protect against the aquatic organisms (such as fish), the EPA established a water quality criterion of 1 ppm for copper in lakes and streams. The EPA has identified an MCLG of 1.3 ppm for copper in drinking water supplies, which serves as an action level such that if more than 10% of tap water samples exceed this value, additional steps are taken to improve water quality. (Copper is regulated by a treat technique approach that considers corrosiveness.) For air, the Occupational Safety and Health Administration (OSHA) has established limits of 0.2 milligram per cubic meter (mg/m^3) of copper fume (vapor generated from heating copper) and 1.0 mg/m^3 of copper dusts (fine metallic copper particles) and mists in workroom air, to protect workers during an 8-hour workday over a 40-hour work week. The National Institute for Occupational Safety and Health (NIOSH) recommends that average concentrations in workplace air be limited to 0.1 mg/m^3 for copper fumes and 1.0 mg/m^3 for copper mist.

Where Can I Find More Information? More information on copper can be found in the primary information source for this overview, the Toxicological Profile for Copper, prepared by the Agency for Toxic Substance and Disease Registry (ATSDR) and available on the Internet at http://www.atsdr.cdc.gov/toxpro2.html. Other web-based sources of information include the ATSDR ToxFAQs (http://www.atsdr.cdc.gov/toxfaq.html) and the Hazardous Substances Data Bank (http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB).

Lead

Symbol:	**Pb**
Atomic Number: *(protons in nucleus)*	**82**
Atomic Weight:	**207**

What Is It? Lead is an element found naturally in rocks, soil, plants and animals. It typically occurs in combination with other elements as lead salts, some of which are soluble in water. The pure metallic form of lead is bluish-gray, but metallic lead rarely occurs naturally. Lead does not evaporate, but it can be present in air as particles. Because it is an element, lead does not degrade nor can it be destroyed. Several radioactive isotopes are naturally present in the environment, with lead-210 being the isotope of most concern. (Information on radioactive isotopes is presented in the companion fact sheets for radium, thorium, and natural decay series.)

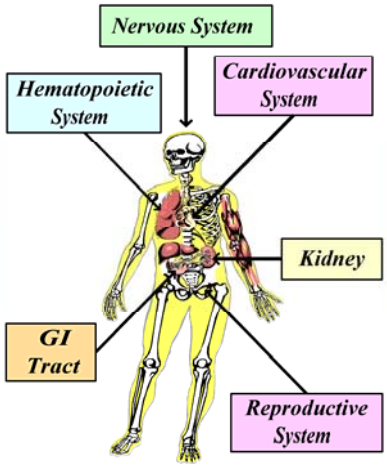
How Is It Used? Lead has been used for thousands of years for a variety of purposes. Today, its major use is in the production of certain types of batteries. Lead is also used to make ammunition, metal products (sheet metal, solder, and pipes), medical equipment (radiation shields and surgical equipment), paints, ceramic glazes, caulking, scientific equipment (circuit boards for computers), and high-precision glass for lasers and other optical equipment. In recent years, the amount used in products such as paints and ceramics has decreased significantly to help minimize exposures of people and animals. Tetraethyl and tetramethyl lead (volatile organic forms) were used for many years in gasoline to increase octane rating. In the United States, this use was phased out during the 1980s, and lead was banned from use in gasoline for transportation in 1996.

What's in the Environment? Lead occurs everywhere in the environment. Concentrations in U.S. soil typically range from less than 10 to 30 milligrams of lead per kilogram of soil (mg/kg). However, amounts in the top layers vary widely and can be much higher due to human activities. For example, concentrations near roadways can be 30 to 2,000 mg/kg higher than natural levels due to past use of leaded gasoline. In air, concentrations typically range from 0.001 to 0.002 microgram per cubic meter ($\mu g/m^3$) in remote areas and from 0.2 to 0.4 $\mu g/m^3$ in urban areas. Levels in surface water and groundwater typically range from 5 to 30 µg/liter. Lead is relatively immobile in soil but can leach to groundwater over time. Concentrations in sandy soil particles are estimated to be 270 times higher than in the water in pore spaces between the soil particles; it binds even more tightly to clay and loam soils, with concentration ratios of about 500 to more than 16,000. Reported concentrations of lead in various foods range from 0.002 to 0.65 mg/kg, with higher levels generally found in vegetables. The typical ratio of the concentration of lead in plants to that in the soil on which they grow is estimated at roughly 0.04 (or 4%).

What Happens to It in the Body? Lead can be taken in by eating food, drinking water, or breathing air. Children, and to a lesser extent, adults, can also be exposed by ingesting soil. Lead can also be absorbed through the skin, although this is usually a less important route of exposure. If air containing lead particles is inhaled, particles deposited in the lungs can lead to about 90% being absorbed. Particles deposited in the upper parts of the lung are usually coughed up and swallowed, while those deposited deep in the lungs can dissolve, allowing lead to enter the bloodstream. If lead is swallowed with food, the amount absorbed into the bloodstream is about 10 to 15% in a typical adult; however, about 60 to 80% is absorbed in adults who have not eaten for a day. In general, if adults and children ingest the same amount of lead, children will absorb a higher percentage (about 50%). After lead enters the bloodstream, it travels to three main compartments: blood, soft tissue, and bone. About 95% and 73% of lead in the body is stored in bones and teeth for adults and children, respectively. Lead has a half-life in blood of about 1 month, whereas lead in bone has a half-life of greater than 20 years. Inorganic lead is not metabolized in the body, but it can be conjugated with glutathione. About 75% of absorbed lead is excreted in urine and about 25% in feces; lead can also be excreted in breast milk.

What Are the Primary Health Effects? Lead can affect almost every organ and system in the body, including the gastrointestinal tract, the hematopoietic system (blood-forming tissues), cardiovascular system, central and peripheral nervous systems, kidneys, immune system, and reproductive system. Young and unborn children can be extremely sensitive. Exposure of pregnant women to high levels of lead can result in premature births and smaller babies, followed by learning difficulties and reduced growth. The latter effects are also seen in young children exposed to lead after birth, as are effects on other organ systems. In adults, peripheral nerve damage has been observed at 40 to 60 micrograms of lead per deciliter of blood (µg/dL) anemia at 80 µg/dL,

and encephalopathy at 100 µg/dL. Although studies indicate that lead acetate and lead phosphate cause cancer in laboratory animals, we do not know if lead can cause cancer in humans after being ingested or inhaled. On the basis of the animal studies, the U.S. Environmental Protection Agency (EPA) has classified lead as a probable human carcinogen. The joint toxicity of lead with other chemicals, including essential nutrients, has been studied more extensively than for most chemicals. Depending on the endpoint and chemical, the joint toxicity can be additive, higher than additive, or less than additive. For example, higher toxicity to the nervous system is predicted in combination with arsenic, cadmium, or manganese. In contrast, kidney toxicity is predicted to be less than additive for these same metal pairs. Zinc can protect against lead toxicity by reversing its enzyme-inhibiting effects, whereas iron deficiency appears to increase the gastrointestinal absorption of lead leading to increased toxicity to the hematopoietic system as well as other effects. Additional information is provided in the companion chemical mixtures fact sheet.



Organs and systems affected when lead is taken in through food, water, or air.

What Is the Risk? Unlike most other chemicals, the potential for adverse health effects from inorganic lead is based on predicted or measured levels of lead in blood rather than on toxicity values. The EPA has developed a mathematical model (the Integrated Exposure Uptake Biokinetic Model, IEUBK), to predict concentrations of lead in the blood of children resulting from exposure to lead in soil, air, drinking water, food, and other sources. Predicted blood-lead concentrations are compared to a concentration of 10 µg/dL to evaluate the health risk to children. Using the IEUBK Model, the EPA estimated that the blood concentration of lead in children could exceed 10 µg/dL if the concentration of lead in soil at residences exceeds 400 mg of lead per kg of soil. Similarly, EPA used the Adult Lead Model to predict a soil concentration of 800 mg/kg that would be protective of the fetus of a female worker in an occupational setting; this concentration would also be protective for male or female adult workers. Although certain lead compounds have been shown to cause cancer in animals, the risk of cancer is not typically evaluated for lead because people are more sensitive to its non-cancer effects. (For radioactive isotopes of lead, the cancer risks are included in the risks for radium and thorium, as indicated in those companion fact sheets.) The organic compound tetraethyl lead is very toxic, and the potential for non-cancer effects from exposure to this form is evaluated using toxicity values, similar to the approach used for other chemicals. The toxicity value for evaluating oral exposure is the reference dose (RfD), which is an estimate of the highest dose that can be taken in every day without causing an adverse non-cancer effect. The RfD for tetraethyl lead is 0.0000001 mg/kg-day. It was developed by studying test animals given relatively high doses and then adjusting those results to a mg/kg-day basis for humans. A toxicity value for assessing non-cancer effects of tetraethyl lead following inhalation exposure is not available.

What Are Current Limits for Environmental Releases and Human Exposures? To help track facility releases to the environment, the Superfund amendments addressing emergency planning and community right-to-know require that releases of eleven lead compounds to air, water, or land be reported annually and entered into a nationwide Toxics Release Inventory. For lead arsenate, a release of over 1 lb (0.454 kg) must be reported immediately, while the quantity for the other lead compounds is 10 lb (4.54 kg). The EPA requires that lead in air not exceed 1.5 µg/m^3 averaged over three months. The drinking water action level for lead is 15 µg/L. The EPA has defined hazardous concentrations of lead as: 40 µg per square foot (ft^2) in dust on floors and 250 µg/ft^2 for interior windowsills of homes; 400 mg/kg in bare soil in children's play areas; and 1,200 mg/kg in bare soil in other parts of the yard. For the workplace, the Occupational Safety and Health Administration has established a permissible exposure limit (PEL) of 0.1 mg/m^3 for metallic lead and 0.05 mg/m^3 for lead from soluble compounds. If the concentration of lead in the blood of a worker exceeds 50 µg/dL, the worker is not allowed to remain in that work area. Many other regulations and recommendations have been developed for lead to protect public health.

Where Can I Find More Information? More information can be found in the primary information source for this overview: the Toxicological Profile for Lead, prepared by the Agency for Toxic Substances and Disease Registry (ATSDR) and available at http://www.atsdr.cdc.gov/toxpro2.html). Other web-based sources of information include the ATSDR ToxFAQs (http://www.atsdr.cdc.gov/toxfaq.html), EPA's Integrated Risk Information System (http://www.epa.gov/iris/subst/index.html), and the National Library of Medicine Hazardous Substances Data Bank (http://www.toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB).

Mercury

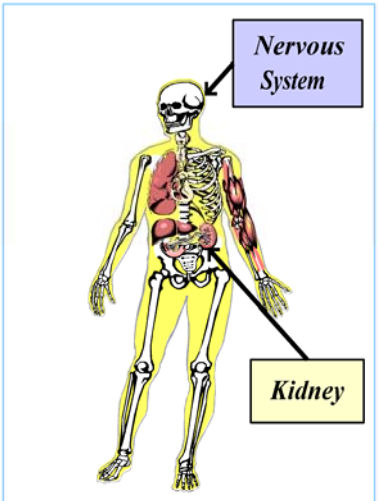
Symbol:	**Hg**
Atomic Number: *(protons in nucleus)*	**80**
Atomic Weight:	**201**

What Is It? Mercury is an element that occurs naturally in the environment, usually in combination with other elements as mercury compounds or salts. Metallic mercury is a shiny, silver-white metal that is a liquid at room temperature. If heated, it enters the atmosphere as a colorless, odorless gas. Mercury combines with other elements to form inorganic mercury compounds, some of which are soluble in water. It also combines with carbon to form organic mercury compounds, such as methylmercury. Because it is an element, mercury does not degrade nor can it be destroyed.

How Is It Used? Metallic mercury is used to produce chlorine gas and caustic soda and to extract gold from ores. It is used in thermometers, dental fillings, and batteries. Some inorganic mercury compounds are used in skin-lightening creams, as antiseptic creams and ointments, and as anti-mildew agents.

What's in the Environment? The most common forms of mercury that occur naturally in the environment are metallic mercury; the inorganic salts, mercuric sulfide and mercuric chloride; and methylmercury. Microorganisms and various natural processes can change these forms of mercury: metallic mercury can combine with other elements to form inorganic mercury compounds, and inorganic mercury compounds can combine with carbon to form organic mercury compounds which, in turn, can be changed to inorganic compounds. Methylmercury is the most common form created by these natural processes. This compound is of particular concern because it can bioaccumulate through the food chain. That is, small organisms and plants take up methylmercury as they feed, animals higher up the food chain eat these plants and organisms, and the process continues with methylmercury levels increasing up the food chain. Of particular concern is bioaccumulation in fish and shellfish, in which mercury concentrations in fish high in the food chain can be much higher than concentrations in the surrounding water or than in fish lower in the food chain. However, mercury does not bioconcentrate in terrestrial plants; the typical ratio of the concentration in plants to that in soil is estimated at 0.2 (or 20%). In the United States, the concentration of mercury in soil containing natural levels of mercury ranges from less than 0.02 to about 6 milligrams of mercury per kilogram of soil (mg/kg). Mercury leaches somewhat slowly, with the concentration in soil particles estimated to be about 10 to 100 times higher than in the water between the soil particles. In air, concentrations of mercury range from about 0.01 to 0.02 microgram per cubic meter ($\mu g/m^3$).

What Happens to It in the Body? Mercury can be taken into the body primarily by breathing air or eating food. How much mercury enters the body and what happens depends on the form of mercury and the route of exposure (that is, through air, food, or skin). When vapors of metallic mercury are inhaled, as much as 80% is absorbed. Following ingestion, absorption is less than 0.01% for metallic mercury, less than 10% for inorganic mercury, and greater than 95% for organic mercury. Mercury can also be absorbed through the skin, but the amount is small compared to breathing or swallowing it.



Organs and systems affected when mercury is taken in through food, water, or air.

Once in the body, metallic mercury and organic mercury compounds easily reach most tissues, including the brain; mercury also reaches the fetus of a pregnant woman. Mercury accumulates in the kidneys. In the brain, metallic mercury and methylmercury can be converted to an inorganic form that is then trapped in the brain. Mercury tends to stay in the body for weeks or months; elemental mercury and mercury vapor have a half-life of 35 to 90 days, mercury salts have a half-life of about 40 days, and methylmercury has a half-life of about 90 days. Eventually, mercury leaves the body through the urine and feces, but the ratio is dependent on the exposure circumstances. For example, urinary excretion accounts for 58% of the body burden following long-term exposure to inorganic mercury compared to only 13% of the body burden from short-term exposure.

What Are the Primary Health Effects? Exposure to high levels of mercury can damage the brain, kidneys, and developing fetus. The nervous system is very sensitive to all forms of mercury, although the brain is most sensitive to metallic mercury and methylmercury because they enter the brain more easily than inorganic mercury salts. Exposure can cause tremors; memory loss; and changes in personality, vision, and hearing. (As a note, the Mad Hatter in the book *Alice in Wonderland* displays personality changes noticed during the 1800s to early 1900s in people engaged in the hat-making business in which mercury was used to process leather.) Children and fetuses are particularly sensitive to the harmful effects of mercury on the nervous system; exposure can result in mild to severe brain damage, including effects on a child's behavior and ability to think and learn. Mercury also damages the kidneys. The essential element, selenium, may be protective against the toxic effects of mercury.

Although studies in animals indicate that mercuric chloride and methylmercury can cause cancer in laboratory animals, we do not know if they can cause cancer in humans ingesting these compounds or breathing them in air. On the basis of the animal studies, the Environmental Protection Agency (EPA) identifies both mercuric chloride and methylmercury as possible human carcinogens.

What Is the Risk? The EPA has developed toxicity values to estimate the risk of developing non-cancer effects as a result of ingesting or inhaling mercury *(see box below)*. The toxicity value for estimating non-cancer effects from oral exposure is called a reference dose (RfD), which is an estimate of the highest dose that can be taken in every day without causing an adverse non-cancer effect. The toxicity value for inhalation exposure is the reference concentration (RfC), which is an estimate of the highest concentration in air that can be breathed every day without causing an adverse effect. These toxicity values have been developed based on studies of workers exposed to mercury in occupational settings (metallic mercury), epidemiological studies of mothers and infants (methylmercury), and by studying test animals given relatively high doses of mercury over their lifetimes, then adjusting and normalizing those results to a mg/kg-day basis for humans (mercuric chloride and phenylmercuric acetate). To illustrate how the RfD may be applied, a 150-pound (lb) person could safely ingest 0.001 mg of methylmercury every day without expecting any adverse effects (2.2 lb = 1 kg, 1,000 g, or 1 million milligrams).

Chemical Toxicity Values

Form of Mercury	Non-Cancer Effect	
	Oral RfD	***Inhalation RfC***
Metallic mercury	None established	0.0003 mg/m^3
Mercuric chloride	0.0003 mg/kg-day	None established
Methylmercury	0.0001 mg/kg-day	None established
Phenylmercuric acetate	0.00008 mg/kg-day	None established

What Are Current Limits for Environmental Releases and Human Exposures? To help track facility releases to the environment, the Superfund amendments addressing emergency planning and community right-to-know require that releases of certain chemicals to air, water, or land be reported immediately if they exceed specified levels, and also require normal releases to be reported annually and entered into a nationwide Toxics Release Inventory. Several mercury compounds are regulated under those amendments. Immediately reportable quantities are 1 lb (0.454 kg) for mercury and mercuric cyanide; 10 lb (4.54 kg) for mercuric nitrate, mercuric sulfate, mercuric thiocyanate, mercurous nitrate, and mercury fulminate; and 100 lb (45.4 kg) for phenylmercuric acetate. The EPA has established a limit of 0.002 milligram per liter (mg/L) for mercury in drinking water. (Mercury is not regulated under the Clean Air Act.) For air in the workplace, the Occupational Safety and Health Administration has set a permissible exposure limit (PEL) for mercury vapor of 0.1 milligram per cubic meter (mg/m^3) as a ceiling limit, and 0.01 mg/m^3 for organo (alkyl) mercury compounds as an 8-hour time-weighted average. The Food and Drug Administration has set an action level in fish and shellfish of 1 mg of methylmercury per kg of fish.

Where Can I Find More Information? More information on mercury can be found in the primary information source for this overview, the Toxicological Profile for Mercury, prepared by the Agency for Toxic Substances and Disease Registry and available on the Internet at http://www.atsdr.cdc.gov/toxpro2.html. Other web-based sources of information include the ATSDR ToxFAQs (http://www.atsdr.cdc.gov/toxfaq.html), the EPA's Integrated Risk Information System (http://www.epa.gov/iris/subst/index.html), and the National Library of Medicine Hazardous Substances Data Bank (http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB).

Zinc

Symbol:	**Zn**
Atomic Number: *(protons in nucleus)*	**30**
Atomic Weight:	**65**

What Is It? Zinc is one of the most common elements in the earth's crust. It is also an essential element for all living things. Pure zinc is a bluish-white, shiny metal. Powdered zinc is explosive and can burst into flames if stored in a damp place. Because it is an element, zinc does not degrade nor can it be destroyed.

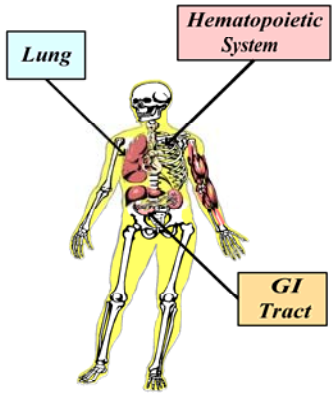
How Is It Used? Zinc has many commercial and industrial uses. Metallic zinc is used to coat iron and other metals to prevent rust, and it is also used in dry cell batteries. Zinc is mixed with other metals to form alloys such as brass and bronze, and pennies are made from a copper-zinc alloy. Zinc is also combined with other elements such as chlorine, oxygen, and sulfur to form zinc compounds used to make white paints, ceramics, rubber, wood preservatives, dyes, and fertilizers. Zinc compounds are also used in the drug industry as ingredients in common products like sun blocks, diaper rash ointments, deodorants, athlete's foot preparations, acne and poison ivy preparations, and anti-dandruff shampoos.

What's in the Environment? Zinc is found throughout the environment in air, soil, and water, and it is present in all foods. It can be released by natural processes, but most results from human activities. Releases to air, water, and soil are common in areas where ores are mined, processed, and smelted for zinc. Because cadmium and lead are commonly present in zinc-containing ores, they are also typically released during these processes and so areas are often jointly contaminated. Zinc can be releases to the atmosphere during the production of steel and burning of coal or waste. Surface water can be impacted by discharges of metal manufacturing and chemical industry wastes, and also by run-off following precipitation on soils high in zinc, either due to the natural setting or human applications, including use of zinc fertilizer on agricultural soils.

The average concentration of zinc in air (as fine dust particles) is typically less than 1 microgram per cubic meter (µg/m^3), although concentrations of 5 µg/m^3 have been measured near industrial sources. In lakes and rivers, some zinc remains dissolved in water or as fine suspended particles, while other zinc settles to the bottom in association with heavier particles. Average concentrations range from 0.02 to 0.05 milligram per liter (mg/L) in surface water and 0.01 to 0.1 mg/L in drinking water. Levels U.S. soils typically range from 10 to 300 mg/kilogram (kg), with an average concentration of about 50 mg/kg. Zinc generally remains in the upper layers bound to soil particles, but it can leach to groundwater depending on soil characteristics, moving more readily in sandy soil. Concentrations of zinc in sandy soil particles are about 200 times higher than in the water between the soil particles, and concentration ratios are even higher (over 1,000) in both loam and clay soils.

Some fish may accumulate zinc, but it does not build up in plants. The typical ratio of the concentration in plants to that in soil is estimated at 0.9 (or 90%). Zinc has been measured in food at concentrations ranging from 2 parts per million (ppm) in leafy green vegetables to 29 ppm in meat, fish, and poultry. On average, people ingest 7 to 163 mg of zinc every day.

What Happens to It in the Body? Zinc is one of the most abundant trace elements in the human body. It is typically taken in by ingestion of food and water, although it can also enter the lungs by inhaling air, including that contaminated with zinc dust or fumes from smelting or welding activities. The amount of zinc that can pass directly through the skin is very small. Absorption of zinc into the bloodstream following ingestion is normally regulated by homeostatic mechanisms, so the balance between zinc intake and excretion is controlled. Absorption from the gastrointestinal tract is 20 to 30% in people with diets containing adequate levels of zinc, but it can reach 80% in those with low levels of zinc in their diets or body tissues. Zinc is normally excreted in the urine and feces, with small amounts excreted in sweat. About 90% of what is retained is found in muscles and bones.



Primary organs affected when zinc is inhaled or ingested.

What Are the Primary Health Effects? Zinc is an essential element in our diet, but too little or too much can be harmful. Without enough dietary intake, people can experience a loss of appetite, decreased sense of taste and smell, decreased immune function, slow healing of wounds, and skin sores. Too little zinc can also result in poorly developed sex organs and retarded growth in young men. If pregnant women do not have enough zinc, babies might have growth retardation. Harmful effects from too much zinc generally begin at levels from 10 to 15 times higher than the recommended dietary allowances of 5, 12, and 15 mg per day for infants, women, and men, respectively. Eating large amounts of zinc can cause stomach cramps, nausea, and vomiting. Taking in large amounts of zinc over an extended period can cause anemia, damage the pancreas, and lower the levels of high-density lipoprotein cholesterol (the good form of cholesterol).

Breathing dust or fumes containing large amounts of zinc can cause a short-term disease called metal fume fever. This disease is an immune response affecting the lungs and body temperature. It is not known if there are health effects from breathing lower levels of zinc over long periods of time. It is also not known if high levels of zinc affect human reproduction or cause birth defects. However, infertility, low birth weight, and skin irritation have been observed in laboratory animals such as rats, guinea pigs, mice, and rabbits given high doses of zinc.

The U.S. Environmental Protection Agency (EPA) has stated that adequate information to evaluate the carcinogenicity of zinc is not available. However, no studies exist that indicate zinc causes cancer in humans.

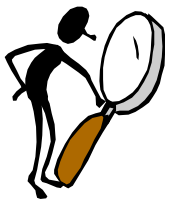
Zinc deficiency may increase the toxic effects of arsenic, copper, cadmium and lead; thus an adequate amount of zinc can be considered protective against the toxicity of these elements. However, too much zinc can increase the absorption of lead, which has been shown to have an additive effect on the hematological effects of zinc.

What Is the Risk? The EPA has developed a toxicity value called a reference dose (RfD) *(see box below)* to estimate the risk of adverse health effects as a result of ingesting zinc. An RfD is an estimate of the highest dose that can be taken in every day without causing an adverse non-cancer effect. This toxicity value has been developed from clinical studies in humans given dietary supplements of zinc. To illustrate how the RfD is applied, a 150-pound (lb) person could safely ingest 21 mg of zinc every day without expecting any adverse effects (2.2 lbs = 1 kg, or 1,000 g, or 1 million mg).

Chemical Toxicity Value
Non-Cancer Effect: *Oral RfD*
0.3 mg/kg-day

What Are the Current Limits for Environmental Releases and Human Exposure? To help track facility releases to the environment, the Superfund amendments that address emergency planning and community right-to-know require the immediate reporting of a release of 1,000 lb (454 kg) or more of any zinc compound that occurs within 24 hours, and also require normal releases to be reported annually and entered into a nationwide Toxics Release Inventory. For drinking water, EPA has established a maximum contaminant level of 5 ppm for zinc and zinc compounds based on taste (not toxicity). The Occupational Safety and Health Administration (OSHA) has established a protective level of 1 milligram per cubic meter (mg/m^3) for zinc chloride fumes and a level of 5 mg/m^3 for zinc oxide fumes during an 8-hour workday over a 40-hour workweek. The National Institute for Occupational Safety and Health (NIOSH) has established the same standards for zinc and zinc chloride fumes for up to a 10-hour workday over a 40-hour workweek.

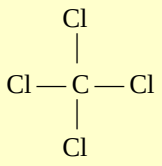
Where Can I Find More Information? More information on zinc can be found in the primary information source for this overview, the Toxicological Profile for Zinc prepared by the Agency for Toxic Substances and Disease Registry (ATSDR) and available on the Internet at http://www.atsdr.cdc.gov/toxpro2.html. Other web-based sources of information include the ATSDR ToxFAQs (http://www.atsdr.cdc.gov/toxfaq.html), EPA's Integrated Risk Information System (http://www.epa.gov/iris/subst/index.html), and the Hazardous Substances Data Bank (http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB).



Carbon Tetrachloride

What Is It? Carbon tetrachloride, CCl_4, is a nonflammable man-made chemical that is produced as a liquid but evaporates easily in the environment and is commonly found as a gas. This colorless liquid is slightly soluble in water and has a sweet odor; most people can smell carbon tetrachloride at concentrations of about 140 to 580 parts per million (ppm). An organic compound, carbon tetrachloride stays in air for a long time, with a half-life of 30 to 100 years. (The chemical half-life is the time it takes half the initial amount to be broken down, e.g., by photolysis or a photochemical reaction). When heated to very high temperatures, it decomposes to toxic phosgene and hydrogen chloride fumes.

Symbol: **CCl_4**



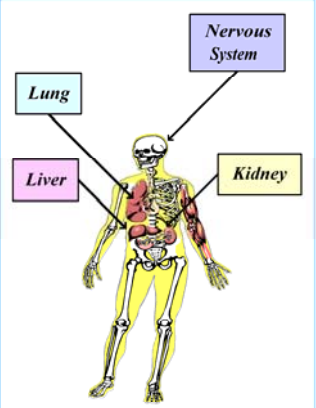
Molecular Weight: 154

How Is It Used? Carbon tetrachloride was widely used for decades as a cleaning fluid and solvent, including for degreasing equipment and machinery parts at facilities such as the Hanford Site. It was also used at the Hanford Site in the refining process during the separation of plutonium. In the past, carbon tetrachloride was commonly used in the dry cleaning industry and in homes as a spot remover and was also used to make refrigerator fluids and propellants for aerosol cans. It was also used in agriculture through the mid-1980s as a fumigant to kill insects in grain. Although carbon tetrachloride is still used in some industrial applications, its production is being phased out in this country and in many others because of concerns about its effects on the earth's ozone layer.

What's in the Environment? Carbon tetrachloride is widespread in the environment due to its extensive past use and persistence. Because it is slightly soluble in water and evaporates quickly from surface water and soil, most is present in air. Unlike many volatile compounds, carbon tetrachloride is quite stable in air. It does not readily dissociate in the lower atmosphere, nor is it easily washed out by rainfall. Only when it reaches the upper atmosphere (above 20 kilometers) does photodissociation become important. There, CCl_4 reacts with ultraviolet light, producing trichloromethyl radicals and chlorine atoms. These two photodegradation products are key catalytic species in reaction pathways that destroy the ozone layer. Worldwide, its concentration in air is about 0.1 part per billion (ppb), while urban levels are higher at 0.2 to 0.6 ppb; reported levels in U.S. cities range from 0.14 to 0.3 ppb. This compound is also often found in air inside buildings (from internal materials).

Carbon tetrachloride is present in many drinking water supplies, usually at less than 0.5 ppb. In groundwater, it can undergo reductive dechlorination in the presence of either free sulfide and ferrous ions or naturally occurring minerals that provide these ions. Microbial degradation has been shown to occur with estimated half-lives (in laboratory studies) of 6-12 months under aerobic conditions and 7-28 days under anaerobic conditions, although data are limited. In soil and sediment, carbon tetrachloride attaches to organic matter, preferring this phase 100 times more than water. It does not appear to accumulate in plants or animals, including fresh- and salt-water organisms.

What Happens to It in the Body? Carbon tetrachloride can enter the body when someone breathes air or ingests water or food containing the chemical, and it can also be easily absorbed through the skin. When carbon tetrachloride is inhaled or ingested (and possibly following absorption through the skin), much of it leaves the body within an hour or two in exhaled air. Of the initial amount breathed in, 30 to 60% can be absorbed across the lungs and retained in the body, notably in fat. The rest is removed, primarily in the feces.



Organs and systems affected when CCl4 is taken in through food, water, or air.

Of the initial amount ingested, most (85% or more) is quickly absorbed into the bloodstream from the gastrointestinal tract. About 80 to 85% is then exhaled beginning about 8 minutes after ingestion (as blood circulates to the lungs). About 4% of the amount that stays in the body is converted to carbon dioxide and then exhaled, while the remainder is metabolized and degraded, with a half-life in the body of about one day. Most of what remains accumulates in fatty tissue such as the liver and can take several weeks to be eliminated from the body in urine or feces, either as carbon tetrachloride or as degradation products such as chloroform.

What Are the Primary Health Effects? Inhaling high concentrations (20,000 ppb or more) of carbon tetrachloride can affect the central nervous system, causing headache and dizziness often accompanied by nausea. If someone breathes air with levels ten times higher (200,000 ppb or more), the liver and kidney can be affected. In the liver, carbon tetrachloride causes fat to build up, making this organ swollen and tender and impairing its function. In the kidney, it reduces the ability to produce urine, causing the body to retain water (especially the lungs) and waste products to build up in the blood. Except in severe cases, these effects disappear after exposure stops, and the liver and kidney begin functioning normally again within a few days or weeks.

Eating food or drinking water with high concentrations of carbon tetrachloride can also cause similar effects in the liver and kidney. Eating food with 2,500 ppm carbon tetrachloride can cause mild effects in most people, but they can be severe, even fatal, in individuals such as heavy drinkers whose liver function is already impaired. Its toxicity is also increased by interactions with other chemicals such as ketones (e.g., acetone). Carbon tetrachloride has been shown to increase the frequency of liver tumors in animals given relatively high concentrations by mouth (through a tube) for a long time. Although data indicate it causes liver cancer in animals, we do not know whether it can cause cancer in humans ingesting it in food or water. We also do not know whether it can cause cancer in animals or humans if it is inhaled. On the basis of the animal studies, the U.S. Environmental Protection Agency (EPA) has identified carbon tetrachloride as a probable human carcinogen.

What Is the Risk? The EPA has developed toxicity values to estimate the risk of getting cancer or other adverse health effects as a result of inhaling or ingesting carbon tetrachloride *(see box below).* The toxicity value for estimating the risk of getting cancer is called a slope factor (SF), and the value for estimating risk following inhalation exposure is called an inhalation unit risk (UR). An SF is an estimate of the chance that a person exposed to the chemical will get cancer from ingesting one milligram per kilogram (mg/kg-day) for a lifetime. The UR is an estimate of the chance that a person will get cancer from continuous exposure to a chemical in air at a unit concentration of one milligram per cubic meter (mg/m^3). The value for the non-cancer effect is called a reference dose (RfD), which is an estimate of the highest dose that can be taken in every day without causing an adverse non-cancer effect. These toxicity values have been developed by studying test animals given relatively high doses over their lifetimes, then adjusting and normalizing those results to a mg/kg-day basis for humans.

To illustrate how the RfD is applied, a 150-pound (lb) person could safely ingest 0.05 mg carbon tetrachloride every day without expecting any adverse effects (2.2 lb = 1 kg, or 1,000 g). In contrast to the RfD, which represents a "safe daily dose" (and so is compared to the amount an individual takes in, as a ratio), the SF is multiplied by the amount taken in to estimate the cancer risk and the UR is multiplied by the air concentration. Using these values, the EPA estimates that a person would have a one-in-a-million chance of developing cancer if they drank about two quarts of water containing 0.3 microgram per liter (μg/L), or inhaled air containing about 0.07 microgram per cubic meter ($\mu g/m^3$) every day for 30 years.

Chemical Toxicity Values		
Cancer Risk		**Non-Cancer Effect**
Oral SF	***Inhalation UR***	***Oral RfD***
0.13 per mg/kg-day	0.015 per mg/m^3	0.0007 mg/kg-day
The RfD for inhaling carbon tetrachloride has been taken to be the same as that developed for ingestion.		

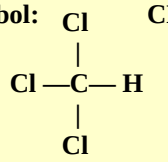
What Are Current Limits for Environmental Releases and Human Exposures? To help track facility releases to the environment, the Superfund amendments addressing emergency planning and community-right-to-know require the immediate reporting of releases of 10 lb (4.54 kg) or more carbon tetrachloride that occur within a 24-hour period, and also require normal releases to be reported annually and entered into a nationwide Toxics Release Inventory. For drinking water supplies, EPA has established a maximum contaminant level of 5 ppb and recommends that the level not exceed 300 ppb for adults or 70 ppb for children for chronic exposures (those extending more than seven years). For air in the workplace, the Occupational Safety and Health Administration has identified a limit of 10,000 ppb for an 8-hour work day over a 40-hour work week.

Where Can I Find More Information? More information can be found in the primary information source used to prepare this overview: the Toxicological Profile for Carbon Tetrachloride, prepared by the Agency for Toxic Substances and Disease Registry (ATSDR) and available at http://www.atsdr.cdc.gov/toxpro2.html. Other web-based sources of information include the ATSDR ToxFAQs (http://www.atsdr.cdc.gov/toxfaq.html), EPA's Integrated Risk Information System (http://www.epa.gov/iris/subst/index.html), and the Hazardous Substances Data Bank (http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB).

Chloroform

What Is It? Chloroform, $CHCl_3$, is a non-flammable, colorless liquid also known as trichloromethane or methyltrichloride. It is slightly soluble in water and has a slightly sweet taste. Chloroform has a pleasant, non-irritating odor, and most people can smell it at concentrations of about 133 to 276 parts per million (ppm) in air. It decomposes in air with a half-life of about 80 days (the chemical half-life is the time it takes half the initial amount to be broken down), to form phosgene and hydrogen chloride.

Symbol: $CHCl_3$



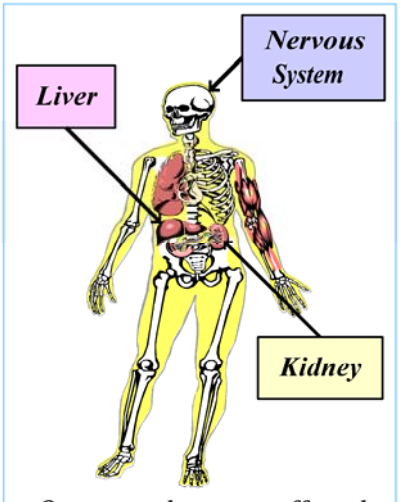
Molecular Weight: 119

How Is It Used? Chloroform is used primarily in the production of chlorodifluoromethane (a refrigerant in home air conditioners and supermarket freezers) and fluoropolymers. It is also used as an intermediate in the production of plastics, pharmaceuticals, dyes, and pesticides; as a heat transfer medium in fire extinguishers; and as a general solvent. In the past, chloroform was used extensively as an anesthetic during surgery, but that use has been discontinued.

What's in the Environment? Chloroform commonly enters the environment from manufacturing processes at industrial facilities, including certain chemical companies and paper mills. It is also formed as a byproduct of chlorinating drinking water and wastewater from sewage treatment plants. (Chlorine is added to these waters to kill bacteria.) Chloroform can also be released into the air during household use of water that contains it, such as during showering.

Most chloroform that enters the environment eventually reaches the atmosphere. The amount of chloroform typically measured in U.S. air ranges from 0.02 to 0.05 part of chloroform per billion parts (ppb), but concentrations as high as 610 ppb have been measured in air at a municipal landfill. Concentrations of chloroform in public water supplies generally range from about 2 to 40 ppb, although concentrations above 300 ppb have been reported. Little information is available on concentrations in soil, surface water, and groundwater in uncontaminated areas. However, soil concentrations are expected to be very low given the tendency of chloroform to either volatilize or move through soil to groundwater. The concentration associated with soil particles has been estimated to be about 30% of that in interstitial water (in pore spaces between soil particles).

What Happens to It in the Body? Chloroform can enter the body by breathing air, eating food, or drinking water containing the chemical and it can also be absorbed through the skin. When chloroform is inhaled, up to 75% is retained in the body and nearly all ingested chloroform is retained. Although chloroform is carried by the blood to all parts of the body, it tends to accumulate in tissues with high concentrations of lipids or fats. The tissues with the highest concentrations are adipose, brain, liver, kidney, and blood. Chloroform is metabolized in the body, and the major end product is carbon dioxide. Studies have shown that over 96% of inhaled chloroform is exhaled through the lungs within 8 hours, mainly as carbon dioxide and unchanged chloroform. Only a small amount leaves the body in urine and feces.



Organs and systems affected when chloroform is taken in through food, water, or air.

What Are the Primary Health Effects? At high concentrations, chloroform depresses the central nervous system and can induce both narcosis and anesthesia (unconsciousness). Inhaling somewhat lower concentrations (about 900 ppm) for a short time can cause fatigue, dizziness, and headache. Exposure to relatively low levels of chloroform in air or water for long periods of time can damage the liver and kidneys, and skin sores have been reported following direct contact. Reproductive and birth defects have been reported in laboratory

animal studies. Miscarriages in pregnant (female) rats and mice as well as abnormal sperm (males) were reported following exposure to between 30 and 400 ppm of chloroform in air. It is not known if chloroform causes reproductive effects in humans.

Some studies suggest a relationship between the ingestion of chlorinated water and cancer of the colon and bladder in humans. Cancer of the liver and kidney has been observed in rats and mice exposed to elevated levels of chloroform in food and drinking water. The U.S. Environmental Protection Agency (EPA) considers chloroform likely to be carcinogenic to humans by all routes of exposure under *high-exposure* conditions that lead to cytotoxicity (toxicity to cells) and regenerative hyperplasia (abnormal increase in number of cells) in susceptible tissues. However, chloroform is *not likely to be carcinogenic to humans by any route of exposure* under lower exposure conditions that do not cause cytotoxicity and regenerative hyperplasia.

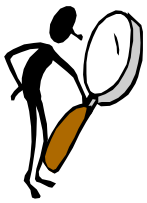
What Is the Risk? The EPA has developed toxicity values *(see box below)* to estimate the risk of getting cancer or other adverse health effects as a result of inhaling or ingesting chloroform. The toxicity value for estimating the cancer risk following inhalation exposure is called an inhalation unit risk (UR). The UR is an estimate of the chance that a person will get cancer from continuous exposure to a chemical in air at a unit concentration of 1 milligram per cubic meter (mg/m^3). Using the inhalation UR, the EPA estimates that a person would have a one-in-a-million chance of developing cancer if exposed daily over a lifetime to air containing 0.04 microgram per cubic meter ($\mu g/m^3$, where 1 mg = 1,000 µg). The toxicity value typically used for estimating the risk of getting cancer following oral exposure is called a slope factor (SF). However, because the carcinogenicity of chloroform following ingestion occurs only at exposures resulting in cytotoxicity, the EPA has determined that protecting against the non-cancer effects of oral exposure to chloroform is also protective against increased risk of cancer and so has not developed an SF.

Chemical Toxicity Values	
Cancer Risk	**Non-Cancer Effect**
Inhalation UR	***Oral RfD***
0.023 per mg/m^3	0.01 mg/kg-day

The toxicity value for non-cancer effects from oral exposure is called a reference dose (RfD), which is an estimate of the highest dose that can be taken in every day without causing an adverse non-cancer effect. To illustrate how the RfD is applied, a 150-pound (lb) person could safely ingest 0.68 mg of chloroform every day without expecting any adverse effects (2.2 lbs = 1 kg, or 1000 g, or 1 million mg). A toxicity value for non-cancer effects following inhalation exposure to chloroform is not available.

What Are Current Limits for Environmental Releases and Human Exposures? To help track facility releases to the environment, the Superfund amendments addressing emergency planning and community right-to-know require the immediate reporting of a release or spill of 10 pounds or more of chloroform that occurs within a 24-hour period, and also requires that normal releases be reported annually and entered into a nationwide Toxics Release Inventory. For drinking water supplies, the EPA has established a maximum contaminant level of 100 ppb for trihalomethanes, a class of chemicals that includes chloroform. For air in the workplace, the Occupational Safety and Health Administration has set a ceiling limit of 50 ppm that is not to be exceeded at any time.

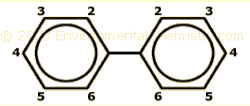
Where Can I Find More Information? More information on chloroform can be found in the primary information source for this overview, the Toxicological Profile for Chloroform, prepared by the Agency for Toxic Substances and Disease Registry (ATSDR) and available on the Internet at http://www.atsdr.cdc.gov/toxpro2.html. Other web-based sources of information include the ATSDR ToxFAQs (http://www.atsdr.cdc.gov/toxfaq.html), EPA's Integrated Risk Information System (http://www.epa.gov/iris/subst/index.html), and the Hazardous Substances Data Base (http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB).



Polychlorinated Biphenyls (PCBs)

What Are They? Polychlorinated biphenyls (PCBs) are a family of 209 chlorinated hydrocarbon compounds (known as congeners) with the same general chemical structure. In the United States, commercial mixtures of these man-made chemicals were commonly sold under the trade name Aroclor, including Aroclors 1248, 1254, and 1260. For most Aroclors, the last two digits indicate the approximate percent of chlorine (e.g., Aroclor 1254 is 54% chlorine by weight.) The PCBs are nonflammable, colorless to light yellow oily liquids or waxy solids that have no odor or taste. They break down very slowly in the environment (with half-lives of months to years) and tend to cycle between air, water, and soil. (The chemical half-life is the time it takes half the initial amount to be broken down.) They can travel long distances in air and water and are found all over the world, including remote areas such as the Arctic.

General PCB Structure



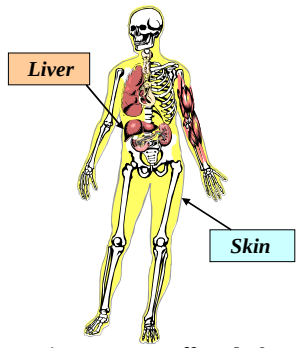
Chlorine atoms can be located in any of the numbered positions.

How Are They Used? The production of PCBs ended in the United States in 1997 due to concerns over their environmental persistence and possible health and ecological effects. Previously, these compounds were used in hundreds of commercial and industrial applications due to their chemical stability, high heat capacity, low flammability, and insulating properties. Prior to 1974, PCBs were used in both closed and open system applications, with the latter including flame-retardants, inks, adhesives, dyes, paints, plasticizers, and fluorescent lighting fixtures. After 1974, the use of PCBs was restricted to closed system applications such as coolants and lubricants in transformers, capacitors, and other electrical equipment. PCBs are still found in closed systems, where, for example, the volume of PCBs can range from only a few milliliters in small capacitors to over several thousand liters in large capacitors.

What's in the Environment? PCBs are found throughout the environment, although concentrations within the United States have been decreasing over time because of restrictions on their use and disposal. They have been released into air, water, and soil from contaminated facilities, including from incineration of PCB-containing wastes, leakage of old electrical equipment and improper disposition of spills. PCBs do not degrade easily, so they tend to persist in the environment as they cycle among air, water and soil. Their movement among these media depends on factors such as their degree of chlorination and climatic conditions. Low-chlorinated PCBs can volatilize from water and soil into air, with the highest release rates occurring in the summer due to higher temperatures. These lighter PCBs can be found far away from their original points of release. The low-chlorinated PCBs are also more reactive in the atmosphere and can be degraded to carboxylic acids in the presence of highly reactive molecules. Highly chlorinated PCBs can stick tightly to airborne particulates, but they do not tend to travel as far as lighter PCBs. PCBs can remain in the air for long periods of time, with half-lives ranging from months to years. In the United States, the average concentration in air ranges from about 0.02 to 3.36 nanograms per cubic meter (1 nanogram is a billionth of gram), with the highest concentrations found in urban areas. Highly chlorinated PCBs also stick strongly to sediments and soils, especially those with high organic content. Because of their strong affinity for soil, PCBs typically do not migrate to groundwater. Concentrations in soil are typically less than 100 micrograms per kg (µg/kg), but concentrations can be more than 10,000 times higher in highly contaminated soils. PCBs are widely detected in surface waters in the low parts per trillion range. Similar to air, the half-lives of PCBs in soil or water ranges from months to years. Of particular concern is bioaccumulation, in which PCB concentrations in fish and animals high in the food chain can be much higher than concentrations in fish or animals lower in the food chain. Levels of PCBs in fish have been found in the parts per million (ppm) range, while levels in meat and dairy products are typically in the low parts per billion range.

What Happens to Them in the Body? PCBs can be taken into the body primarily by eating contaminated food or breathing contaminated air, and to a more limited degree through ingestion of or dermal contact with contaminated water or soil. When PCBs are ingested, up to 100% can be absorbed into the blood; the amount absorbed following inhalation is not known. PCBs tend to accumulate in tissues with high concentrations of lipids and fats and can remain in the body for a long time. The tissues with the highest PCB concentrations are adipose, blood, brain, liver, and lungs. Due to the complex structure and number of different congeners, PCBs can undergo a variety of metabolic reactions. The rate of metabolism is generally lower for the more highly chlorinated congeners, but the number and position of the chlorine atoms is also important. These compounds are excreted in feces and urine. Some metabolites may be as harmful or more harmful than the unchanged congener. Reported half-lives of PCB mixtures in the body range from 2.6 to 8 years.

What Are the Primary Health Effects? The most commonly observed effects in people (primarily workers) exposed to relatively high concentrations of PCBs are skin conditions such as chloracne and rashes and effects on liver function. Liver effects are more severe in people with impaired liver function, such as alcoholics. Studies in animals indicate that exposure to PCBs can harm the liver, thyroid, and immune and endocrine systems. Women exposed to large amounts of PCBs during pregnancy were found to have children with lower birth weights, abnormal behavior, irregular development of the immune system, and other effects. Because PCBs concentrate in breast milk, nursing infants might be at higher risk depending on exposure levels. In animals, PCBs have been shown to reduce conception rates and live birth rates. Animal studies indicate that exposure to relatively low levels over a long time (years) can result in liver and possibly other cancers, with suggestive evidence from human studies. The U.S. Environmental Protection Agency (EPA) and International Agency for Research on Cancer have determined that PCBs are probably carcinogenic to humans.



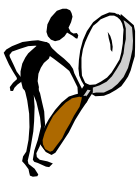
Primary organs affected when PCBs are inhaled or ingested.

What Is the Risk? The EPA has developed toxicity values (*see box below)* to estimate the risk of getting cancer or other adverse health effects as a result of ingesting or inhaling PCBs. The toxicity values were developed based on studies of animals exposed to PCB mixtures and individual PCB congeners. The toxicity value for estimating the risk of getting cancer following oral exposure is a slope factor (SF), and the value for estimating risk following inhalation exposure is a unit risk (UR). An SF is an estimate of the chance that a person exposed to a chemical will get cancer from ingesting 1 milligram per kilogram per day (mg/kg-day) for a lifetime, and the UR is an estimate of the chance that a person will get cancer from continuous exposure to a chemical in air at a concentration of 1 mg/m^3. Because PCBs are mixtures with different toxicities that are also subject to different environmental processes (e.g., volatilization and degradation), EPA developed a range of SFs, corresponding to high, low, and lowest risk and persistence conditions. The criteria for selection of the appropriate SF depend on the available information and exposure context. For example, the high value is used to evaluate food chain exposure and soil ingestion, and the low value is typically used to evaluate inhalation of volatile congeners. To illustrate how the UR might be applied, using the low risk and persistence UR, a person would have a one-in-a-million chance of developing cancer if exposed daily over a lifetime to air containing 0.01 $\mu g/m^3$ of PCBs. The toxicity value for estimating non-cancer effects from oral exposure is called a reference dose (RfD), which is an estimate of the highest dose that can be taken in every day without causing adverse non-cancer health effects. Using the RfD, a 150-pound (lb) person could safely ingest 0.001 mg of Aroclor 1254 or 0.0005 mg of Aroclor 1016 every day without expecting any adverse effects. The EPA is developing further guidance for assessing PCBs at contaminated sites.

Chemical Toxicity Values			
Cancer Risk		**Non-Cancer Effect: Oral RfD**	
Oral SF	***Inhalation UR***	**Aroclor 1254**	**Aroclor 1016**
0.07 per mg/kg-day *(lowest)* 0.4 per mg/kg-day *(low)* 2 per mg/kg-day *(high)*	0.02 per mg/m^3 *(lowest)* 0.1 per mg/m^3 *(low)* 0.6 per mg/m^3 *(high)*	0.00002 mg/kg-day	0.00007 mg/kg-day

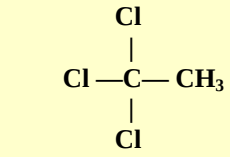
What Are Current Limits for Environmental Releases and Human Exposures? To help track facility releases to the environment, the Superfund amendments that address emergency planning and community right-to-know require immediate reporting of a release of 1 lb (0.45 kg) or more of PCBs to air, water, or land, and also require that normal releases be reported annually and entered into a nationwide Toxics Release Inventory. For drinking water supplies, the EPA has established a maximum contaminant level of 0.5 ppb, with a goal of zero. For air in the workplace, the Occupational Safety and Health Administration has identified limits of 1 mg/m^3 and 0.5 mg/m^3 for PCBs containing 42% and 54% chlorine, respectively, for an 8-hour work day over a 40-hour work week. The Food and Drug Administration and Federal Insecticide, Fungicide, and Rodenticide Act have set tolerance levels for PCBs in consumer products such as milk and manufactured dairy products (1.5 ppm), poultry (3 ppm), eggs (0.3 ppm), animal feed for food producing livestock (0.2 ppm), fish and shellfish (2 ppm), infant and junior foods (0.2 ppm), and paper (10 ppm).

Where Can I Find More Information? More information on PCBs can be found in the primary information source used to prepare this overview, the Toxicological Profile for PCBs, prepared by the Agency for Toxic Substances and Disease Registry (ATSDR) and available at http://www.atsdr.cdc.gov/toxprofiles/tp17.html. Other web-based sources of information include the ATSDR ToxFAQs (http://www.atsdr.cdc.gov/tfacts17.html), the EPA's Integrated Risk Information System (http://www.epa.gov/iris/subst/index.html), and EPA's report, PCBs: Cancer Dose Response Assessment and Application to Environmental Mixtures (http://www.epa.gov/opptintr/pcb/pcb.pdf).



Trichloroethane

Symbol: $C_2H_3Cl_3$



Molecular Weight: 133

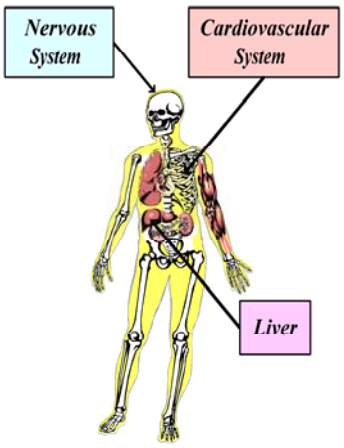
What Is It? Trichloroethane, $C_2H_3Cl_3$, is a man-made, colorless liquid with a sweet, chloroform-like odor. Most people can smell it at levels of about 120 to 500 parts per million (ppm). Also known as TCA; 1,1,1-trichloroethane; and methylchloroform, this compound is slightly soluble in water. It evaporates readily and has an atmospheric residence time of about six years. Trichloroethane is nonflammable, but notably at higher temperatures its vapors form phosgene gas and hydrogen chloride.

How Is It Used? Trichloroethane production in the United States ended in 1996 as part of the phase-out of ozone-depleting chemicals. Although not prohibited, its use has declined since then as extant stores of trichloroethane have been depleted. Previously, trichloroethane was widely used as a solvent in about a fourth of all U.S. industries. It served as a cleaner and degreaser for various equipment, as a precursor in the manufacture of chemicals such as vinylidene chloride, and as a coolant during the drilling and tapping of metals. Trichloroethane was also used in a variety of consumer products, including drain cleaners, cleaning agents, shoe polish, spot removers, wallpaper and carpet glues, food packaging adhesives, photographic films, insecticides, rodenticides, and in many arts and crafts supplies such as printing inks and paints. In addition, trichloroethane was occasionally used as an aerosol propellant.

What's in the Environment? Trichloroethane has been released to the environment by both process and fugitive emissions as a result of industrial and consumer applications, although these emissions have decreased significantly since production ended nearly 10 years ago. Because of its high volatility, trichloroethane released to surface soil and water evaporates quickly, and most is found in air. In the atmosphere, trichloroethane is transformed over time to other chemicals through reactions with hydroxyl radicals. About 15% migrates to the stratosphere where it is converted into chlorine atoms by lower-wavelength ultraviolet light; it is these chlorine atoms that contribute to degradation of the ozone layer. When large amounts of trichloroethane are released to soil, some can move rapidly down to groundwater while some evaporates. The concentration associated with soil particles has been estimated to be about 80% of that in interstitial water (in pore spaces between soil particles). What does reach groundwater can evaporate and move back up through soil as a gas into the air; its predicted half-life in groundwater is 200 to 300 days. Organisms naturally present in both soil and groundwater can break down this compound, and it does not accumulate in plants or animals.

Typical concentrations of trichloroethane in air range between 0.1 and 0.9 parts per billion (ppb) outdoors and 0.3 and 4.4 ppb indoors, although concentrations as high as 290 ppb have been reported in air in new and recently renovated buildings. Higher concentrations in indoor air are likely due to the use of trichloroethane in consumer products, although that use continues to decline. Up to 0.01 ppm trichloroethane has been detected in rivers and lakes, and drinking water concentrations have been reported as 0.01 to 3.5 ppb, with higher levels of 0.8 to 142 ppb in municipal water systems. Concentrations in groundwater typically range from 0 to 18 ppb, although highly contaminated wells have been found to contain up to 3 grams per liter (g/L). Soil can be contaminated with trichloroethane through accidental spills, leaching of contaminated water from landfills or treatment and storage lagoons, application of insecticides and rodenticides, wet deposition, and percolation of contaminated rainwater. Trichloroethane has been detected in soil at levels up to 120 ppm. It has also been found in raw, processed, and prepared food products, with average concentrations ranging from about 0.004 ppb (ice from a commercial machine) to 16,000 ppb (allspice).

What Happens to It in the Body? Inhalation is the primary route by which people are exposed to trichloroethane. It is rapidly absorbed from the lungs following inhalation and from the gastrointestinal tract following ingestion. Although it is also absorbed through the skin, this route is typically less important because trichloroethane evaporates so quickly. The highest concentrations of trichloroethane are found in fat, liver, kidney and brain. It is rapidly eliminated from the body through the lungs and urine, with about 90% of the amount taken in leaving unchanged in exhaled air, irrespective of the route of exposure (that is, whether inhaled, ingested, or absorbed through the skin). The remainder is metabolized and excreted either in the urine as trichloroethanol and trichloroacetic acid or exhaled from the lungs as carbon dioxide.



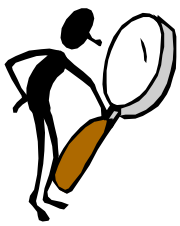
Primary organs affected when trichloroethane is inhaled or ingested.

What Are the Primary Health Effects? Trichloroethane is relatively less toxic than most chlorinated solvents (which accounts in part for its extensive use before effects on the ozone layer were recognized). At elevated concentrations, trichloroethane can depress the central nervous system (slowing functions) and cause an irregular heartbeat, loss of balance and coordination, and fluid buildup in the lung. In animals, exposure to lower concentrations can damage the nervous system and liver. Trichloroethane is one of many solvents that have been intentionally inhaled to alter mood or consciousness, and it has been associated with "sudden sniffing death" syndrome. Although some studies suggest that this compound is weakly mutagenic, those effects may be associated with another chemical present as a contaminant and not trichloroethane itself. The U.S. Environmental Protection Agency (EPA) has stated that adequate information is not available to evaluate its carcinogenicity.

What Is the Risk? The EPA has developed toxicity values called reference doses (RfDs) to estimate the risk of non-cancer health effects as a result of ingesting various chemicals. An RfD is an estimate of the highest dose than can be taken in every day without causing an adverse health effect. Although the EPA previously established an oral RfD for trichloroethane, the agency withdrew that value pending further scientific evaluation. A reference concentration (RfC), which is the concentration in air that can be inhaled every day without causing non-cancer health effects, has not been established for this compound.

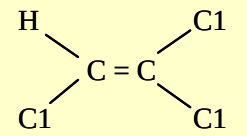
What Are Current Limits for Environmental Releases and Human Exposure? To help track facility releases to the environment, the Superfund amendments that address emergency planning and community right-to-know require the immediate reporting of a release of 1,000 pounds (454 kilograms) or more of trichloroethane that occurs within 24 hours, and also require normal releases to be reported annually and entered into a nationwide Toxics Release Inventory. For drinking water, the EPA has established a maximum contaminant level of 0.2 milligram per liter (mg/L). For air in the workplace, the Occupational Safety and Health Administration (OSHA) has established a limit of 350 ppm for an 8-hour workday over a 40-hour week.

Where Can I Find More Information? More information on trichloroethane can be found in the primary information source for this overview, the Toxicological Profile for 1,1,1-Trichloroethane, prepared by the Agency for Toxic Substance and Disease Registry (ATSDR) and available at http://www.atsdr.cdc.gov/toxpro2.html. Other web-based sources of information include the ATSDR ToxFAQs (http://www.atsdr.cdc.gov/toxfaq.html), EPA's Integrated Risk Information System (http://www.epa.gov/iris/subst/index.html), and the Hazardous Substances Data Bank (http://www.toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB).



Trichloroethylene

Symbol: C_2HCl_3



Molecular Weight: 131

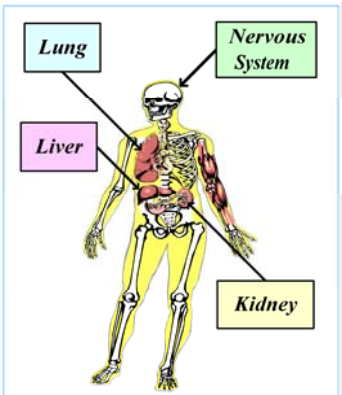
What Is It? Trichloroethylene, C_2HCl_3, is a man-made, colorless liquid with a sweet odor that most people can detect at levels of about 100 parts per million (ppm). Also known as trichloroethene and often called TCE, this compound is moderately soluble in water. Trichloroethylene evaporates readily and has an atmospheric half-life of about a week. It is converted to phosgene gas and hydrogen chloride.

How Is It Used? Trichloroethylene is an industrial solvent used primarily in metal cleaning and degreasing operations, including to remove grease from metal parts used to make automobiles. It is also used to make other chemicals such as polyvinyl chloride, and in varnishes, adhesives, paints, and lacquers. Trichloroethylene is present in many household products, such as spot removers, carpet cleaning fluids, typewriter correction fluids, and paint removers. It was once used as a dry-cleaning solvent, fumigant, and general anesthetic, but those uses have been discontinued. Before 1977, it was used to remove caffeine from coffee beans. An estimated 200 million pounds of trichloroethylene are used each year in the United States.

What's in the Environment? Trichloroethylene is mainly present in air and water, with metal degreasing representing a major release source for air. Although rapidly broken down in the atmosphere, primarily via reactions with hydroxyl radicals to produce phosgene, dichloroacetyl chloride, and formyl chloride, TCE is used in such large amounts that those levels continue to be replenished. Found throughout the world, the average concentration in U.S. air ranges from about 30 to 460 parts per trillion (ppt), with higher levels in urban and industrial areas.

Trichloroethylene evaporates easily from surface water, but it can remain in the subsurface, including groundwater, for a long time. In soil and groundwater, it can be broken down by bacteria to produce vinylidene chloride (a suspected human carcinogen) and vinyl chloride (a known human carcinogen). The concentration of trichloroethylene associated with the soil particles has been estimated to be about 60% of that in interstitial water (in pore spaces between soil particles). From a study by the U.S. Environmental Protection Agency (EPA), up to a third of drinking water sources in this country contain trichloroethylene. The median concentration in waters from groundwater sources is around 0.3 parts per billion (ppb). Trichloroethylene has also been found at very low levels in various prepared foods. It does not accumulate to significant levels in plants, animals, or fish.

What Happens to It in the Body? Trichloroethylene can enter the body by breathing air, drinking water, or eating food containing this chemical, and it can also be absorbed through the skin. When trichloroethylene is inhaled, up to 75% can be retained in the body. A substantial fraction is also retained following ingestion. Some trichloroethylene is stored in fatty tissue, while the rest remains in the blood to circulate throughout the body, and it is rapidly metabolized to dichloroacetic acid, trichloroacetic acid, trichloroethanol, and other chemicals. Most of these metabolites leave the body in the urine.



Organs and systems affected when TCE is taken in through food, water, or air.

What Are the Primary Health Effects? Exposure to trichloroethylene can potentially affect a number of organs and systems, including the nervous system, liver, kidney, blood, cardiovascular system, immune system, and reproductive system. Skin contact with trichloroethylene can cause rashes, while inhalation or ingestion of high concentrations for a short time primarily

affects the central nervous system. Inhalation or ingestion of very high concentrations can lead to loss of consciousness and death. At somewhat lower concentrations, people can become sleepy or dizzy or get headaches. Except in severe cases, the effects disappear after the exposure stops. Exposure to relatively low levels of trichloroethylene in air or water for a long period of time (years) can damage the liver and kidney, with more severe effects in people with impaired liver or kidney function, such as alcoholics.

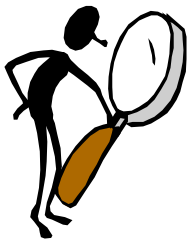
Abnormal skeletal development and other effects have been observed in the offspring of exposed animals; however, it is not known if similar effects can occur in humans. Trichloroethylene has been shown to cause cancer in animals, but we do not know if it causes cancer in humans. The International Agency for Research on Cancer has determined that trichloroethylene is not classifiable as to human carcinogenicity. The EPA is currently reviewing its carcinogenicity, and in a 2001 draft health assessment, characterized trichloroethylene as "highly likely to produce cancer in humans" based on the 1999 proposed (and now accepted) cancer guidelines and as a probable human carcinogen, based on the former 1986 cancer guidelines.

What Is the Risk? The EPA has developed toxicity values to estimate the risk of getting cancer or other adverse health effects as a result of inhaling or ingesting trichloroethylene. These toxicity values were developed by studying test animals given relatively high doses over their lifetimes, then adjusting and normalizing those results to a milligram per kilogram per day (mg/kg-day) basis for humans and by studying humans exposed in occupational settings. However, at this time, EPA has withdrawn all previously published toxicity values as further scientific data have become available and are being evaluated.

The toxicity value for estimating the risk of getting cancer is called a slope factor (SF) for ingestion exposure (also sometimes used for inhalation exposure). An SF is an estimate of the chance that a person exposed to a chemical will get cancer from taking in 1 milligram per kilogram of body weight per day (mg/kg-day), for a lifetime. The EPA identified draft oral SFs ranging from 0.02 to 0.4 per mg/kg-day in the 2001 draft health assessment. The toxicity value used to evaluate the non-cancer effect is called a reference dose (RfD) for ingestion exposure, and a reference concentration (RfC) for inhalation exposure. An RfD is an estimate of the highest dose that can be taken in every day without causing an adverse non-cancer effect. The RfC is the highest concentration in air that can be breathed everyday without causing non-cancer health effects. The oral RfD withdrawn from IRIS was 0.006 mg/kg-day. A draft RfD of 0.0003 mg/kg-day and an inhalation RfC of 0.04 milligram per cubic meter (mg/m^3) were later derived in EPA's 2001 draft health assessment.

What Are Current Limits for Environmental Releases and Human Exposures? To help track facility releases to the environment, the Superfund amendments that address emergency planning and community right-to-know require the immediate reporting of releases of 100 pounds (45.4 kg) or more of trichloroethylene that occur within a 24-hour period, and also require normal releases to be reported annually and entered into a nationwide Toxics Release Inventory. For drinking water supplies, the EPA has established a maximum contaminant level of 5 ppb for TCE with a goal of zero. For air in the workplace, the Occupational Safety and Health Administration has identified a limit of 100 ppm for an 8-hour work day over a 40-hour work week.

Where Can I Find More Information? More information on trichloroethylene can be found in the primary information source used to prepare this overview, the Toxicological Profile for Trichloroethylene, prepared by the Agency for Toxic Substances and Disease Registry and available at http://www.atsdr.cdc.gov/toxpro2.html. Other web-based sources of information include the ATSDRs ToxFAQs (http://www.atsdr.cdc.gov/toxfaq.html), the EPA's Integrated Risk Information System (http://www.epa.gov/iris/subst/index.html), and the National Library of Medicine Hazardous Substances Data Bank (http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB).



Cyanide

Symbol: CN^- **(-C ≡ N)**

Molecular Weight: 26

What Is It? Cyanide can occur naturally and can also be man-made. The most common forms are hydrogen cyanide (HCN) and the sodium and potassium salts (NaCN and KCN). Hydrogen cyanide is a colorless or pale-blue liquid at room temperature (below 78°F) and a colorless gas at higher temperatures; the gas is highly flammable and potentially explosive. Sodium and potassium cyanide are white crystals that can release hydrogen cyanide gas if they contact acids. Both the gas and crystals have a faint, bitter almond-like odor that is perceptible at a concentration of about 1 part per million (ppm).

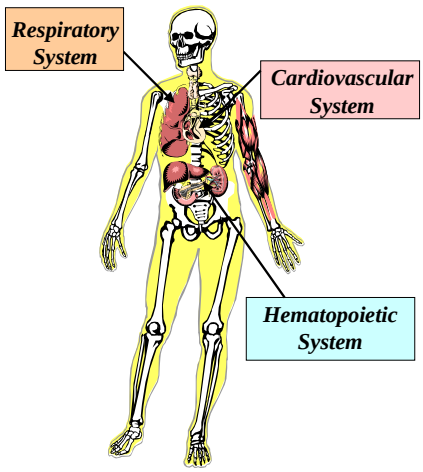
How Is It Used? Cyanide is used in electroplating, metallurgy, and mining. It is also used to make synthetic fibers, plastics, dyes, pharmaceuticals, and pesticides, including fumigants. In addition, cyanide serves as a chemical intermediate in various production processes. (Because of its acute lethality, cyanide has been used in suicide attempts, and hydrogen cyanide gas has been used in the past as a chemical weapon, including in gas chambers.)

What's in the Environment? Natural cyanides are produced by certain bacteria, fungi, and algae, and they are present in a number of plants and foods as cyanogenic glycosides. Man-made cyanides typically enter the environment from metal finishing and organic chemical industries. Other sources include iron and steel works, municipal waste burning, cyanide-containing pesticides, and vehicle exhaust. Cyanide is also present in cigarette smoke and smoke generated when certain synthetic materials such as plastics are burned.

The estimated concentration of hydrogen cyanide in non-urban air is less than 0.2 parts per billion (ppb), where its half-life can be from 1 to 3 years. Cyanide concentrations measured in urban runoff have ranged from 2 to 33 micrograms/liter (μg/L), and concentrations of 5 to 1,400 μg/L have been measured in leachates from U.S. landfills. Most cyanide in surface water forms hydrogen cyanide and evaporates. In drinking water, chloramine treatment can produce cyanogen chloride, with reported concentrations ranging from 0.45 to 0.80 μg/L. Concentrations in mainstream cigarette smoke range from 10 to 550 μg/cigarette, with lower concentrations (up to 111 μg/cigarette) in sidestream smoke. Cyanide is present in foods and plants at concentrations up to 4,000 ppm. It does not appear to accumulate in organisms, e.g., in fish.

What Happens to It in the Body? Cyanide typically enters the body primarily by breathing air, but people can also be exposed by eating foods containing cyanogenic glycosides, breathing cigarette smoke, and through contact with the skin. Skin contact is common only among people who work in cyanide-related industries. After being ingested or inhaled, hydrogen cyanide is rapidly absorbed through the gastrointestinal tract and lungs into the bloodstream, while the absorption of cyanide salts tends to be slower. Approximately 58% of inhaled hydrogen cyanide is absorbed.

Most cyanide that enters the body is changed to the less toxic form, thiocyanate. Some cyanide reacts to form vitamin B_{12}. The primary route of elimination is through excretion of thiocyanate in the urine. Some free hydrogen cyanide is also excreted unchanged in the breath, saliva, sweat, and urine. Most cyanide and its products leave the body within 24 hours after exposure.



Primary organs affected when cyanide is inhaled or ingested.

What Are the Primary Health Effects? Hydrogen cyanide and its salts are among the most rapidly acting poisons known. Cyanide exerts its toxic effects by blocking the use of oxygen by the cells. Within minutes, exposure to toxic levels of cyanide causes rapid breathing, weakness, headache, vomiting, and an increased heart rate. Exposure to lethal concentrations causes convulsions, low blood pressure, and

respiratory failure leading to death. Because of their slower absorption, death from ingesting cyanide salts can be delayed, but effects still usually occur within one hour. Survivors of cyanide poisoning may develop heart and brain damage. Exposure to lower concentrations of cyanide over extended periods of time can also result in health effects. In tropical regions of Africa, ataxic neuropathy (reduced coordination due to effects on the nervous system), goiter, poor vision, and other disorders have been associated with chronic ingestion of cassava, notably the root (manioc), a dietary staple containing cyanogenic glycosides that release hydrogen cyanide. A decrease in the production of thyroid hormone in newborns is also reported in areas where cassava is a basic food. Fetal abnormalities have been observed in animal studies. There are no reports that cyanide can cause cancer in animals or people.

What Is the Risk? The U.S. Environmental Protection Agency (EPA) has developed toxicity values to estimate the risk of developing non-cancer effects as a result of inhaling or ingesting cyanide compounds *(see box at right).* The toxicity value for non-cancer effects from oral exposure is called a reference dose (RfD), which is an estimate of the highest dose that can be taken in every day without causing an adverse non-cancer effect. The toxicity value for inhalation exposure is called a reference concentration (RfC), which is an estimate of the highest concentration in air that can be inhaled every day without causing an adverse non-cancer effect. The RfDs have been developed by studying test animals given relatively high doses of cyanide over their lifetimes, then adjusting and normalizing those results to a milligram per kilogram per day (mg/kg-day) basis for humans. The RfC for hydrogen cyanide is based on a study of workers exposed in an occupational setting. To illustrate how the RfD may be applied, a 150-pound (lb) person could safely ingest 2.7 mg of sodium cyanide every day without expecting any adverse effects (2.2 lb =1 kg, 1000 g, or 1 million mg).

Chemical Toxicity Values

Form of Cyanide	Non-Cancer Effect	
	Oral RfD	***Inhalation RfC***
Hydrogen cyanide	0.02 mg/kg-day	0.003 mg/m^3
Cyanide, free	0.02 mg/kg-day	None established
Calcium cyanide Cyanogen Sodium cyanide	0.04 mg/kg-day	None established
Chlorine cyanide Potassium cyanide Zinc cyanide	0.05 mg/kg-day	None established
Copper cyanide	0.005 mg/kg-day	None established
Silver cyanide	0.1 mg/kg-day	None established
Potassium silver cyanide	0.2 mg/kg-day	None established

What Are Current Limits for Environmental Releases and Human Exposures? To help track facility releases to the environment, the Superfund amendments addressing emergency planning and community right-to-know require that releases of certain chemicals to air, water, or land be reported immediately if they exceed specified levels, and they also require normal releases to be reported annually and entered into a nationwide Toxic Release Inventory. Several cyanide compounds are regulated under these amendments. Immediately reportable quantities are 1 lb (0.454 kg) for potassium silver cyanide and silver cyanide; 10 lb (4.45 kg) for calcium cyanide, copper cyanide, cyanogen chloride, hydrogen cyanide, potassium cyanide, and sodium cyanide; 100 lb (45.4 kg) for cyanogen; and 5,000 lb (2,270 kg) for ammonium thiocyanate. The EPA has established a limit of 0.2 mg/L for cyanide in drinking water. For air in the workplace, the Occupational Safety and Health Administration has set a permissible exposure limit (PEL) over an 8-hour work day of 11 milligrams per cubic meter (mg/m^3) for hydrogen cyanide, and a PEL of 5 mg/m^3 for potassium cyanide and sodium cyanide (as CN^-).

Where Can I Find More Information? More information on cyanide can be found in the primary information source for this overview, the Toxicological Profile for Cyanide, prepared by the Agency for Toxic Substances and Disease Registry (ATSDR) and available at http://www.atsdr.cdc.gov/toxpro2.html. Other web-based sources of information include the ATSDR ToxFAQs (http://www.atsdr.cdc.gov/toxfaq.html), the Hazardous Substances Data Bank (http://www.toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB), EPA's Integrated Risk Information System (http://www.epa.gov/iris/subst/index.html), and the Center for Disease Control and Prevention's Emergency Preparedness and Response Chemical Agents List (http://www.bt.cdc.gov/agent/cyanide/index.asp).

Nitrate and Nitrite

Symbol:	**NO_3 / NO_2**
Molecular Weight:	**62 / 46**

What Are They? Nitrate and nitrite are compounds that contain a nitrogen atom joined to oxygen atoms, with nitrate containing three oxygen atoms and nitrite containing two. In nature, nitrates are readily converted to nitrites and vice versa. Both are anions, or ions with a negative charge. They tend to associate with cations, or ions with a positive charge, to achieve a neutral charge balance.

How Are They Used? Nitrates are used primarily to make fertilizer, but they are also used to make glass and explosives. These compounds also are used in various chemical production and separation processes. Nitrites are manufactured mainly for use as a food preservative, and both nitrates and nitrites are used extensively to enhance the color and extend the shelf life of processed meats.

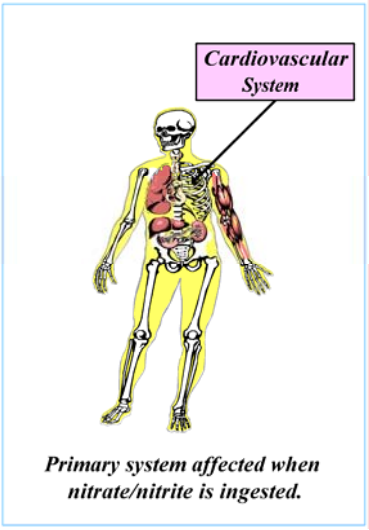
What's in the Environment? Nitrates are naturally present in soil, water, and food. In the natural nitrogen cycle, bacteria convert nitrogen to nitrate, which is taken up by plants and incorporated into tissues. Animals that eat plants use the nitrate to produce proteins. Nitrate is returned to the environment in animal feces, as well as through microbial degradation of plants and animals after they die.

Microorganisms can convert nitrate or the ammonium ion (which is a nitrogen atom combined with four hydrogen atoms) to nitrite; this reaction occurs in the environment as well as within the digestive tract of humans and other animals. After bacteria convert (reduce) nitrate to nitrite in the environment, the nitrogen cycle is completed when they then convert the nitrite to nitrogen. Normally, this natural cycling process does not allow excessive amounts of nitrates or nitrites to accumulate in the environment. However, human activities have increased environmental nitrate concentrations, with agriculture being the major source. This includes increased use of nitrogen-containing fertilizers as well as concentrated livestock and poultry farming; the latter two produce millions of tons of nitrate-containing manure each year. Nitrate and nitrite compounds are very soluble in water and quite mobile in the environment. They have a high potential for entering surface water when it rains, as nitrates in applied fertilizers can dissolve in runoff that flows into streams or lakes; they also have a high potential for entering groundwater through leaching. The concentration associated with soil particles has been estimated to be about half the concentration in interstitial water (the water in the pore spaces between the soil particles).

What Happens to It in the Body? Nitrate is a normal component of the human diet, with the average daily intake from all sources estimated at 75 milligrams (mg), or about 0.0026 ounce. Upon ingestion, about 5% of the nitrate taken in by healthy adults is converted (reduced) to nitrite by bacteria in saliva. Further nitrate is converted by bacteria inside the alimentary tract. Certain conditions in the stomach can increase the conversion of nitrate to nitrite, specifically when the pH of the gastric fluid is high enough (above 5) to favor the growth of nitrate-reducing bacteria. This process is of major concern for infants, whose gastrointestinal systems normally have a higher pH than those of adults. Nitrites in the stomach can react with food proteins to form N-nitroso compounds; these compounds can also be produced when meat containing nitrites or nitrates is cooked, particularly using high heat. While these compounds are carcinogenic in test animals, evidence is inconclusive regarding their potential to cause cancer (such as stomach cancer) in humans.

What Are the Primary Health Effects? Nitrates themselves are relatively nontoxic. However, when swallowed, they are converted to nitrites that can react with hemoglobin in the blood, oxidizing its divalent iron to the trivalent form and creating methemoglobin. This methemoglobin cannot bind oxygen, which decreases the capacity of the blood to transport oxygen so less oxygen is transported from the lungs to the body tissues, thus causing a condition known as methemoglobinemia.

Normal individuals have low levels (0.5 to 2%) of methemoglobin in their blood. When this level increases to 10%, the skin and lips can take on a bluish tinge (cyanosis), and levels above 25% can cause weakness and a rapid pulse. At levels above 50 to 60%, a person can lose consciousness, go into a coma, and die. Infants are much more sensitive than adults to nitrates/nitrites, and essentially all deaths from nitrate/nitrite poisoning have been in infants. Long-term exposure to lower levels of nitrates and nitrites can cause diuresis (an increase in the amount of urine, and starchy deposits and hemorrhaging of the spleen).



Primary system affected when nitrate/nitrite is ingested.

What Are the Risks? The U.S. Environmental Protection Agency (EPA) has developed toxicity values to estimate the risk of non-cancer health effects from ingesting nitrates and nitrites. The toxicity value used to estimate a non-cancer effect following ingestion is called a reference dose (RfD). An RfD is an estimate of the highest dose that can be taken in every day without causing an adverse effect.

The RfD for nitrate was developed considering the concentration at which methemoglobinemia was indicated at levels above 10% for 0- to 3-month-old infants. This was based on a daily intake of formula made with water containing 10 mg per liter (mg/L) of nitrate as nitrogen. (This reflects the amount of nitrogen within a nitrate molecule, where 1 mg nitrate as nitrogen = 4.4 mg nitrate as measured in the water.) Most cases of infant methemoglobinemia are associated with exposure to formula prepared with drinking water at nitrate-nitrogen levels at least two times higher, or exceeding 20 mg/L nitrate-nitrogen. For nitrite, the RfD is based on a 10-kg (22-pound [lb]) child drinking 1 liter, or about 1 quart, of water every day. The RfD represents a "safe daily dose" and so is compared to the amount an individual is estimated to take in every day, as a ratio. No adverse effects have been linked with inhaling nitrates or nitrites, so reference concentrations (RfCs), which are used to assess inhalation toxicity, have not been developed. The contribution of nitrites, and indirectly nitrates, to potential human carcinogenicity and the magnitude of the associated risk are unclear. Nitrites react with secondary amines in food to form nitrosamines, many of which are carcinogenic in experimental animals and exert other toxic effects. While the EPA has not developed slope factors (toxicity values used to estimate cancer risk) for nitrates or nitrites, some are available for nitrosamines.

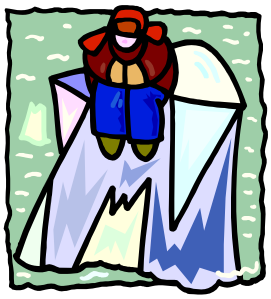
Chemical Toxicity Values	
Non-Cancer Effect	
Oral RfD: NO_3	***Oral RfD: NO_2***
1.6 mg/kg-day	0.1 mg/kg-day

What Are Current Limits for Environmental Releases and Human Exposure? The EPA requires that sodium nitrite releases of more than 100 lb (45.4 kg) and nitrate releases of more than 10,000 lb (4,540 kg) be reported immediately and that normal releases be reported annually for inclusion in the nationwide Toxics Release Inventory. The limits for nitrosamines range from 1 to 10 pounds. The EPA primary drinking water standards for nitrate and nitrite are 10 and 1 parts per million (ppm), respectively. The Food and Drug Administration allows these compounds to be used as food additives as long as they are of food grade and are added only in the amount needed. The maximum amount of nitrite allowed in smoked and cured fish and meat is 200 ppm.

Where Can I Find More Information? More information on nitrates and nitrates can be found in the primary information sources used to prepare this overview: (1) Consumer Fact Sheet on Nitrates/Nitrites, EPA Office of Groundwater and Drinking Water, available through http://www.epa.gov/OGWDW/dwh/c-ioc/nitrates.html; (2) the EPA's Integrated Risk Information System, http://www.epa.gov/iris/subst/0076.htm (nitrate) and /0078.htm (nitrite); and (3) the National Library of Medicine Hazardous Substances Data Bank (http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB).

Basic Concepts for Mixtures Risk Assessment

Why Are Mixtures an Issue? Every day we are exposed to different combinations of chemicals in our homes, at work, in food, and in the environment (air, water, and soil). These mixtures can be composed of tens, hundreds, or even thousands of different chemicals. To further complicate matters, each chemical could have several toxic effects, ranging from cancer to different kinds of noncancer or *systemic* effects. A question commonly asked is: How could the combined chemicals affect my health? Risk assessment has historically looked at effects from single chemicals then simply added the risks together, so in a way this is looking at the "tip of the iceberg" for chemical mixtures. To better evaluate the many cumulative exposures and effects that could be possible, approaches are being developed to assess risks more comprehensively. The U.S. Environmental Protection Agency (EPA) first published risk assessment guidelines for mixtures in 1986, with an update in 2000. The Agency formally defined a cumulative risk policy and offered planning and scoping guidance in 1997, and a cumulative risk assessment framework was published in 2003 following extensive input from many people. Three types of cumulative risk "icebergs" are worth noting: interactions, exposures, and effects.

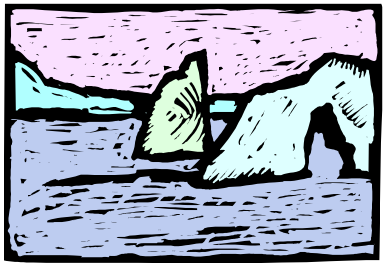


What Are Toxic Interactions? We might understand the toxic effects of certain chemicals in the mixtures to which we are exposed, but we simply don't have this information for all possible mixtures. Where appropriate data exist for the mixture itself, such as diesel exhaust or cigarette smoke, it is evaluated as a whole mixture. Where such data are not available, information about the individual chemicals in the mixture is used, considering their proportions and relative toxicities. We usually assume each chemical would cause the same harm regardless of whether it was in a mixture or by itself. Thus, *dose addition* is our basic assumption, where the toxicity of a mixture is predicted by adding the toxicities caused by the doses of its individual chemicals, as adjusted to account for their relative toxicities. For example, if a mixture has two chemicals in equal amounts and the first is twice as toxic as the second, the toxicity of the mixture would be the same as adding three doses of the first. The special case where the chemicals are toxicologically independent (i.e., cause harm in different ways) is described by *response addition*. Here, the combined toxic response is the same as if the responses caused by the individual chemicals were added. For instance, tranquilizers and alcohol both depress the central nervous system but by different means. If a person is exposed to both, the effect on the central nervous system is the same as the sum of the effects caused by each separately.

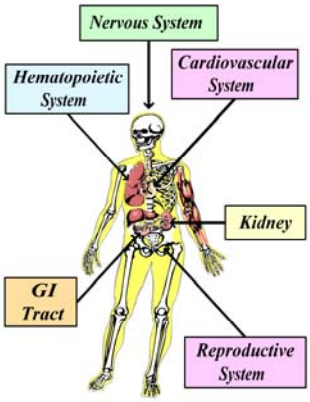
In fact, the toxicity of a mixture might be lower or higher than predicted from the known effects of each chemical acting alone. The influence one chemical has on the toxic effect of another is called a *toxic interaction*. The EPA mixtures guidance identifies toxic interaction as being something other than our default assumption of simple addition. Four types of interactions have been defined, relative to dose addition (see box). A *synergistic* interaction can be illustrated by alcohol and the solvent carbon tetrachloride, each of which harms the liver. Exposure to both damages the liver much more than predicted by simple dose addition. An *antagonistic* interaction can be illustrated by arsenic and lead. Exposure to both causes less harm to the kidney and blood than predicted by dose addition. *Potentiation* can be illustrated by carbon tetrachloride and isopropanol, which is rubbing alcohol (also found in perfume) and is not considered a liver toxin. Inside the body, isopropanol interacts with carbon tetrachloride and causes it to damage the liver more than it would have alone. *Inhibition* is the basis of some antidotes, where you take a dose of a chemical that does not harm you to reduce the harmful effect of another. When risks are estimated by assuming only dose or response addition without considering toxic interactions, perhaps only the tip of the iceberg is being addressed.

Interaction	Definition
Synergism	The combined effect of two or more chemicals is > predicted by dose addition
Antagonism	The combined effect of two or more chemicals is < predicted by dose addition
Potentiation	Exposure to one chemical that is not toxic itself increases the toxicity of second chemical when exposed to both
Inhibition	Exposure to one chemical at a nontoxic dose decreases the toxic effect of another

What Are Mixture Exposures? Some mixtures are manufactured before they are released to the environment (like a specific pesticide formulation), while others are formed during combustion (such as in our car engines) or by other chemical transformations. Chemical interactions in the environment can alter the composition and behavior of a mixture over time. We are exposed to mixtures everywhere. For example, when we breathe outside we can be taking in a mixture of all the air emissions from point sources (such as chimneys and smokestacks) and mobile sources (such as cars, buses, and planes) in the area. This has led many federal programs to look at cumulative exposures at the community scale, by studying releases alone or by combining modeling with emissions measurements. For example, a regional air impact modeling initiative (RAIMI) launched by EPA in 1999 considers risks at the neighborhood level from exposure to multiple air contaminants from multiple sources and pathways. An initial RAIMI study has been completed for an area in Jefferson County, Texas. This type of integrated assessment is being pursued under several national programs. These include the Cumulative Exposure Project (CEP), the National-Scale Air Toxics Assessment (NATA), the Residual Risk Report to Congress, and the Integrated Urban Air Toxics Strategy. These types of assessments look at combined doses from many sources rather than only considering the chemicals from a single location. A full look at this second exposure "iceberg" could include lifestyle-related exposures, such as to exposures to household pesticides, carpeting, or cigarettes.



What Are Mixtures Effects? In addition to considering the combination of chemicals in environmental mixtures and the many ways we can be exposed to them, we also have to realize that each chemical in a mixture could cause multiple health effects. Under the classic risk assessment approach, the *critical* or *primary effect* is generally the most sensitive effect (that first seen as the dose is increased above the level where no adverse effects are observed), and it is often determined from animal studies; this generally serves as the basis for the toxicity value we use to estimate human health risks. However, virtually all chemicals can also have *secondary effects*, which do not occur until we are exposed to larger amounts. Information about primary effects and some secondary effects is contained in EPA's Integrated Risk Information System (IRIS) and in the Agency for Toxic Substances and Disease Registry (ATSDR) toxicological profiles, both of which are available on-line. As science progresses, we will continue to improve our ability to evaluate the role of secondary effects. This is important because in theory, risks calculated based only on critical effects might not be considered a problem if the chemicals in a mixture each impact different organs (e.g., liver) or systems (e.g., nervous system) at tolerable levels. However, the evaluation of possible adverse health effects should extend beyond the chemicals' primary targets to also consider their cumulative impact on other organs or systems that could be harmed as exposures increase. This requires an extensive toxicity evaluation. In a sense, assessing only the primary effects might miss important cumulative secondary effects – a third "iceberg" to keep in mind.



Where Are We Heading? In the past, risk assessments have typically evaluated risks from distinct chemicals, operations, processes, waste streams, or contaminated media. As assessment methods keep pace with emerging scientific knowledge, EPA and others will continue to further evaluate contributions from (1) other pollutants and processes beyond those at the facility being assessed, (2) many sources of a single pollutant in a given community, and (3) the combination of sources, chemicals, and exposures that affect a given community. Interactions among chemicals and the cumulative result of secondary effects are being considered as part of these enhanced assessments. Additional research will better illuminate the often "hidden" components of cumulative health risk from environmental exposures.

Where Can I Find More Information? Key sources include: the EPA mixtures guidance (http://www.epa.gov/ncea/raf/pdfs/chem_mix/chem_mix_08_2001.pdf), EPA cumulative risk framework (http://cfpub.epa.gov/ncea/raf/recordisplay.cfm?deid=54944.), EPA IRIS database (http://www.epa.gov/iris), and RAIMI (http://www.epa.gov/earth1r6/6pd/rcra_c/raimi/raimi.htm), and the ATSDR interaction profiles (http://www.atsdr.cdc.gov/iphome.html).

Mixtures of Arsenic, Cadmium, Chromium, and Lead

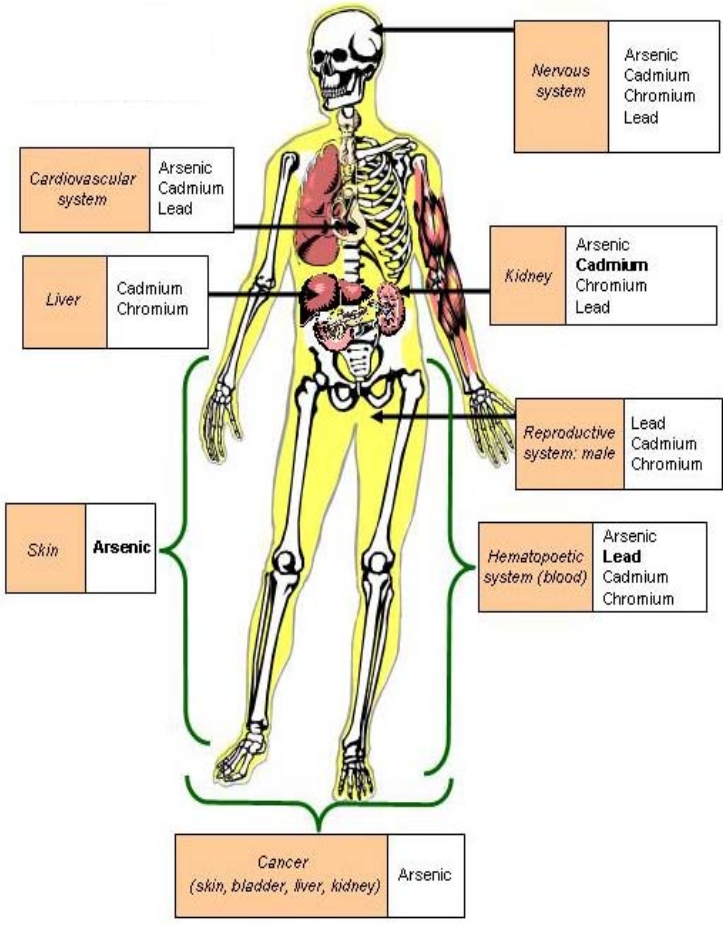
(This fact sheet summarizes information for combinations of metals in this set, with a focus on ingestion of inorganic forms, to support analyses at contaminated sites. Companion fact sheets provide chemical-specific information on common use, general environmental levels, and toxicity, also by other routes.)

Do These Metals Naturally Coexist? Arsenic, cadmium, chromium, and lead all occur naturally in the environment and are found in all materials – soils, plants, animals, and humans – typically as salts. These metals cannot be destroyed, nor do they degrade; however they can be converted to organic forms by biological action both in the environment and within the body. (This fact sheet focuses on inorganic forms.)

What Common Uses Could Result in a Combined Presence? These metals share many industrial uses, including in metallurgy. Arsenic and cadmium are byproducts of lead production, and various combinations are used in materials and processes as highlighted at right. The Agency for Toxic Substances Disease Registry (ATSDR) has identified these four metals as soil contaminants at about 15% of the waste sites reviewed. Amounts and proportions of each vary in content and concentration across different sites.

Uses	Combinations
Metal products, alloys	All four
Dyes, pigments, paints	Chromium, cadmium, lead
Batteries	Cadmium; lead and arsenic
Anti-corrosion/rust coat	Chromium and arsenic
Solder	Lead and arsenic
Wood preservative	Arsenic and chromium

What Health Effects Are Indicated? Chronic oral exposures often represent a route of concern for contaminated sites as contaminants can migrate to groundwater over time. Thus, this fact sheet focuses on ingestion-related effects. Key organs or systems affected by intermediate (subchronic) to long-term (chronic) oral exposures to the individual metals are highlighted at right. Shown are the critical organs and systems (where the first adverse effect is observed, in bold) and those affected at higher levels that are common across two or more metals, or that are known to be affected by another metal in the mixture. (No critical effect has been defined for chromium by the oral route; the respiratory system is the primary target for inhalation.)



Primary Organs/Systems Affected Following Ingestion

What Are The Joint Toxicities? Joint toxicity refers to the outcome of two or more chemicals acting together; three categories of joint toxicity are: greater than additive (synergism and potentiation); additive (no interaction); and less than additive (antagonism and inhibition). Additivity is the default assumption for evaluating health effects of multiple chemicals. Toxicological

interactions can either increase or decrease the apparent toxicity of a mixture relative to that expected from simple addition. No specific health study has been conducted for the quaternary mixture of arsenic, cadmium, chromium, and lead. A few studies have investigated the effects of three metals in combination, but most have studied pairs within this set. The lead-cadmium pair has been studied most, including in human epidemiological studies and oral animal studies. Data from key studies and evidence of interactions as given in the draft ATSDR interaction profile are summarized in the table below.

Joint Toxicity for Selected Organ/System Endpoints Following Ingestion

Endpoint	*Arsenic* on			*Cadmium* on			*Chromium* on			*Lead* on		
	Cd	Cr	Pb	As	Cr	Pb	As	Cd	Pb	As	Cd	Cr
Nervous system			↑			↑				↑	↔	
Kidney	↔	↓	↓	↔	↔	↓	↓	↔		↓	↔	
Hematological system	↓		↓	↓		↓				↓	↔	
Reproductive: male	↓			*na*		↑	*na*			*na*	↑	
Skin	*na*	*na*	*na*		*na*	*na*	↑	*na*	*na*		*na*	
Cancer	*na*	*na*	*na*		*na*	*na*	↑	*na*	*na*		*na*	*na*

As = arsenic, Cd = cadmium, Cr = chromium, Pb = lead; ↑ = interactive effects are more than additive or one metal enhances an effect induced only by the other metal; ↓ = interactive effects are less than additive or one metal protects against an effect induced only by the other metal; ↔ = results are inconclusive or do not suggest that effects are more or less than additive; blank = relevant information is not available; na = not applicable because oral exposure to this metal does not cause the indicated health endpoint. Note that for the cardiovascular system, results were inconclusive or unavailable for all pairs.

For neurological effects, the predicted direction of joint toxic action for the mixture is greater than additive for several pairs (arsenic-lead, cadmium-lead, lead-arsenic), which indicates the health hazard of those mixtures might be somewhat greater than that estimated by endpoint-specific hazard indices for this endpoint; the same higher-than-additive effect is indicated for the male reproductive system (testes) for cadmium and lead acting on each other. In contrast, for effects on the kidney and blood, the predicted direction of joint toxic action is less than additive for several metal pairs, indicating that the health hazard might be somewhat less than estimated by endpoint-specific hazard indices.

What Is the Joint Risk? No specific data exist to quantify the joint risks of the mixture of arsenic, lead, cadmium and chromium. Endpoints of potential concern for this mixture include critical effects of the individual metals as well as the common targets of toxicity that might become significant due to additivity (considering secondary effects) or certain interactions, as indicated in the table above (noting again that additional interactions protect against other adverse effects). The ATSDR recommends using a hazard index method with the target-organ toxicity dose (TTD) modification and qualitative weight-of-evidence (WOE) method to assess the additive and interactive actions of the mixture components. These methods are suggested only when exposures are significant, i.e., only if the hazard quotients for two or more metals are 0.1 or greater. If only one or none of the metals have a hazard quotient at or above that level, then no further assessment of joint toxic action is needed because additivity and/or interactions are unlikely to result in a significant health hazard.

Where Can I Find More Information? More information can be found in the primary information source for this overview: the draft interaction profile for arsenic, cadmium, chromium and lead prepared by ATSDR (http://www.atsdr.cdc.gov/iphome.html). Additional information can be found in the companion fact sheets for each metal and in the sources listed in those fact sheets.

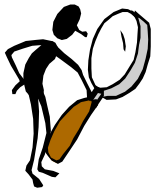


TABLE 1. Mortality and Morbidity Risk Coefficients for Selected Radionuclides[a]

Isotope	Lifetime Cancer Risk					
	Mortality			Morbidity		
	Inhalation	Ingestion	External	Inhalation	Ingestion	External
Americium-241	2.4×10^{-8}	9.5×10^{-11}	1.9×10^{-8}	2.8×10^{-8}	1.3×10^{-10}	2.8×10^{-8}
Americium-242m	1.3×10^{-8}	6.8×10^{-11}	-	1.6×10^{-8}	9.0×10^{-11}	-
Americium-243	2.3×10^{-8}	9.8×10^{-11}	4.3×10^{-7}	2.7×10^{-8}	1.4×10^{-10}	6.4×10^{-7}
Berkelium-247	4.0×10^{-8}	1.2×10^{-10}	2.1×10^{-7}	4.8×10^{-8}	1.6×10^{-10}	3.1×10^{-7}
Cadmium-109	2.0×10^{-11}	4.2×10^{-12}	-	2.2×10^{-11}	6.7×10^{-12}	-
Cadmium-113	8.1×10^{-11}	2.0×10^{-11}	-	1.1×10^{-10}	2.9×10^{-11}	-
Cadmium-113m	9.3×10^{-11}	2.5×10^{-11}	-	1.3×10^{-10}	3.6×10^{-11}	-
Californium-248	2.4×10^{-8}	3.8×10^{-11}	-	2.6×10^{-8}	6.2×10^{-11}	-
Californium-249	4.0×10^{-8}	1.2×10^{-10}	9.3×10^{-7}	4.8×10^{-8}	1.6×10^{-10}	1.4×10^{-6}
Californium-250	3.5×10^{-8}	8.0×10^{-11}	-	3.7×10^{-8}	1.1×10^{-10}	-
Californium-251	4.1×10^{-8}	1.3×10^{-10}	2.6×10^{-7}	4.9×10^{-8}	1.7×10^{-10}	3.8×10^{-7}
Californium-252[b]	2.1×10^{-8}	4.1×10^{-11}	-	2.2×10^{-8}	5.7×10^{-11}	-
Carbon-14	6.5×10^{-12}	1.4×10^{-12}	-	7.1×10^{-12}	2.0×10^{-12}	-
Cesium-134	1.1×10^{-11}	3.5×10^{-11}	4.8×10^{-6}	1.6×10^{-11}	5.1×10^{-11}	7.1×10^{-6}
Cesium-135	1.3×10^{-12}	4.0×10^{-12}	-	1.9×10^{-12}	5.9×10^{-12}	-
Cesium-137	8.1×10^{-12}	2.5×10^{-11}	1.7×10^{-6}	1.2×10^{-11}	3.7×10^{-11}	2.5×10^{-6}
Chlorine-36	9.6×10^{-11}	2.9×10^{-12}	-	1.0×10^{-10}	4.4×10^{-12}	-
Cobalt-57	1.8×10^{-12}	9.0×10^{-13}	2.4×10^{-7}	2.1×10^{-12}	1.5×10^{-12}	3.6×10^{-7}
Cobalt-60	3.0×10^{-11}	1.4×10^{-11}	8.5×10^{-6}	3.6×10^{-11}	2.2×10^{-11}	1.2×10^{-5}
Curium-242	1.4×10^{-8}	3.2×10^{-11}	-	1.5×10^{-8}	5.5×10^{-11}	-
Curium-243	2.4×10^{-8}	8.5×10^{-11}	2.9×10^{-7}	2.7×10^{-8}	1.2×10^{-10}	4.2×10^{-7}
Curium-244	2.3×10^{-8}	7.5×10^{-11}	-	2.5×10^{-8}	1.1×10^{-10}	-
Curium-245	2.4×10^{-8}	9.5×10^{-11}	1.6×10^{-7}	2.8×10^{-8}	1.3×10^{-10}	2.4×10^{-7}
Curium-246	2.4×10^{-8}	9.3×10^{-11}	-	2.8×10^{-8}	1.3×10^{-10}	-
Curium-247	2.2×10^{-8}	9.1×10^{-11}	9.3×10^{-7}	2.5×10^{-8}	1.3×10^{-10}	1.4×10^{-6}
Curium-248[c]	8.8×10^{-8}	3.4×10^{-10}	-	1.0×10^{-7}	4.8×10^{-10}	-
Curium-250[c]	5.0×10^{-7}	2.0×10^{-9}	9.7×10^{-7}	5.8×10^{-7}	2.8×10^{-9}	1.4×10^{-6}
Europium-150	2.1×10^{-10}	3.6×10^{-12}	4.4×10^{-6}	2.6×10^{-10}	6.1×10^{-12}	6.5×10^{-6}
Europium-152	1.5×10^{-10}	5.0×10^{-12}	3.6×10^{-6}	1.9×10^{-10}	8.7×10^{-12}	5.3×10^{-6}
Europium-154	1.7×10^{-10}	8.5×10^{-12}	4.0×10^{-6}	2.1×10^{-10}	1.5×10^{-11}	5.8×10^{-6}
Europium-155	1.7×10^{-11}	1.6×10^{-12}	8.4×10^{-8}	1.9×10^{-11}	2.8×10^{-12}	1.2×10^{-7}
Iodine-129	6.2×10^{-12}	2.0×10^{-11}	-	6.1×10^{-11}	1.9×10^{-10}	-
Iodine-131	2.1×10^{-12}	6.9×10^{-12}	1.1×10^{-6}	1.9×10^{-11}	6.5×10^{-11}	1.6×10^{-6}
Iridium-192	2.1×10^{-11}	6.0×10^{-12}	2.3×10^{-6}	2.4×10^{-11}	1.1×10^{-11}	3.4×10^{-6}
Iridium-192m	1.7×10^{-11}	8.7×10^{-13}	3.7×10^{-7}	2.0×10^{-11}	1.3×10^{-12}	5.4×10^{-7}
Iridium-194m	4.0×10^{-11}	7.3×10^{-12}	6.9×10^{-6}	4.6×10^{-11}	1.3×10^{-11}	1.0×10^{-5}
Neptunium-235	1.0×10^{-12}	2.8×10^{-13}	-	1.2×10^{-12}	5.1×10^{-13}	-
Neptunium-236	2.6×10^{-9}	1.5×10^{-11}	2.2×10^{-7}	3.0×10^{-9}	2.3×10^{-11}	3.2×10^{-7}
Neptunium-237	1.5×10^{-8}	5.8×10^{-11}	5.4×10^{-7}	1.8×10^{-8}	9.1×10^{-11}	8.0×10^{-7}
Nickel-59	3.6×10^{-13}	2.3×10^{-13}	-	4.7×10^{-13}	3.9×10^{-13}	-
Nickel-63	1.4×10^{-12}	5.7×10^{-13}	-	1.6×10^{-12}	9.5×10^{-13}	-

Isotope	Lifetime Cancer Risk					
	Mortality			Morbidity		
	Inhalation	Ingestion	External	Inhalation	Ingestion	External
Plutonium-236	2.1×10^{-8}	6.9×10^{-11}	-	2.3×10^{-8}	9.9×10^{-11}	-
Plutonium-238	3.0×10^{-8}	1.3×10^{-10}	-	3.4×10^{-8}	1.7×10^{-10}	-
Plutonium-239	2.9×10^{-8}	1.3×10^{-10}	-	3.3×10^{-8}	1.7×10^{-10}	-
Plutonium-240	2.9×10^{-8}	1.3×10^{-10}	-	3.3×10^{-8}	1.7×10^{-10}	-
Plutonium-241	2.8×10^{-10}	1.9×10^{-12}	-	3.3×10^{-10}	2.3×10^{-12}	-
Plutonium-242	2.8×10^{-8}	1.3×10^{-10}	-	3.1×10^{-8}	1.7×10^{-10}	-
Plutonium-244[d]	2.7×10^{-8}	1.3×10^{-10}	1.0×10^{-6}	3.1×10^{-8}	1.6×10^{-10}	1.5×10^{-6}
Polonium-210	1.0×10^{-8}	1.6×10^{-9}	-	1.1×10^{-8}	2.3×10^{-9}	-
Potassium-40	2.1×10^{-10}	2.2×10^{-11}	5.5×10^{-7}	2.2×10^{-10}	3.4×10^{-11}	8.0×10^{-7}
Protactinium-231	2.5×10^{-7}	6.0×10^{-10}	1.1×10^{-6}	2.6×10^{-7}	8.8×10^{-10}	1.6×10^{-6}
Radium-226	2.4×10^{-8}	2.9×10^{-9}	5.8×10^{-6}	2.5×10^{-8}	4.0×10^{-9}	8.5×10^{-6}
Radium-228	9.0×10^{-8}	1.3×10^{-9}	8.4×10^{-6}	9.7×10^{-8}	1.9×10^{-9}	1.2×10^{-5}
Samarium-146	1.2×10^{-8}	4.0×10^{-11}	-	1.4×10^{-8}	5.3×10^{-11}	-
Samarium-151	8.6×10^{-12}	4.6×10^{-13}	-	9.2×10^{-12}	8.1×10^{-13}	-
Selenium-79	2.3×10^{-12}	6.7×10^{-12}	-	3.3×10^{-12}	9.7×10^{-12}	-
Strontium-90	1.0×10^{-10}	7.5×10^{-11}	-	1.1×10^{-10}	9.5×10^{-11}	-
Technetium-97	7.6×10^{-13}	2.3×10^{-13}	-	8.5×10^{-13}	3.9×10^{-13}	-
Technetium-98	2.6×10^{-11}	6.0×10^{-12}	4.4×10^{-6}	3.0×10^{-11}	1.0×10^{-11}	6.5×10^{-6}
Technetium-99	1.3×10^{-11}	2.3×10^{-12}	-	1.4×10^{-11}	4.0×10^{-12}	-
Thorium-229	2.2×10^{-7}	4.7×10^{-10}	7.8×10^{-7}	2.3×10^{-7}	7.2×10^{-10}	1.2×10^{-6}
Thorium-230	2.7×10^{-8}	8.0×10^{-11}	-	2.9×10^{-8}	1.2×10^{-10}	-
Thorium-232	4.1×10^{-8}	9.1×10^{-11}	-	4.3×10^{-8}	1.3×10^{-10}	-
Tin-121m	4.1×10^{-11}	2.9×10^{-12}	-	4.4×10^{-11}	5.1×10^{-12}	-
Tin-126	3.9×10^{-10}	3.0×10^{-11}	8.8×10^{-6}	4.2×10^{-10}	5.3×10^{-11}	1.3×10^{-5}
Tritium (H-3)	3.9×10^{-14}	4.4×10^{-14}	-	5.6×10^{-14}	6.5×10^{-14}	-
Uranium-232	1.8×10^{-8}	2.7×10^{-10}	-	1.9×10^{-8}	3.9×10^{-10}	-
Uranium-233	1.1×10^{-8}	6.3×10^{-11}	-	1.2×10^{-8}	9.7×10^{-11}	-
Uranium-234	1.1×10^{-8}	6.1×10^{-11}	-	1.1×10^{-8}	9.5×10^{-11}	-
Uranium-235	9.5×10^{-9}	6.2×10^{-11}	3.7×10^{-7}	1.0×10^{-8}	9.8×10^{-11}	5.4×10^{-7}
Uranium-236	9.9×10^{-9}	5.8×10^{-11}	-	1.0×10^{-8}	9.0×10^{-11}	-
Uranium-238	8.8×10^{-9}	7.5×10^{-11}	-	9.3×10^{-9}	1.2×10^{-10}	-
Zirconium-93	8.4×10^{-12}	1.7×10^{-12}	-	9.2×10^{-12}	2.6×10^{-12}	-

[a] This table provides selected risk coefficients for inhalation and dietary ingestion of various radionuclides, and for external gamma irradiation where that entry is appropriate. (Source: *Cancer Risk Coefficients for Environmental Exposure to Radionuclides*, Federal Guidance Report 13, U.S. Environmental Protection Agency, EPA 402-R-99-001, September 1999.) The mortality risk represents the lifetime risk of incurring a fatal cancer, and the morbidity risk represents the risk of incurring all cancers (fatal and non-fatal). Values are averaged over all ages and both genders. (For context, 10^{-9} is a billionth, 10^{-12} is a trillionth, and a pCi is a picocurie, or a trillionth of a curie.) To convert to standard international units, multiply by 27 pCi per becquerel (Bq). Values shown here include the contributions from short-lived decay products, as indicated in the radionuclide-specific fact sheets. (For example, strontium-90 includes the contribution from yttrium-90, and uranium-238 includes the contribution from thorium-234).

For ingestion and inhalation, units are risk per pCi. For inhalation, the values corresponding to the recommended default absorption type for particulates are shown; the maximum value is given if no absorption type was

recommended. For ingestion, the dietary values shown are the highest for ingestion exposures; values for tap water ingestion are typically 70 to 80% of those for diet. The values for tritium are for tritiated water.

For external exposure, risk coefficients are given for those radionuclides having gamma-ray energies in excess of 0.03 MeV per decay, accounting for the fraction of time that the radioactive decay results in the emission of gamma rays. A dash indicates the radionuclide or its decay products does not emit significant gamma radiation *(*see the companion fact sheet on *Radioactive Properties, Internal Distribution, and Risk Coefficients).* Units for external gamma risk coefficients shown in the table are risk per pCi/g soil for one year of exposure. Although no inhalation or ingestion coefficients are available for krypton isotopes, coefficients do exist for external gamma exposures. Submersion in a cloud of krypton gas poses the highest risk, and the following values are in units of risk per pCi/cm^3 air for one year of exposure. For krypton-81, mortality and morbidity risk coefficients are 1.5×10^{-5} and 2.3×10^{-5}, respectively. For krypton-85, mortality and morbidity risk coefficients are 8.5×10^{-6} and 1.2×10^{-5}, respectively.

[b] Standard risk coefficients are not available for californium-252. To help address this gap, values shown here have been derived by multiplying the standard risk coefficients for californium-250 by the ratios of the dose conversion factors for californium-252 to californium-250, for the given exposure pathways and endpoints.

[c] Standard risk coefficients are not available for curium-248 or curium-250. To help address this gap, values shown here have been derived. For curium-248, standard risk coefficients for curium-246 were multiplied by the ratios of the standard dose conversion factors for curium-248 to curium-246, for the given exposure pathways and endpoints. For curium-250, standard risk coefficients for curium-246 were multiplied by the ratios of the standard dose conversion factors for curium-250 to curium-246, for the given exposure pathways and endpoints. The risk coefficient for external exposure for curium-250 is attributable to its short-lived radioactive decay products.

[d] Standard risk coefficients are not available for plutonium-244. To help address this gap, values shown here have been derived by multiplying the standard risk coefficients for plutonium-242 by the ratios of the dose conversion factors for plutonium-244 to plutonium-242, for the given exposure pathways and endpoints. The risk coefficient for external exposure is attributable to its short-lived radioactive decay products, principally neptunium-240m.

TABLE 2. Summary Radioactive Properties for Selected Radionuclides[a]

Isotope	Half-Life	Specific Activity *(Ci/g)*	Decay Mode	Radiation Energy *(MeV)*		
				Alpha *(α)*	Beta *(β)*	Gamma *(γ)*
Americium-241	430 yr	3.5	α	5.5	0.052	0.033
Americium-242m	150 yr	9.8	IT	0.025	0.044	0.0051
Americium-242	*16 hr*	*820,000*	*β, EC*	-	*0.18*	*0.018*
Americium-243	7,400 yr	0.20	α	5.3	0.022	0.056
Neptunium-239	*2.4 days*	*230,000*	*β*	-	*0.26*	*0.17*
Berkelium-247	1,400 yr	1.1	α	5.6	0.061	0.11
Cadmium-109	1.3 yr	2,600	EC	-	0.083	0.026
Cadmium-113[e]	9.3×10^{15} yr	3.4×10^{-13}	β	-	0.093	-
Cadmium-113m	14 yr	240	β	-	0.19	-
Californium-248	330 days	1,600	α	6.3	0.0060	0.0013
Californium-249	350 yr	4.1	α	5.8	0.044	0.33
Californium-250	13 yr	110	α	6.0	0.0057	0.0012
Californium-251	900 yr	1.6	α	5.8	0.20	0.13
Californium-252	2.6 yr	540	α	5.9	0.0056	0.0012
Carbon-14[e]	5.700 yr	4.5	β	-	0.049	-
Cesium-134	2.1 yr	1,300	β	-	0.16	1.6
Cesium-135	2.3 million yr	0.0012	β	-	0.067	-
Cesium-137	30 yr	88	β	-	0.19	-
Barium-137m (95%)	*2.6 min*	*540 million*	*IT*	-	*0.065*	*0.60*
Chlorine-36	300,000 yr	0.033	β, EC	-	0.027	<0.001
Cobalt-57	270 days	8,600	EC	-	0.019	0.13
Cobalt-60	5.3 yr	1,100	β	-	0.097	2.5
Curium-242	160 days	3,400	α	6.1	0.010	0.0018
Curium-243	29 yr	52	α	5.8	0.14	0.13
Curium-244	18 yr	82	α	5.8	0.086	00017
Curium-245	8,500 yr	0.17	α	5.4	0.065	0.096
Curium-246	4,700 yr	0.31	α	5.4	0.0080	0.0015
Curium-247	16 million yr	0.000094	α	4.9	0.021	0.32
Plutonium-243	*5.0 hr*	*2.6 million*	*β*	-	*0.17*	*0.026*
Curium-248	340,000 yr	0.0043	α	4.7	0.0060	0.0012
Curium-250	6,900 yr	0.21	α, β	1.3	0.0016	-
Plutonium-246 (25%)	*11 days*	*49,000*	*β*	-	*0.13*	*0.14*
Berkelium-250 (14%)	*3.2 hr*	*3.9 million*	*β*	-	*0.29*	*0.89*
Americium-246 (25%)	*39 min*	*20 million*	*β*	-	*0.66*	*0.70*
Europium-150	34 yr	70	EC	-	0.044	1.5
Europium-152	13 yr	180	β, EC	-	0.14	1.2
Europium-154	8.8 yr	270	β	-	0.29	1.2
Europium-155	5.0 yr	470	β	-	0.063	0.061
Iodine-129	16 million yr	0.00018	β	-	0.064	0.025
Iodine-131	8.0 days	130,000	β	-	0.19	0.38
Iridium-192	74 days	9,200	β, EC	-	0.22	0.82
Iridium-192m	240 yr	7.8	IT	-	-	0.16

Isotope	Half-Life	Specific Activity *(Ci/g)*	Decay Mode	Radiation Energy *(MeV)*		
				Alpha *(α)*	Beta *(β)*	Gamma *(γ)*
Iridium-194m	170 days	4,000	β	-	0.16	2.3
Krypton-81	210,000 yr	0.021	EC	-	0.0051	0.012
Krypton-85	11 yr	400	β	-	0.25	0.0022
Neptunium-235	1.1 yr	1,400	EC	<0.001	0.010	0.0071
Neptunium-236	120,000 yr	0.013	β, EC	-	0.21	0.14
Plutonium-236 (9%)	*2.9 yr*	*540*	*α*	*5.8*	*0.013*	*0.0021*
Neptunium-237	2.1 million yr	0.00071	α	4.8	0.070	0.035
Protactinium-233	*27 days*	*21,000*	*β*	-	*0.20*	*0.20*
Nickel-59	75,000 yr	0.082	EC	-	0.0046	0.0024
Nickel-63	96 yr	60	β	-	0.17	-
Plutonium-236	2.9 yr	540	α	5.8	0.013	0.0021
Plutonium-238	88 yr	17	α	5.5	0.011	0.0018
Plutonium-239	24,000 yr	0.063	α	5.1	0.0067	<0.001
Plutonium-240	6,500 yr	0.23	α	5.2	0.011	0.0017
Plutonium-241	14 yr	100	β	<0.001	0.0052	<0.001
Plutonium-242	380,000 yr	0.0040	α	4.9	0.0087	0.0014
Plutonium-244	83 million yr	0.000018	α	4.6	0.0071	0.0012
Uranium-240	*14 hr*	*940,000*	*β*	-	*0.14*	*0.0076*
Neptunium-240m	*7.4 min*	*110 million*	*β*	-	*0.68*	*0.34*
Polonium-208	2.9 yr	590	α	5.1	<0.001	<0.001
Polonium-208	100 yr	17	α	4.9	<0.001	<0.001
Potassium-40[e]	1.3 billion yr	0.0000071	β, EC	-	0.52	0.16
Protactinium-231[e]	33,000 yr	0.048	α	5.0	0.065	0.048
Actinium-227[e]	*22 yr*	*73*	*α, β*	*0.068*	*0.016*	*<0.001*
Thorium-227[e] (99%)	*19 days*	*31,000*	*α*	*5.9*	*0.053*	*0.11*
Francium-223[e] (1%)	*22 min*	*39 million*	*β*	-	*0.40*	*0.059*
Radium-223[e]	*11 days*	*52,000*	*α*	*5.7*	*0.076*	*0.13*
Radon-219[e]	*4.0 sec*	*13 billion*	*α*	*6.8*	*0.0063*	*0.056*
Polonium-215[e]	*0.0018 sec*	*30 trillion*	*α*	*7.4*	*<0.001*	*<0.001*
Lead-211[e]	*36 min*	*25 million*	*β*	-	*0.46*	*0.051*
Bismuth-211[e]	*2.1 min*	*420 million*	*α*	*6.6*	*0.010*	*0.047*
Thallium-207[e]	*4.8 min*	*190 million*	*β*	-	*0.49*	*0.0022*
Radium-226[e]	1600 yr	1.0	α	4.8	0.0036	0.0067
Radon-222[e]	*3.8 days*	*160,000*	*α*	*5.5*	*<0.001*	*<0.001*
Polonium-218[e]	*3.1 min*	*290 million*	*α*	*6.0*	*<0.001*	*<0.001*
Lead-214[e]	*27 min*	*33 million*	*β*	-	*0.29*	*0.25*
Bismuth-214[e]	*20 min*	*45 million*	*β*	-	*0.66*	*1.5*
Polonium-214[e]	*0.00016 sec*	*330 trillion*	*α*	*7.7*	*<0.001*	*<0.001*
Lead-210[e]	*22 yr*	*77*	*β*	-	*0.038*	*0.0048*
Bismuth-210[e]	*5.0 days*	*130,000*	*β*	-	*0.39*	-
Polonium-210[e]	*140 days*	*4,500*	*α*	*5.3*	*<0.001*	*<0.001*
Radium-228[e]	5.8 yr	280	β	-	0.017	<0.001
Actinium-228[e]	*6.1 hr*	*2.3 million*	*β*	-	*0.48*	*0.97*

Isotope	Half-Life	Specific Activity *(Ci/g)*	Decay Mode	Radiation Energy *(MeV)*		
				Alpha *(α)*	Beta *(β)*	Gamma *(γ)*
Thorium-228[e]	*1.9 yr*	*830*	*α*	*5.4*	*0.021*	*0.0033*
Radium-224[e]	*3.7 days*	*160,000*	*α*	*5.7*	*0.0022*	*0.010*
Radon-220[e]	*56 sec*	*930 million*	*α*	*6.3*	*<0.001*	*<0.001*
Polonium-216[e]	*0.15 sec*	*350 billion*	*α*	*6.8*	*<0.001*	*<0.001*
Lead-212[e]	*11 hr*	*1.4 million*	*β*	-	*0.18*	*0.15*
Bismuth-212[e]	*61 min*	*15 million*	*α, β*	*2.2*	*0.47*	*0.19*
Polonium-212[e] (64%)	*0.00000031 sec*	*180,000 trillion*	*α*	*8.8*	-	-
Thallium-208[e] (36%)	*3.1 min*	*300 million*	*β*	-	*0.60*	*3.4*
Samarium-146	100,000,000 yr	0.000024	α	2.5	-	-
Samarium-151	90 yr	27	β	-	0.020	<0.001
Selenium-79	650,000 yr	0.070	β	-	0.056	-
Strontium-90	29 yr	140	β	-	0.20	-
Yttrium-90	*64 hr*	*550,000*	*β*	-	*0.94*	*<0.001*
Technetium-97	2.6 million	0.0014	EC	-	0.0056	0.011
Technetium-98	4.2 million	0.00088	β	-	0.16	1.4
Technetium-99	210,000	0.017	β	-	0.10	-
Thorium-229	7,300 yr	0.22	α	4.9	0.12	0.096
Radium-225	*15 days*	*40,000*	*β*	-	*0.11*	*0.014*
Actinium-225	*10 days*	*59,000*	*α*	*5.8*	*0.022*	*0.018*
Francium-221	*4.8 min*	*180 million*	*α*	*6.3*	*0.010*	*0.031*
Astatine-217	*0.032 sec*	*1.6 trillion*	*α*	*7.1*	*<0.001*	*<0.001*
Bismuth-213	*46 min*	*20 million*	*α, β*	*0.13*	*0.44*	*0.13*
Polonium-213 (98%)	*0.0000042 sec*	*13,000 trillion*	*α*	*8.4*	-	-
Thallium-209 (2%)	*2.2 min*	*410 million*	*β*	-	*0.69*	*2.0*
Lead-209	*3.3 hr*	*4.7 million*	*β*	-	*0.20*	-
Thorium-230[e]	77,000 yr	0.020	α	4.7	0.015	0.0016
Thorium-232[e]	14 billion yr	0.00000011	α	4.0	0.012	0.0013
Tin-121m	55 yr	54	β, IT	-	0.035	0.0049
Tin-121 (78%)	*27 hr*	*970,000*	*β*	-	*0.11*	-
Tin-126	250,000 yr	0.029	β	-	0.17	0.057
Antimony-126	*12 days*	*85,000*	*β*	-	*0.28*	*2.8*
Tritium (H-3)[e]	12 yr	9,800	β	-	0.0057	-
Uranium-232	72 hr	22	α	5.3	0.017	0.0022
Uranium-233	160,000 yr	0.0098	α	4.8	0.0061	0.0013
Uranium-234[e]	240,000 yr	0.0063	α	4.8	0.013	0.0017
Uranium-235[e]	700 million yr	0.0000022	α	4.4	0.049	0.16
Thorium-231[e]	*26 hr*	*540,000*	*β*	-	*0.17*	*0.026*
Uranium-236	23 million yr	0.000065	α	4.5	0.011	0.0016
Uranium-238[n]	4.5 billion yr	0.00000034	α	4.2	0.010	0.0014
Thorium-234[e]	*24 days*	*23,000*	*β*	-	*0.060*	*0.0093*
Protactinium-234m[e]	*1.2 min*	*690 million*	*β*	-	*0.82*	*0.012*
Zirconium-93	1.5 million yr	0.0025	β	-	0.020	-

Isotope	**Half-Life**	**Specific Activity** *(Ci/g)*	**Decay Mode**	**Radiation Energy** *(MeV)*		
				Alpha *(α)*	**Beta** *(β)*	**Gamma** *(γ)*
Niobium-93m	*14 yr*	*290*	*IT*	-	*0.028*	*0.0019*

[a] This table summarizes key radioactive properties of selected radionuclides and their associated decay products, which are indicated in italics. An "e" indicates the isotope exists naturally in the environment. A dash means the entry is not applicable, EC = electron capture, IT = isomeric transition, Ci = curie, g = gram, and MeV = million electron volts. Values are given to two significant figures. See the radionuclide-specific fact sheets for further information, and the companion fact sheet on *Radioactive Properties, Internal Distribution, and Risk Coefficients* for an explanation of terms and interpretation of radiation energies.

TABLE 3. Key References for the Radiological Fact Sheets[a]

Isotope	References
Multiple	Argonne National Laboratory, 1993, Manual for Implementing Residual Radioactive Material Guidelines Using RESRAD, Version 5.0, ANL/EAD/LD-2, working draft for comment, September Argonne National Laboratory, 2001, User's Manual for RESRAD Version 6, ANL/EAD/4, July Eisenbud, Merril, 1987, *Environmental Radioactivity from Natural, Industrial, and Military Sources*, Third Edition, Academic Press, Inc., Orlando, Florida Eisenbud, Merril and Thomas Gesell, 1997, *Environmental Radioactivity from Natural, Industrial, and Military Sources*, Fourth Edition, Academic Press, San Diego, California International Commission on Radiological Protection, 1979, Limits for Intakes of Radionuclides by Workers, ICRP Publication 30, Part 1, Annals of the ICRP, 2 (3/4) International Commission on Radiological Protection, 1980, Limits for Intakes of Radionuclides by Workers, ICRP Publication 30, Part 2, Annals of the ICRP, 4 (3/4) International Commission on Radiological Protection, 1981, Limits for Intakes of Radionuclides by Workers, ICRP Publication 30, Part 3, Annals of the ICRP, 6 (2/3) International Commission on Radiological Protection, 1983, *Radionuclide Transformations, Energy and Intensity of Emissions*, ICRP Publication 38, Annals of the ICRP, 11-13 International Commission on Radiological Protection, 1989, *Age-Dependent Doses to Members of the Public from Intake of Radionuclides*, Part 1, ICRP Publication 56, Annals of the ICRP, 20 (2) International Commission on Radiological Protection, 1989, *Age-Dependent Doses to Members of the Public from Intake of Radionuclides*, Part 2, ICRP Publication 67, Annals of the ICRP 23 (3/4) International Commission on Radiological Protection, 1994, *Dose Coefficients for Intakes of Radionuclides by Workers*, ICRP Publication 68, Annals of the ICRP, 24 (4) National Council on Radiation Protection and Measurements, 1987, *Exposure of the Population in the United States and Canada from Natural Background Radiation*, NCRP Report 94, Washington, D.C., December 30 Shleien, Bernard (editor), 1992, *The Health Physics and Radiological Health Handbook*, Revised Edition, Scinta, Inc., Silver Spring, Maryland U.S. Environmental Protection Agency, 1988, *Limiting Values of Radionuclide Intake And Air Concentration and Dose Conversion Factors For Inhalation, Submersion, And Ingestion*, Federal Guidance Report No. 11, Office of Radiation Programs, EPA-520/1-88-020, September U.S. Environmental Protection Agency, 1993, *External Exposure to Radionuclides in Air, Water, and Soil*, Federal Guidance Report No. 12, Office of Radiation and Indoor Air, EPA 402-R-93-081, September U.S. Environmental Protection Agency, 1999, *Cancer Risk Coefficients for Environmental Exposure to Radionuclides*, Federal Guidance Report No. 13, Office of Radiation and Indoor Air, EPA 402-R-99-001, September U.S. Nuclear Regulatory Commission, 1979, *Final Generic Environmental Impact Statement on Handling and Storage of Spent Light Water Reactor Fuel*, Office of Nuclear Material Safety and Safeguards, Project No. M-4, NUREG-0575, August Information was also obtained from the following Internet addresses: http://atom.kaeri.re.kr/ http://cdfc.rug.ac.be/HealthRisk/default.htm http://environmentalchemistry.com/yogi/periodic/index.html http://ie.lbl.gov/fission/235ut.txt http://micronmetals.com/toc.htm http://nautilus.fis.uc.pt/st2.5/index-en.html http://nobel.scas.bcit.ca/resource/navptable.htm http://pearl1.lanl.gov/periodic/default.htm http://www.bayerus.com/msms/fun/pages/periodic/i_table.html

Isotope	References
Multiple *(cont'd)*	Information was also obtained from the following Internet addresses *(cont'd)*: http://www.cancer.org/docroot/STT/stt_0.asp, (Cancer Facts & Figures 2004), and http://www.cancer.org/downloads/STT/CAFF_finalPWSecured.pdf http://www.census.govhttp://www.principalmetals.com/utilities/periodic.htm http://www.chemicalelements.com/ http://www.chemsoc.org/viselements/pages/pertable_fla.htm (requires Shockwave plugin) http://www.resource-world.net/PerTable.htm http://www.speclab.com/elements/ http://www.ucc.ie/ucc/depts/chem/dolchem/html/elem/elem000.html http://www.umich.edu/~radinfo/ http://www.vcs.ethz.ch/chemglobe/ptoe/index.html http://www.webelements.com/
Americium	International Commission on Radiological Protection, 1986, *The Metabolism of Plutonium and Related Elements*, ICRP Publication 48, Annals of the ICRP, 16(2/3) Radionuclide-specific information was obtained from the following Internet addresses: http://www.encarta.msn.com/find/Concise.asp?ti=017E7000 http://www.world-nuclear.org/info/inf57.htm http://www.encyclopedia.com/printable/00435.html http://www.britannica.com/seo/a/americium/ http://www.uic.com.au/nip35.htm
Cadmium	Radionuclide-specific information was obtained from the following Internet addresses: http://minerals.usgs.gov/minerals/pubs/commodity/cadmium http://atom.kaeri.re.kr http://ntp-server.niehs.nih.gov/htdocs/8_RoC/RAC/Cadmium&cmpds.html http://www.ijc.org/boards/iaqab/meyer/cadmium.htm http://webmineral.com/data/Cadmium.html http://www.speclab.com/elements/cadmium.htm
Californium	International Commission on Radiological Protection, 1986, *The Metabolism of Plutonium and Related Elements*, ICRP Publication 48, Annals of the ICRP, 16(2/3) Radionuclide-specific information was obtained from the following Internet addresses: http://www.britannica.com/nobel/micro/98_61.html http://education.jlab.org/itselemental/ele098.html http://www.chemicalelements.com/elements/cf.html http://www.lenntch.com/Periodic-chart-elements/Cf-en.htm http://www.vcs.ethz.ch/chemglobe/ptoe/_/98.html
Carbon-14	Cember, Herman, 1983, *Introduction to Health Physics*, Pergamon Press, Inc., Elmsford, New York National Council on Radiation Protection and Measurements, 1985, *Carbon-14 in the Environment*, NCRP Report 81, Washington, D.C., May 15 Radionuclide-specific information was obtained from the following Internet addresses: http://www.phschool.com/atschool/chemistry/AW/Student_Area/AWCHEM_SC5_ACT.html http://www.britannica.com/seo/n/nuclear-medicine/ http://crystal.biol.csufresno.edu:8080/projectsF98/535.html http://www.users.globalnet.co.uk/~freya01/carbon14.htm http://seattletimes.nwsource.com/news/health-science/html98/carb_082598.html
Cesium	National Council on Radiation Protection and Measurements, 1977, *Cesium-137 from the Environment to Man: Metabolism and Dose*, NCRP Report 52, Washington, D.C., January 15 Radionuclide-specific information was obtained from the following Internet addresses: http://www.encyclopedia.com/articles/02502.html http://encarta.msn.com/find/Concise.asp?ti=04B4F000

Isotope	References
Chlorine	Radionuclide-specific information was obtained from the following Internet addresses: http://www.encyclopedia.com/articles/02695.html http://www.c3.org/ http://www.cl2.com/benefits/index.html http://www.history.rochester.edu/class/hanford/cdcPaper/onethree.html http://wwwrcamnl.wr.usgs.gov/isoig/period/cl_iig.html
Cobalt	Radionuclide-specific information was obtained from the following Internet addresses: http://www.infoplease.com/ce6/sci/A0812692.html http://www.eco-usa.net/toxics/cobalt.html http://www.atsdr.cdc.gov/tfacts33.html
Curium	International Commission on Radiological Protection, 1986, *The Metabolism of Plutonium and Related Elements*, ICRP Publication 48, Annals of the ICRP, 16(2/3) Radionuclide-specific information was obtained from the following Internet addresses: http://www.encyclopedia.com/articles/03336.html http://www.britannica.com/seo/c/curium/ http://encarta.msn.com/find/Concise.asp?ti=0457F000
Europium	Radionuclide-specific information was obtained from the following Internet addresses: http://www.doegjpo.com/programs/hanf/AXReport/Report/sect03.htm http://minerals.usgs.gov/minerals/pubs/commodity/rare_earths/740301.pdf
Iodine	National Council on Radiation Protection and Measurements, 1977, *Protection of the Thyroid Gland in the Event of Releases of Radioiodine*, NCRP Report 55, Washington, D.C., August 1 National Council on Radiation Protection and Measurements, 1983, *Iodine-129: Evaluation of Releases from Nuclear Power Generation*, NCRP Report 75, Bethseda, Maryland, December 1 Radionuclide-specific information was obtained from the following Internet addresses: http://www.doh.wa.gov/hanford/publications/health/mon10.htm http://cpmcnet.columbia.edu/dept/thyroid/RAI.html
Ionizing Radiation	Agency for Toxic Substances and Disease Registry, 1997 *Toxicological Profile for Ionizing Radiation*, draft for public comment, prepared by Research Triangle Institute, September National Research Council, 1990, *Health Effects of Exposure to Low Levels of Ionizing Radiation*, BEIR V Report, report of the Committee on the Biological Effects of Ionizing Radiations, National Academy Press, Washington, D.C. U. S. Environmental Protection Agency, *Risk Assessment Guidance for Superfund Volume I Human Health Evaluation Manual (Part A)*, Interim Final, EPA/540/1-89/002, Office of Emergency and Remedial Response, Washington, D.C., December
Iridium	Radionuclide-specific information was obtained from the following Internet addresses: http://education.jlab.org/itselemental/ele077.html http://www.ualberta.ca/~slowpoke/1996/r19.html http://www.scn.org/~bh162/iridium.html http://www.nrc.gov/reading-rm/doc-collections/cfr/paro020/appb/ http://www.goodfellow.com/csp/active/static/A/IR00.html http://www.uic.com.au/nip26.htm http://periodic.lanl.gov/elements/77.html http://en.wikipedia.org/wiki/Iridium
Krypton	National Council on Radiation Protection and Measurements, 1975, *Krypton-85 in the Atmosphere – Accumulation, Biological Significance, and Control Technology*, NCRP Report No. 44, Washington, D.C., July 1 Radionuclide-specific information was obtained from the following Internet addresses: http://encyclopedia.com/articles/07092.html http://encarta.msn.com/index/conciseindex/34/0344A000.htm?z=1&pg=2&br=1

Isotope	References
Krypton *(cont'd)*	Radionuclide-specific information was obtained from the following Internet addresses http://www.c-f-c.com/specgas_products/krypton.htm http://www.jamals.com/ehsan/electrical.htm
Neptunium	International Commission on Radiological Protection, 1986, *The Metabolism of Plutonium and Related Elements*, ICRP Publication 48, Annals of the ICRP, 16(2/3) Radionuclide-specific information was obtained from the following Internet addresses: http://nobel.scas.bcit.ca/resource/ptable/np.htm http://encarta.msn.com/find/Concise.asp?z=1&pg=2&ti=761564503 http://search.eb.com/bol/topic?eu=56688&sctn=1 http://www.isis-online.org/publications/fmct/primer/Section_I.html
Nickel	Radionuclide-specific information was obtained from the following Internet addresses: http://encarta.msn.com/find/Concise.asp?ti=066E6000 http://www.nipera.org/pro&use.htm
Plutonium	International Commission on Radiological Protection, 1972, *The Metabolism of Compounds of Plutonium and other Actinides*, ICRP Publication 19, May International Commission on Radiological Protection, 1986, *The Metabolism of Plutonium and Related Elements*, ICRP Publication 48, Annals of the ICRP, 16(2/3) Radionuclide-specific information was obtained from the following Internet addresses: http://www.llnl.gov/csts/publications/sutcliffe/ http://www.pu.org/ http://plutonium-erl.actx.edu/
Polonium	National Research Council, 1961, *The Radiochemistry of Polonium*, P.E. Figgens, Mound Laboratory, Miamisburg, Ohio, National Academy of Sciences, NAS-NS 3037, Washington, D.C. National Research Council, 1988, *Health Effects of Radon and Other Internally Deposited Alpha Emitters*, BEIR IV Report, report of the Committee on the Biological Effects of Ionizing Radiations, National Academy Press, Washington, D.C. Radionuclide-specific Information was obtained from the following Internet addresses: http://www.webelements.com/ http://www.wsu.edu/~wsurso/Isotopes/Pol210.html http://education.jlab.org/itselemental/ele084.html http://www.speclab.com/elements/ http://pearl1.lanl.gov/periodic/default.htm
Potassium	Radionuclide-specific information was obtained from the following Internet addresses: http://www.encyclopedia.com/articles/10439.html http://encarta.msn.com/index/conciseindex/1C/01CB4000.htm?z=1&pg=2&br=1
Protactinium	Radionuclide-specific information was obtained from the following Internet addresses: http://education.jlab.org/itselemental/ele091.html http://www.webspinners.futura.net/apurdy/element_91_100.htm http://www.encyclopedia.com/articlesnew/10559.html http://encarta.msn.com/index/conciseindex/5C/05C22000.htm?z=1&pg=2&br=1 http://140.198.18.108/periodic/Pa.html
Radium	National Research Council, 1988, *Health Risks of Radon and Other Internally Deposited Alpha-Emitters*, BEIR IV Report, report of the Committee on the Biological Effects of Ionizing Radiations, National Academy Press, Washington, D.C. Radionuclide-specific information was obtained from the following Internet addresses: http://www.infoplease.com/ce6/sci/A0840951.html http://www.atsdr.cdc.gov/ToxProfiles/phs9022.html http://www.epa.gov/ttn/uatw/hlthef/radionuc.html

Isotope	References
Samarium	Radionuclide-specific information was obtained from the following Internet addresses: http://minerals.usgs.gov/minerals/pubs/commodity/rare_earths/740301.pdf http://encarta.msn.com/index/conciseindex/0D/00D52000.htm?z=1&pg=2&br=1 http://www.ornl.gov/isotopes/s_sm.html http://www.encyclopedia.com/articles/11397.html http://www.geology.wisc.edu/~jill/samar.html
Selenium	Radionuclide-specific information was obtained from the following Internet addresses: http://www.lbl.gov/MicroWorlds/Wetlands/WetlandsClue1.html http://www.state.sd.us/doa/das/selenium.htm http://www.encyclopedia.com/articles/11665.html http://encarta.msn.com/index/conciseindex/22/0223E000.htm?z=1&pg=2&br=1=1 http://sutekh.nd.rl.ac.uk/cgi-bin/CoNquery?nuc=Se79
Strontium	National Council on Radiation Protection and Measurements, 1991, *Some Aspects of Strontium Radiobiology*, NCRP Report No. 110, Bethesda, Maryland., August 31 Radionuclide-specific information was obtained from the following Internet addresses: http://www.britannica.com/seo/s/strontium/ http://www.epa.gov/iris/subst/0550.htm
Technetium	Radionuclide-specific information was obtained from the following Internet addresses: http://encarta.msn.com/find/Concise.asp?ti=03A19000 http://www.encyclopedia.com/articles/12685.html
Thorium	Archer, V.E., J.K. Wagoner, and F.E. Lundin, 1973, *Cancer Mortality among Uranium Mill Workers*, Journal of Occupational Medicine, *15*:11-14 Burkart, W., 1991, *Uranium, Thorium, and Decay Products*, in Metals and Their Components in the Environment: Occurrence, Analysis, and Biological Relevance, E. Merian (editor), VCH Publications, NY National Research Council, 1988, *Health Risks of Radon and Other Internally Deposited Alpha-Emitters*, BEIR IV Report, report of the Committee on the Biological Effects of Ionizing Radiations, National Academy Press, Washington, D.C. Polednak, A.P., A.F. Stehney, and H.F. Lucas, 1983, *Mortality among Male Workers at a Thorium-Processing Plant*, Health Physics, *44*(Supplement 1):239-251 Stehney, A.F., et al., 1980, *Health Status and Body Radioactivity of Former Thorium Workers*, NUREG/CR-1420 (ANL-80-37), prepared by Argonne National Laboratory, Argonne, Ill., for U.S. Department of Energy and U.S. Nuclear Regulatory Commission, January Radionuclide-specific information was obtained from the following Internet addresses: http://www.atsdr.cdc.gov/ToxProfiles/phs9025.html http://tonto.eia.doe.gov/FTPROOT/nuclear/047896.pdf http://www.thorium-waste.com/Appl.html http://www.thorium-waste.com/Radiation.html http://www.thorium-waste.com/Gensci.html http://encarta.msn.com/find/Concise.asp?ti=01C11000 http://www.infoplease.com/ce6/sci/A0848573.html http://www.britannica.com/seo/t/thorium/ http://www.encyclopedia.com/articles/08646.html
Tin	Zhang, S., et al., 1996, J. Radioanal.Nucl Chem., Letters, 212(2):93-9 Radionuclide-specific information was obtained from the following Internet addresses: http://encarta.msn.com/find/Concise.asp?ti=02ECC000 http://www.itri.co.uk/tinuses.htm http://www.environmentalchemistry.com/yogi/periodic/Sn.html

Tritium	Argonne National Laboratory, 1999, *Risk/Impact Technical Report for the Hanford Groundwater/Vadose Zone Integration Project*, prepared for the U.S. Department of Energy, December Makhijani, A., 1999, *Statement on Tritium before the House Committee on Intergovernmental Coordination, State of Georgia*: Institute for Energy and Environmental Research, October 19, accessed at http://www.ieer.org/comments/tritstmt.html National Council on Radiation Protection and Measurements, 1979, *Tritium in the Environment*, NCRP Report No. 62, Washington, D.C., March 9 U.S. Department of Energy, 1999, *Final Environmental Impact Statement for the Production of Tritium in a Commercial Light Water Reactor*, DOE/EIS-0288, Appendix C, March U.S. Department of Energy, 1999, Final Environmental Impact Zerriffi, Hisham, 1996, *Tritium: The environmental, health, budgetary, and strategic effects of the Department of Energy's decision to produce tritium*, Institute for Energy and Environmental Research, March 20, accessed at http://www.ieer.org/reports/tritium.html
Uranium	Agency for Toxic Substances and Disease Registry, 1990, *Toxicological Profile for Uranium*, TP-90/29, prepared by Syracuse Research Corporation, December Archer, V.E., J.K. Wagoner, and F.E. Lundin, 1973, *Cancer Mortality among Uranium Mill Workers*, Journal of Occupational Medicine, *15*:11-14 Argonne National Laboratory, 1999, *Risk/Impact Technical Report for the Hanford Groundwater/Vadose Zone Integration Project*, prepared for the U.S. Department of Energy, December Berlin, M., and B. Rudell, 1986, *Uranium*, in Handbook on the Toxicology of Metals, 2nd ed., L. Friberg, G.F. Nordberg, and V. Voik (editors), Elsevier Science Publishers, New York Burkart, W., 1991, *Uranium, Thorium, and Decay Products*, in Metals and Their Components in the Environment: Occurrence, Analysis, and Biological Relevance, E. Merian (editor), VCH Publications, New York, pp. 1275-1287 Dreesen, D.R., et al., 1982, *Mobility and Bioavailability of Uranium Mill Tailings Contaminants*, Environmental Science and Technology, *16*(10):702-706 Hodge, H.C., J.N. Stannard, and J.B. Hursh (editors), 1973, *Uranium-Plutonium Transplutonic Elements*, Springer-Verlag, New York Maynard, E.A., and H.C. Hodge, 1949, *Studies of the Toxicity of Various Uranium Compounds When Fed to Experimental Animals*, in The Pharmacology and Toxicology of Uranium Compounds, C. Voegtlin and H.C. Hodge (editors), McGraw-Hill Book Company, New York, Vol. I, pp. 309-376 Morrow, P.E., et al., 1982, *Metabolic Fate and Evaluation of Injury in Rats and Dogs Following Exposure to the Hydrolysis Products of Uranium Hexafluoride*, NUREG/CR-2268, prepared by University of Rochester, Department of Radiation Biology and Biophysics, Rochester, N.Y., for U.S. Nuclear Regulatory Commission, Office of Nuclear Regulatory Research, Washington, D.C., December National Research Council, 1988, *Health Risks of Radon and Other Internally Deposited Alpha-Emitters*, BEIR IV Report, report of the Committee on the Biological Effects of Ionizing Radiations, National Academy Press, Washington, D.C. Radionuclide-specific information was obtained from the following Internet addresses: http://www.britannica.com/seo/u/uranium/ http://www.atsdr.cdc.gov/ToxProfiles/phs9029.html
Depleted uranium (DU)	ATSDR (Agency for Toxic Substances and Disease Registry), 1999, *Toxicological Profile for Uranium. Update,* U.S. Department of Health and Human Services, Public Health Service, September EPA (U.S. Environmental Protection Agency), 1992, *Overview of Air Pathway Assessments for Superfund Sites (Revised), Interim Final*, Office of Air Quality Planning and Standards, Research Triangle Park, N.C. Air/Superfund National Technical Guidance Study Series, Report ASF-1a, Vol. I. November EPA (U.S. Environmental Protection Agency), 1996, *Soil Screening Guidance, Technical Background Document, Appendix B: Route-to-Route Extrapolation of Inhalation Benchmarks*, Office of Solid Waste and Emergency Response, Washington, D.C. EPA/540/R-95/128, July

DU *(cont'd)*	EPA (U.S. Environmental Protection Agency), 2005, *Integrated Risk Information System (IRIS)*. Online database, Substance file for uranium, soluble salts, Office of Research and Development, National Center for Environmental Assessment, http://www.epa.gov/ngispgm3/iris/index.html, August Fisher, D. R., et al., 1994, *Uranium Hexafluoride Public Risk, Letter Report*, PNL-10065, Pacific Northwest Laboratory, Health Protection Department, Richland, Washington, August Gilman, A. P., et al., 1998, Uranyl nitrate — 91-Day Toxicity Studies in the New Zealand White Rabbit. *Toxicol. Sci.* 41(1):129-137, January ICRP (International Commission on Radiological Protection), 1994, *Human Respiratory Tract Model for Radiological Protection*, ICRP Publication 66, Pergamon Press, Oxford, United Kingdom Maynard, E. A., and Hodge, H. C., 1949, Studies of the Toxicity of Various Uranium Compounds when Fed to Experimental Animals. In: *Pharmacology and Toxicology of Uranium Compounds*, National Nuclear Energy Series (VI), pp. 309-376 (Voegtlin, I. C., and Hodge, H. C., Eds.), New York, McGraw-Hill McGuire, S. A. 1991, *Chemical Toxicity of Uranium Hexafluoride Compared to Acute Effects of Radiation*, NUREG-1391, Final Report. U.S. Nuclear Regulatory Commission, Office of Nuclear Regulatory Research, Washington, D.C., February U.S. Nuclear Regulatory Commission, 1994, 10 CFR Part 19, Certification of Gaseous Diffusion Plants Final Rule, Discussion on Section 76.85, "Assessment of Accidents," *Fed. Reg*. 59(184):48944. September 23 U.S. Nuclear Regulatory Commission, 2001, 10 CFR Part 20, Standards for Protection Against Radiation, Appendix B: Annual Limits on Intake (ALIs) and Derived Air Concentrations (DACs) of Radionuclides for Occupational Exposure; Effluent Concentrations; Concentrations for Release to Sewerage
Zirconium	Radionuclide-specific information was obtained from the following Internet addresses: http://encarta.msn.com/index/conciseindex/53/053DF000.htm?z=1&pg=2&br=1 http://www.amm.com/ref/zirc.htm http://search.eb.com/bol/search?type=topic&&query=Zirconium&Dbase=Articles http://www.ornl.gov/isotopes/s_zr.html

[a] This table summarizes key sources of information used to prepare the radiological fact sheets.
CFR = Code of Federal Regulations.