2. RELEVANCE TO PUBLIC HEALTH

2.1 BACKGROUND AND ENVIRONMENTAL EXPOSURES TO AMERICIUM IN THE UNITED STATES

Americium is a human-made, radioactive, actinide element; it has no stable isotopes. The only isotope used commercially is ^{241}Am, several kilograms of which are produced annually. The only other americium isotope produced in macroscopic quantity (10–100 g annually) is ^{243}Am. Both ^{241}Am and ^{243}Am can be formed when either ^{238}U (the major uranium isotope used in nuclear power reactor fuel) or ^{238,239}Pu (primarily used in nuclear weapons) are exposed to neutrons, as occurs in a nuclear reactor or nuclear explosion. Neutron activation of ^{238}U to ^{239}U followed by combinations of decay to ^{239}Np and ^{239}Pu and neutron activation to higher masses of each produces a range of isotopes of these elements. Similarly, neutron activation of ^{238,239}Pu yields higher mass isotopes. Any ^{241}Pu or ^{243}Pu formed by these processes will decay to ^{241}Am and ^{243}Am, respectively. The most common application of americium is in ionization smoke detectors; a typical home smoke detector contains about 0.9 μCi (33.3 kBq) of radioactivity. One gram of $^{241}AmO_2$ provides enough active material for more than 5,000 smoke detectors. Americium is also used in a wide range of industrial gauging applications.

Very low levels of ^{241}Am may be found in environmental media worldwide, a legacy of atmospheric nuclear weapons testing that predominantly took place between 1945 and 1963. In nuclear reactors, ^{241}Am has been detected in primary coolant water, stack aerosols, and waste water (Rosner et al. 1978). Because ^{241}Am is produced from the decay of ^{241}Pu (half-life=14.4 years), releases of ^{241}Pu result in increased environmental levels of ^{241}Am. The maximum concentration of ^{241}Am following an accidental release of ^{241}Pu occurs 70–80 years post release (EPA 1976). Consequently, the impact of ^{241}Am from atmospheric nuclear weapons testing will reach its peak in about the year 2035, when its rate of production from ^{241}Pu decay equals its rate of decay. Environmental levels of ^{241}Am will subsequently decline. Exposure of the general population to ^{241}Am via air, water, soil, and food is generally very low; these 'background' levels are a result of fallout from past atmospheric nuclear weapons tests. Since 1973, ^{241}Am air concentrations have been <1 aCi/m^3 (1 attocurie [aCi]=1x10^{-18} Ci) (0.037 μBq/m^3) and are expected to continue to decline, assuming no significant additional atmospheric nuclear testing after 1976 (Bennett 1979). Levels around nuclear power plants are indistinguishable from fallout background levels (EPRI 1981). ^{241}Am levels in surface seawater of the North Sea and North Atlantic Ocean stayed around

10 Bq/m^3 (270 pCi/m^3) between 1976 and 1988 (Pattenden and McKay 1994), but may be considerably higher near discharges from nuclear facilities. In the Food and Drug Administration (FDA) Total Diet Studies in 1983–1986 and 1987–1992, the concentrations of radionuclides were sufficiently low as to require no specific action or simple monitoring.

2.2 SUMMARY OF HEALTH EFFECTS

Americium exists only in the form of radioactive isotopes; there are no stable forms. Reports of adverse health effects in animals that were administered massive doses of americium are most certainly the result of the ionizing radiation, not the chemical toxicity of americium. The specific activity of ^{241}Am is approximately 17 times greater than that of ^{243}Am. Its higher specific activity and greater abundance make ^{241}Am of greater health concern than ^{243}Am. Both ^{241}Am and ^{243}Am release alpha particles and gamma rays during decay. Alpha radiation is primarily an internal hazard, and the low energy, low intensity gamma radiation is typically not the main health concern. The charged alpha particles generally travel a straight path, interacting with or colliding into other atomic particles. A collision between an atom and an alpha particle can result in a transfer of energy sufficient to "knock out" an electron from the atom, producing an ionized or excited atomic state. Alpha particles have short ranges, and those from americium isotopes are not able to penetrate the outer layers of skin; thus, they are not considered an external hazard. Once americium enters the body via ingestion, inhalation, dermal transport, or a dermal wound, however, the alpha particles that it emits present an internal hazard. The alpha energy induces rapid physical changes to localized cell matter in its path and via chemical interactions with the water in human cells. Radiation hydrolysis of water produces a cascade of rapid chemical reactions, forming ionized and excited chemical species referred to as radiolysis products. These reactive species can interact with biological molecules in ways that can damage cells. Cellular damage can result both directly from radiation interactions and indirectly from the chemical reactions involving reactive species of radiolysis products. The gamma rays emitted during the decay of ^{241}Am are the only components of external radiation exposure, but they represent a small component of the decay scheme and are sufficiently low in energy and intensity so as to pose little hazard from external exposure. Internalized gamma rays can travel greater distances than the heavier alpha particles, but deposit a lower amount of energy with each interaction. Although the gamma rays emitted from the decaying americium atom are of low energy and intensity, their activity and energy could contribute to both localized and more distant cell damage.

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Information regarding health effects in humans following exposure to americium is mainly limited to a case report of an individual accidentally exposed to high levels of americium resulting in a significant internal dose from americium that was absorbed via external wounds. Lymphopenia, thrombocytopenia, and histological signs of bone marrow peritrabecular fibrosis, bone cell depletion, and bone marrow atrophy were noted (Filipy et al. 1995; Priest et al. 1995). These data are supported by findings in laboratory animals exposed to high doses of americium. Elevated hematopoietic activity, leukopenia, decreased hematocrit and platelet levels, and increased cellularity in bone marrow were observed in animals exposed to americium via inhalation (Buldakov et al. 1972; Thomas et al. 1972) or parenteral injection (Dougherty 1970); these effects are likely due to the inability to support hematopoiesis and the reduced colony-forming capacity. Degenerative changes in bone, liver, kidneys, and thyroid have also been observed following inhalation (Moushatova et al. 1996; Thomas et al. 1972) or parenteral (Lloyd et al. 1970; Taylor et al. 1991, 1993a) exposure to ^{241}Am. Comparisons between single exposure and repeated exposure studies suggest that the severity of the tissue damage increases with duration of exposure. Although many of the animal studies employed parenteral injection as the route of exposure, available inhalation data indicate similarity in targets of toxicity. The radiation dose resulting from inhaling large quantities of ^{241}Am can also produce respiratory insufficiency and pneumonia (Buldakov et al. 1972; DOE 1978; Thomas et al. 1972). Experimental studies in animals demonstrate that internal exposure to the radioactive isotope, ^{241}Am, results in the development of cancer in the tissues that sequester this element. Animal studies indicate increased risk of bone cancer in areas of bone containing relatively high levels of americium. Increases in bone cancer have occurred in dogs receiving a single inhalation exposure to $^{241}AmO_2$ (Gillett et al. 1985) and in dogs (Jee et al. 1985; Lloyd et al. 1994a, 1994b), rats (Carter et al. 1951), and mice (Schoeters et al. 1991; Taylor et al. 1983; Van Den Heuvel et al. 1995) receiving a single intraperitoneal or intravenous injection of ^{241}Am. Studies of cancer risk specifically associated with exposure of humans to radioactive americium isotopes were not located. However, the EPA has determined that ionizing radiation is a Group A known carcinogen, and by extension, all radionuclides, including americium, are considered to be known carcinogens.

Based on the outcome of accidental human exposures and experimental animal study data, as well as the application of physiological model simulations, the toxic effects following exposure to americium compounds can be predicted from the associated radiation dose. The potential for adverse health effects occurs when americium enters the body via ingestion, inhalation, dermal absorption, or dermal penetration where radiation from the americium compounds and their radioactive decay products inside the body can cause localized cell damage. Because americium accumulates in the bone and remains there for a long time, the ionizing properties of radiation from ^{241}Am and ^{243}Am can result in damage to the

hematopoietic system if the americium is deposited at a sufficient depth in the bone that its alpha particles can reach the marrow. Although human studies have not correlated americium exposure with an increase in cancer rate, one can calculate a theoretical risk based on dose and values from cancer risk tables. Risk may be significant at high doses, but such high-level exposures are not likely among the general population.

2.3 MINIMAL RISK LEVELS (MRLs)

Inhalation MRLs

No acute-, intermediate-, or chronic-duration inhalation MRLs were derived for americium due to the lack of suitable human or animal data regarding health effects following inhalation exposure to americium.

Oral MRLs

No acute-, intermediate-, or chronic-duration oral MRLs were derived for americium due to the lack of suitable human or animal data regarding health effects following oral exposure to americium.