Plutonium – Health Implications for Man: A Review

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December 2000

Abstract

Sponsored by the Los Alamos Scientific Laboratory, Plutonium – Health Implications for Man, edited by J. W Healy, addresses the concern over the health and environmental hazards of plutonium. It represents a set of articles that covers the influence of plutonium chemistry on its behavior, the exposure pathways, the distribution, metabolism, and transport in the environment. By reviewing the data available as of 1975, the authors attempt to summarize the impact of plutonium on human health, the environment, and evaluate the relevance of the maximum permissible limits. They express uncertainty over the oversimplified metabolic models and the appropriate uptake limits. Listing the numerous gaps in our knowledge of plutonium effects, every author stresses that further research is necessary. Since 1975, most of the assumptions were confirmed by further studies, and the processes described were quantified in various models. The most significant environmental pathway of Pu to man is inhalation followed by ingestion. Absorption through skin is possible only in case of wounds and cuts. Plutonium enters the environment within radioactive particles, migrating through soil and water by mechanical transport. Its transfer to plants and up the ecological chain to humans is insignificant.

Introduction

It is nearly 50 years since plutonium was first produced and isolated by Glenn Seaborg. This element is virtually nonexistent in the earth's natural crust. Created by a man, it is widely used as a fissile material of nuclear weapons and a source of energy in nuclear reactor fuel.

It is wasted mainly as a result of reprocessing (separation from spent nuclear fuel that produces highly radioactive liquid wastes as well as huge volumes of low-level radioactive waste)[1]. After reprocessing and chemical dilution, the liquid wastes are hundreds of times more radioactive than the separated plutonium, and safely storing these wastes is a difficult and expensive task. Another source of contamination is nuclear testing.

As of 1995, there were approximately 270 metric tons of separated plutonium in military inventories and roughly 180 metric tons of separated plutonium in civilian inventories worldwide [2]. In the USA one hundred million gallons of high-level reprocessing waste have been stored in large holding tanks at Savannah River Site, South Carolina, and Hanford Site, Washington.

There is a potential that tanks containing reprocessing wastes might explode. In 1957, a reprocessing waste tank at Chelyabinsk in Russia blew up due to a cooling system failure, spewing a plume of radioactivity 1,000 meters high and contaminating from 15,000 to 23,000 square kilometers.

Plutonium has 15 isotopes with mass numbers ranging from 232 to 246. Only two plutonium isotopes have commercial and military applications and ease of production. Plutonium-238 (the half-life is 87.4 years), which is made in nuclear reactors from neptunium-237, is used to make compact thermoelectric generators; plutonium-239

having a half-life of 24,000 years is used for nuclear weapons and in nuclear reactor fuel.

Plutonium is toxic beyond human experience. Like any radionuclides and, especially ? -emitters, it is demonstratively carcinogenic in microgram quantities. One millionth of gram injected in mice causes local cancer.

The purpose of the book was to review the data on human health and environmental impacts of plutonium available as of 1975, and point out existing problems and bottlenecks. Some of them are still to be addressed. The authors stressed that “little was known definitively about the impact of plutonium and resulting contamination on the health of workers and surrounding communities, though numerous health studies have been undertaken.”

Plutonium in Mammals

Chemical Properties of Biological Importance

The four oxidation states of Pu that exist in aqueous solution are Pu (III), Pu (IV), P(V), and Pu (VI). The oxidation states are stabilized by appropriate agents. All the cations are associated with water to some degree. As a result of hydrolysis of Pu (IV) insoluble polymers might be formed. Small highly charged cations form stable complexes in such biological fluids as citrate, ascorbate, amonoacids and proteins. The most important biological complex of Pu in plasma is that formed with transferrin (TF), the $?_1$-globulin transporting iron. In the mammalian body Pu tends to be in a single oxidation state – Pu (IV) as a result of stabilization by biological fluids, and exists as a

soluble, transportable, and monomeric complex or as an insoluble, colloidal, and polymeric hydrolysis product. The small highly charged Pu ion reacts with water, and the tendency to hydrolyze decreases from Pu (IV) to Pu (VI). The complexes of Pu(IV) with EDTA or DTPA are absorbed almost completely and then rapidly and quantitatively excreted by the kidney. This property is used in the therapeutic removal of Pu.

Most Pu compounds are insoluble. The fluorides, hydroxides, and oxides are very poorly absorbed from the gastrointestinal tract or lung. They cannot form complexes hence they are essentially non-transportable. Such Pu compounds initially deposited in the lungs or beneath the skin might move to local lymph nodes or remain for a long time at the entry point. Some long term dissolution and some transfer via both blood and lymph to other organs is possible.

Absorption

As stated in the book, the main routes of Pu absorption are the lung, gastrointestinal tract, and skin wounds or cuts. Being an ? -emitter, plutonium is stopped by the outer dead layer of skin. Both the amounts and rates of the absorption from the gastrointestinal tract, wound sites, or the lung decrease in the following order: soluble complexes, hydrolizable salts, insoluble compounds.

When introduced as a metabolizable or simple salt, Pu is transported in plasma mainly by the iron-transport protein, transferrin (TF). Circulating as a Pu-transferrin complex, Pu is deposited as single atoms in liver parenchymal cells and the cell mineral interface of the bone surfaces located close to erythropoetic marrow. After the death of hepatic cells, Pu accumulates in liver reticulorendothelian cells. Some of the phagocytized Pu is eventually released from the liver. In case of ingestion or inhalation,

Pu deposits mainly on bone surfaces endosteal) as a result of the transport across cell barriers. Bone surfaces might be resorbed by osteoclasts, dissolved, recirculated and redeposited. The resorbed Pu in bone partially accumulates in osteoclasts of the marrow. Following the death of the osteoclasts, the Pu is taken up by marrow RE cells, which slowly disappear from the marrow.

Gastrointestinal Absorption

Absorption from the gastrointestinal tract is greater in very young animals than in adults. The mechanism by which Pu is absorbed across the intestinal mucosa had not been studied by the time the book was written. However, it was predicted, that some factors might inhibit its absorption as a multivalent cation:

1. hydrolysis of weak complexes and salts in the alkaline intestinal content;
2. reactions with anions such as oxalates and phosphates in the intestinal lumen forming unsoluble compounds;
3. adsorption of multivalent cations onto food particles;
4. the inability of particulate material to penetrate through the intact intestinal epithelium.

Ingestion of Pu results mainly in skeletal deposition. The later studies confirm that the gastrointestinal absorption is not the main route by which it enters the body [2].

Absorption from Parenteral Sites

When injected directly into muscle or subcutaneous tissue, Pu is placed in intestinal fluid. As the concentration of transferrin (TF) is only about one-sixth of those in plasma, only a fraction of Pu can be transported. The remainder hydrolizes and Pu hydroxide precipitates at the site of injection. Subsequent solubilization and

transformations is slow. Translocation of particulate Pu from an implantation site proceeds more slowly by the way of phagositosis and sequestration in local lymph nodes.

Absorption from the Lung

Initially absorption from the lung depends on the size, density, and shape of inhaled particles. Some of the lung deposit is cleared from the body by the mucociliary apparatus, some remains in the lung and its local lymph nodes, and some is solubilized and absorbed into the body. A significant fraction (up to 95%) of complexed Pu or the less easily hydrolyzed salts of Pu (III) or Pu (IV) immediately crosses alveolar membranes into lymph and eventually deposits in the skeleton and liver. Absorption of less soluble forms of Pu proceeds more slowly and is less efficient. After 1975 the data accumulated confirmed that following the deposition in the lung the most mobile Pu complexes are promptly taken up by phagocytic cells either free in alveolar air spaces or fixed in alveolar membranes. Some phagocytized Pu is eliminated from the body by the mucociliary apparatus, but some of the Pu, especially insoluble compounds, remains in the lung for a long time. Eventually the Pu-laden phagocytes die from normal attribution or radiation damage. Their Pu laden debris is taken up by other phagocytes passes into lymph and then into reticuloendothelian cells in local lymph nodes. The process of cell death and phagocytosis is repeated many times. The follow-up studies ascertained that cell debris and intact phagocytes containing Pu escape from lymph nodes [3].

Body Distribution of Soluble Plutonium

When a Pu complex or a simple salt in acid solution is administered by any route, or when Pu can react with TF, the body distributions soon after exposure are similar for a particular species at a given age. The major sites of deposition are skeleton and liver. Parenteral injection results mainly in the deposition in the liver, and oral administration or uptake through the lung leads preferably to skeletal deposition.

Most mammalian studies with Pu were in rats and dogs. Four differences in the distribution and metabolism of Pu were observed in young adults of these two species.

1. The rat skeleton accumulates a larger fraction of absorbed Pu than that of the dog, presumably because the skeleton of an adult rat is never fully mature.
2. A larger fraction of parenterally injected Pu is initially present in the soft tissues (other than liver) of the rat than of the dog.
3. Multivalent cations, including Pu (IV), are efficiently cleared from the rat liver and excreted into the intestine by the bile. The same process from the dog liver proceeded slowly.
4. Urinary clearance of Pu in the rat is about twice of that in the dog.

Tissue Distribution of Soluble Plutonium

Cell fractionation of dog and rat liver sampled after the injection of Pu (IV) showed that most of the Pu was associated with ferritin. Only single atoms of Pu were uniformly distributed throughout the tissue. Soon after the injection of soluble Pu, its concentrations could be tracked in renal papillae. In other tissues Pu was initially widely distributed, associated with blood vessels, and its presence was transient.

Soluble Pu in the skeleton was found mainly on those bone surfaces that were closest to the circulation and most intimately associated with erythropoietic marrow.

Plutonium Deposition and Skeleton Maturity

The higher uptake of Pu in developing mammal bone was demonstrated in 1960th. The body distributions of Pu in most studied species showed that in the growing animals Pu accumulation in soft tissues and the liver was less compared to those in bones. It was mentioned that 1.5-years old dogs did not fit into this scheme. It was proposed that the kinds and amounts of bone surface might be more important in determining skeletal Pu deposition than skeletal age.

The growing mammalian skeleton has not only a greater blood flow an a large surface area compared to the mature skeleton, but the rate of bone resorption is higher. The author proposes that bone resorption activity play the most significant role in the deposition of Pu.

Tissue Distribution of Particulate Plutonium

Upon entry into the body, colloidal particles of any composition are rapidly and completely taken up by Re cells that located in all tissues, but mainly concentrated in liver, spleen, lymph nodes, marrow, and lung. Colloidal preparations of Pu (IV) are deposited mainly in the liver and spleen (75-85%). The results were obtained after the injection of Pu preparations in dogs and rats.

Long Term Fate of Particulate Plutonium

In the 1960's, insoluble Pu was believed to be retained in the lung or transferred to local lymph nodes, slowly rendered soluble, then transferred to blood, and eventually deposited on bone surfaces and in hepatic cell. Later, numerous studies clarified that

particulate Pu can be transported with no solubilization. Particles of Pu were tracked within RE cells. In the case of ingestion, a very large percentage of it will be eliminated from the body quite rapidly in body wastes.

The further studies confirmed that if plutonium oxide is inhaled, part of it, usually between 20 and 60 percent depending upon the size of the particles, is retained in the lung. The rest is eliminated from the body within several days. Of that which remains in the lungs, about half will be removed each year, some to be excreted, some to lodge in the lymph nodes, and a very small amount will cross the gut wall and be deposited in other organs, mainly bone [4].

Long Term Fate of Initially Soluble Plutonium

Formation of the Pu-TF complex in plasma inhibits renal filtration of Pu. Endogenous fecal excretion of Pu in man is also low as was shown by the human metabolic study. Consequently, upon release from an initial deposition site Pu is more likely to be redistributed than excreted.

Within hepatic cells, Pu is associated originally with soluble forms of ferritin. After a few weeks Pu is associated with subcellular structures. The average lifetime of hepatic cells is about 150 days in rats. Upon their death, local phagocytes take up the debris. Its insoluble part accumulates in the RE cells. The slow rate of Pu transfer from hepatic cells at low concentrations of Pu is accelerated by radiation-induced cell death at higher concentrations of Pu. The lifetime of liver RE cells is 50 days in rats. As result, cell debris containing Pu has an option to aggregate or transfer.

Once deposited in bone Pu is believed not to be released until the bone containing it is physically destroyed. The bone surfaces upon which Pu is initially

deposited are not permanent, although in adults of many species bone remodeling rates are quite slow. According to the authors, either new bone might lay down burying the Pu or the Pu containing surfaces might be resorbed by osteoclasts, phagocytic cells of bones. It was proposed that some of the Pu in resorbed bone could be dissolved, recirculated, and redeposited on the bone surface. In this case, the osteoclasts cannot dissolve all the Pu in the resorbed bone, and in time, Pu-laden osteoclasts accumulate in marrow close to the bone surfaces. The number of osteoclasts and the rate at which they accumulate are directly related to the bone resorptive activity. It was suggested that the Pu-labeled material in the marrow phagocytes remained insoluble. After four to six years after injection, the Pu containing phagocytes disappear completely from the marrow. Pu leaves the dog skeleton with a T_b of about 5600 days. Plutonium is eventually released from the liver, skeleton, and other soft tissues of the dog, but the mechanisms of clearance were unknown. The loss of Pu from the liver and skeleton is dose dependent. T_b of Pu in the dog liver is inversely related to dose, while of Pu in the dog skeleton is directly related to dose. The authors mentioned that the above data were not applicable to the metabolism of the very small amounts of Pu, likely to be encountered in occupational or environmental exposures.

All the authors concluded by stressing the necessity for further research. The permissible limits for Pu were based either on an empirical permissible body burden (0.1 µg ^{226}Ra in human bone and the comparison of the toxicity of ^{226}Ra and ^{239}Pu in animals) or a limiting annual radiation dose (15 rem/yr to the lungs). The uptake limits were calculated from a metabolic model. It was mentioned that some uncertainties

caused both the Pu toxicity studies and the metabolic model that did not take into account the Pu long-term behavior.

Human metabolic data are unavailable for Pu. Animal studies are the only source of information, not wholly satisfactory. The studies reviewed provide a general outline of short-term Pu metabolism, but each has one or more defects: “poor analytical or chemical techniques and failure to collect excreta or seek a material balance; uncertainty in the physical state of the Pu compound; a high dose; short post-injection intervals; reliance on short-lived animals, particularly the rat with is perpetually young skeleton; the use of combined dose levels to construct metabolic curves.” The following focuses of further research were proposed:

- More and better data from balance and distribution studies in species other than rats and dogs are needed.
- Study of the chemical analogues of Pu to interpret its metabolism.
- Tracer studies of metabolism in developing and pregnant animals, continuous occupational and environmental Pu exposure, and the movement of Pu in the environment, in plants and animals as well in human food chain.

According to further studies, plutonium that is inhaled is far more hazardous than plutonium that is ingested, because it is more readily absorbed into the blood stream via the lungs than via the gastrointestinal (GI) tract. Inhaled plutonium will deliver a radiation dose to the lungs; ingested plutonium will deliver a radiation dose to the walls of the GI tract [3,4].

Plutonium and the Environment

Distribution of Plutonium in Soils

Plutonium enters the environment as large fragments, submicron particles in aerosols, and aqueous solutions. It was mentioned that only limited information was available about the chemical forms of plutonium released into the environment or about the changes that occurred in these forms after they entered into the environment. Fallout Pu was thought to occur as PuO_2 since the metal rapidly oxidized in the air. Both $^{238}PuO_2$ and $^{239}PuO_2$ are relatively stable and insoluble. The effects of the ecosystem on the chemical and physical forms of Pu has not been extensively studied yet [4].

Pu deposited on the earth surface in either solution or particulate form penetrates within two month to depths of more than 1 cm. The distribution with is frequently well described by an exponential function:

$S_m(z) = S_m(0)\exp(-? z)$,

Where $S_m(z)$ is the activity per unit mass of soil at depth z.

The formula does not take into account the factors influencing the distribution, i.e. soil properties, erosion, and climate.

The authors reviewed the attempts to model the resuspension process, considering them oversimplified. As mentioned, a desirable model must quantify the resuspension process as an upward flux of contaminant aerosol that is a function of the soil contamination per unit area, the soil surface characteristics, vegetation cover, micrometeorological parameters and time since deposition. Soil erosion and sand movement also contribute to Pu redistribution in soils.

The vertical migration of Pu to soil profile depths of 7.5 – 30 cm was noted over a 25-year period at Trinity site. Total quantities of Pu were estimated at 500 kCi ^{239}Pu and about 25 kCi ^{238}Pu in soils as of 1974. As stated in [5], Pu migrates through soil and water by mechanical transport.

Environmental Pathways of Plutonium into Terrestrial Plants and Animals

The ecological behavior of plutonium was also reviewed in the book. The authors stressed that there were only a few data on this subject, as Pu showed a low physiological availability.

External deposition of Pu on plant surfaces is the primary route of Pu movement into vegetation as direct root absorption is low. However, it was proposed that plutonium liquid wastes might be more available for plant uptake because their chemical composition changed pH and chelate content that influence plutonium solubility.

The authors noted higher plutonium concentrations in native grasses compared to those in forbs, shrubs, or trees. The explanation was that the morphological structure of grass presented more surface area for the entrapment of air-borne particulates moving near the ground surface. On the other hand, the physical structure of the roots and their position within the soil profile might be more favorable for Pu uptake. Further studies showed that seed heads of grains serve as a trapping device for deposited material. The authors stressed the necessity to differentiate vegetation contamination mechanisms and their relative importance through time and in different ecosystems.

Ingestion and inhalation were indicated as pathways of plutonium movement into native animals in which the highest concentrations were found in the gastrointestinal tract (GI), pelt, bone, and lungs. Some studies showed measurable concentrations in

the liver, muscle, and bone of rats. Unusually high Pu concentrations were found in the GI contents of birds and explained by their habit to ingest coarse soil particles.

A survey of the Trinity site area for residual plutonium in soils, plants, and animals showed that the concentrations in rodents were about a factor of 10 lower than in corresponding grass samples. It was proposed that residual Pu became less biologically available with time.

Estimates of the relative Pu content of several ecosystems indicated that 96-98% of the Pu was associated with the soil and 1-3% in the root and litter samples. Small relative amounts of Pu are associated with vegetation and animals. The further studies confirmed the generally low concentrations in internal tissues of animals. These facts show that environmental plutonium is relatively unavailable for absorption on a short term basis regardless of the intake mechanism. The physical and biological processes running the Pu movement to soils and plants are being quantified now. The question whether plutonium in plants and animals increase or decrease with time as a result the transformation of Pu into a more biologically available form by weathering process is still to be answered.

The data accumulated by 1997 were used to develop a model to predict the transport and distribution of different radionuclides including Pu in soils and plants [6]. There are two fallout processes of Pu on soils and plants – dry and wet deposition. The first process depends on the aerodynamic resistance, the surface layer resistance, and the transfer resistance. The aerodynamic resistance represents the transport from the free atmosphere to the laminary,. boundary surface layer and influenced by meteorological factors, such as wind speed and turbulence, and the structure of the

surface. The surface area resistance can be described by diffusion, impaction and sedimentation, according to both the size of the particles or material and the structure and condition of the surface itself. Transfer resistance implies the interaction between the deposited material and the surface layer. This process involves chemical and physical interactions, depending on valency, loading, and size of particle. These parameters determine the extent of the transfer into soils or plants. They are partly independent, but are difficult to separate. The amount of deposited nuclides is expressed by the deposition velocity and the concentration in the air near the ground. The dynamic radioecological model, ECOSYS, describing the transfer of radionuclides, including Pu through food chains represents the total dry deposition on plants and soils by:

$D_d(T) = D_p(T) + D_s(T) = v_p\ (T) + v_s c_a$,where

$D_d(T)$ - total dry deposition (Bq/m^2);

$D_p(T)$ - dry deposition on plants(Bq/m^2);

$D_s(T)$ - dry deposition on soil(Bq/m^2);

Wet deposition depends on:

- The water storage capacity of plants, which might change according to the plant's development stage, the plant species, and their morphological characteristics;
- Physical and chemical form of the deposited radionuclide;
- The leaf area index;
- The amount of rain.

Plutonium in Aqueous Systems

Both ^{238}Pu and ^{239}Pu are present in aquatic environments in many areas of the world. The aquatic environment has been the recipient of large quantities of plutonium isotopes as a result of nuclear testing. The Pu which is deposited on land can be transported by erosion to the streams and eventually deposited in lakes, rivers, and oceans. As of 1975, only limited information was available on the interactions of Pu in the natural aqueous environment.

One of the most significant aspects of Pu distribution in the aqueous environment is its physical-chemical states in different conditions. The distribution of sizes among colloidal plutonium species influences the fate of the element in interactions with minerals or other suspended particles in natural waters. Pu can exist in four oxidation states in the aquatic environment: Pu (III), Pu (IV), Pu (V), Pu (VI), depending on the environmental conditions (first of all pH). Under sea water conditions, pH 8.0 – 8.2, Pu presents in the III, IV, and VI valence states, existing as particulate, colloidal, and soluble. As the carbonate ion concentration is usually high in the aqueous systems, one of the soluble species is plutonyl carbonato anion. The amount of plutonium bound in the organic complexes is also significant in the aquatic environment since Pu forms chelates. Under certain conditions, particulate plutonium is removed from water and deposits in the sediments. The “mean residence time” of Pu is difficult to assess because of its different chemical states. If the majority is in the IV or VI valence states or complexed in organic molecules, the mean residence time might be many years. In this case the potential build-up in pelagic organisms is higher. The summarized ^{239}Pu values range between 0.2 and 1.2 fCi/L in sea water. The measurements of Pu in the waters

of Michigan and Ontario gave 0.4 – 1.0 fCi/L in 1972. Some studies showed that only about 3% of the total input of ^{238}Pu was actively cycling, and the remainder was associated with the sediments. In contrast, a homogenous concentration of ^{239}Pu became the same from year to year. Water filtration experiments demonstrated that about 75% of Pu was non-filterable and existed as colloidal or soluble fractions.

Significant bioconcentration of ^{238}Pu and ^{239}Pu was found in fresh water and marine plants. Plankton has the highest concentration factors, and the concentration factors decrease with increasing complexity of the organisms. In fresh and marine water algae measured for ^{238}Pu, the concentration factor ranges between 1000 and 6000. The bioconcentration factors for fish are 14 – 270 (the highest level was observed in bottom feeding fish). The mussel was reported to have exceptionally high concentrations of ^{239}Pu. The marine vertebrates have the lowest concentration factor in the muscle, higher in the liver, and the highest in the backbone.

A systematic understanding of the several complex interactions of Pu in aqueous systems is still to be developed.

Conclusion

Plutonium is of interest as a potential environmental hazard because it is the most abundant of the transuranium elements. The problems associated with the release of plutonium into the environment differ from those of other radionuclides such as ^{90}Sr, ^{137,} Cs or ^{131}I. The plutonium radionuclides are not of concern from the standpoint of external exposure unless direct skin contamination through wounds and cuts or inhalation occurs. The main route of entry is inhalation followed by ingestion. Plutonium

that is inhaled is far more hazardous than plutonium that is ingested, because it is more readily absorbed into the blood stream via the lungs than via the gastrointestinal tract. Inhaled plutonium will deliver a radiation dose to the lungs; ingested plutonium will deliver a radiation dose to the walls of the GI tract. From either of these entry points, plutonium may migrate via the blood stream to selectively concentrate in the bone and liver.

Pu radionuclides are also characterized by a relatively low transfer through food chains to man.

The book also reflects uncertainty inherent to any toxicological study. Twenty-five years have passed, but scientists still refer to the lack of data on many aspects of plutonium behavior that prevents its comprehensive toxicological assessment.

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