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In This Issue

Why ^{210}Po?

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When Alexander Litvinenko, the former detective and critic of the Russian government, was reportedly killed by ^{210}Po in November 2006, health physicists' first reaction must have been "Why ^{210}Po?" We have become accustomed to thinking about ^{239}Pu as the radionuclide of concern. But suddenly we had new concerns: What are the properties of ^{210}Po that would make it poisonous to a person? Why did Litvinenko die so soon after the alleged exposure and with such symptoms? How was it possible that ^{210}Po could be so easily transported? Where was the material produced? Also, what do we know about the effects on people who might have been more casually exposed to ^{210}Po?

To answer these questions and to make more clear the allegations as the press covered this incident, most of us ran to our libraries and the Internet for more information about the properties of ^{210}Po.

We asked Dr. Raymond Guilmette, HPS Past President and an expert in this field, to provide detailed information on the physical, chemical, and biological properties of this radionuclide to help us better understand the whole episode. Following is our interview with Dr. Guilmette.

What is polonium/^{210}Po?

Polonium, element number 84, was discovered by Marie Sklodowska Curie and her husband Pierre in 1898 by purifying it from pitchblende. The Curies had discovered that refined pitchblende was still significantly radioactive after the uranium had been removed, leading them to conclude that at least one other radioactive element was present. The element was named after Marie Curie's native Poland.

Polonium is the first element of the periodic table for which all of its isotopes (a total of 28 with masses ranging from 192 to 218; Stannard 1988) are radioactive. It is a very rare natural element, existing in uranium ores in amounts of about 100 µg per ton of ore. There are seven polonium isotopes that arise from the naturally occurring thorium (^{212}Po, ^{216}Po), actinium (^{211}Po, ^{215}Po), and uranium (^{210}Po, ^{214}Po, ^{218}Po) decay series. Because none of these isotopes has a long half time, they do not accumulate in the natural environment to any significant extent.

^{210}Po, which was called Radium F by the Curies, is an alpha-emitting (5.297 MeV) radionuclide with a half time of 138 days. Only two isotopes of polonium have half times longer than that of ^{210}Po (^{208}Po, 2.9 years, and ^{209}Po, 102 years), but neither occurs in nature.

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Why ^{210}Po?

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Why was research done on the effects of ^{210}Po so early?

My sense, which was confirmed by Bill Bair and information I gleaned from a little Internet searching, is that there was concern for worker safety because of the early use of ^{210}Po for neutron initiators in nuclear weapons. The original polonium project, which was known as the Dayton Project, was undertaken in 1943 to produce the Po-Be neutron sources. In addition to the production aspects, the Dayton project, which was transferred to Mound in 1949, also studied the physical, metallurgical, and biological properties of polonium. The comprehensive toxicological studies, begun in the 1940s at the University of Rochester, also included polonium as one of the important radioelements. The concern over worker protection for polonium mirrored similar concerns that the Manhattan District managers had for plutonium. Knowledge spawned from these early and subsequent studies certainly justified the historical concerns for protecting workers from exposure and health effects from these radioelements, as well as others.

What are the sources of ^{210}Po?

As mentioned previously, ^{210}Po occurs naturally in the environment as a decay product of the uranium series, but its concentration is very low (about 0.1 ppb in uranium ores). It is impractical to obtain commercial quantities by extracting polonium from, for example, uranium ores. A second source of ^{210}Po would be to purify it from aged radium sources. Aging is necessary because a precursor of ^{210}Po, ^{210}Pb, is removed from radium during processing and must grow into the radium source. However, handling large-activity radium sources is not simple because of the relatively high external dose rate, which would require local shielding. The principal commercial method for producing large quantities of ^{210}Po is to neutron-irradiate a bismuth target in a reactor. This procedure activates the stable bismuth into ^{210}Bi, which decays by beta emission with a five-day half time to ^{210}Po. About 8 g of ^{210}Po is shipped to the United States every month from Russia. This is equivalent to 1.33 PBq (36 kCi) per month. There are several companies in the United States that sell static eliminators that use ^{210}Po.

Early Animal Studies with ^{210}Po

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Ray mentions the studies on ^{210}Po that began at the University of Rochester in the early days. Dr. Robert (Bob) G. Thomas was actively involved in this work during the 1950s. His dissertation, published in 1955, was titled "Studies on Polonium in Blood."

Since that time Bob has been the senior author on nearly 50 publications on radionuclide internal exposures. He's retired now, but we wanted to get his input for this article, so we asked him about the early animal studies with ^{210}Po. His reply follows:

Correlation of a chemical form of polonium administered by several routes to a variety of animal species was at the forefront of early experimentation at the University of Rochester. Use of ^{210}Po in the laboratory was with caution, as little was known of its radiobiological effects. Pinpointing the most critical organs for potential damage was a major purpose of these studies.

It was soon discovered that the pH of the injected solution was an important variable and studies with radiocolloids opened a new door for correlating organs at risk with intake. This was particularly important in extrapolating to humans from results with laboratory animals for the purpose of establishing exposure guidelines.

How were the workers protected? Appropriate gloves were always at hand, as were color-coded laboratory smocks and shoe covers. Blood samples for routine hematology were removed from workers on a monthly basis. These, along with routine urine analyses for polonium, were used as biological monitors to determine if any undue exposure may have occurred. No accidental exposures were recorded during these early years, and common sense was the major mind-set for avoidance.

Information about the organs of interest has been published, as has evidence of potential life shortening versus body burdens of ^{210}Po. Although a lethal amount is infinitesimally small, radiation safety practices in the laboratory would prevent the accumulation of even this small an amount by a worker.

What are its physical and chemical properties?

Polonium metal is silvery gray in color and interacts to varying extent with O_2 depending on the temperature. Practically, the metal can be maintained only under inert atmosphere. Because the specific activity of ^{210}Po is so high (166 TBq g^{-1}), milligram quantities will exist at temperatures well above ambient. The electron configuration of neutral polonium resembles those of selenium and tellurium. They and other members of the oxygen family are sometimes called chalcogens. Polonium has stable oxidation states of -2, +2, +4, and +6, of which the tetravalent state is the most stable in solution.

Polonium forms soluble salts with chlorides, bromides, acetates, nitrates, and other inorganic anions. One of its most important chemical properties from the biological perspective is its tendency to hydrolyze and form colloids when there is sufficient mass available. In neutral, weakly acid or alkaline solutions, polonium appears to form a colloid or "radiocolloid." Prevention of colloid formation requires that polonium solutions be maintained in acid solutions that are at least 1N. Polonium can be spontaneously deposited on metals such as silver, copper, nickel, and bismuth or it can be electroplated, for example, on platinum. The latter technique has the advantage of being able to plate polonium in the presence of other elements, for example, bismuth, lead, mercury, and gold.

As mentioned previously, ^{210}Po decays by emitting a 5.297 MeV alpha particle. It also emits a single 0.802 MeV gamma ray, but at a low abundance (1.06×10^{-5}).

Does it have any unusual properties?

Some of the properties of polonium are legendary. The most notable is its tendency to "creep," that is, to disperse itself with time. This is the case whether the polonium is in solution in a beaker, in an unsealed plated source, or as a free powder. The polonium just moves with time and contaminates its local environment. This is attributed to the very high specific activity of ^{210}Po and the resultant recoil energy imparted to the polonium atoms when they decay by alpha-particle emission. This property may help explain why there seem to be so many occurrences of polonium contamination associated with the Litvinenko incident in England.

A second property that early chemists recognized was the tendency of polonium to stick rather avidly to glass, even from dilute acid solution. Once adsorbed, it is very difficult to remove. The problem is alleviated by coating glass containers with paraffin wax or by using plastic containers such as polyethylene.

Another property, which has constrained the sample-preparation techniques that can be used on polonium-containing matrices, is its tendency to volatilize at surprisingly low temperatures. This is particularly the case for polonium halides. Temperatures higher than 100°C are typically not used in radiochemical analyses.

Polonium also has some important unusual biological properties. Bruce Boecker reminded me that Newell Stannard pointed out some of these in his 1988 book, *Radioactivity and Health* (Stannard 1988). In one section, Newell summarized polonium research done at the University of Rochester. Animal research in the early 1960s showed that polonium was very different biochemically and pharmacologically from radium. The studies showed that in contrast to radium which localizes in bone, polonium tends to be a soft-tissue seeker.

Is it easily handled and transported?

I don't have enough knowledge to answer this question, but my gut feeling is that great care needs to be exercised to properly handle and ship this material, both because of the contamination threat and the need to deal with the potentially significant amounts of decay heat. The larger the mass handled, the greater these problems would likely be.

How is it measured in people?

The most sensitive and common method for measuring internal contamination with ^{210}Po is by measuring ^{210}Po in urine. The measurement sensitivity is good, typically about 0.5 mBq. Applying standard ICRP (International Commission on Radiological Protection) models to an acute ingestion intake 10 days prior to a single 24-hour urine bioassay sampling, the 0.5 mBq would correspond to a committed effective dose of 0.3 μSv. However, interpreting such low activity levels of ^{210}Po in urine becomes complicated, particularly in active smokers, as there is "blank" level of ^{210}Po excretion that must be accounted for. For example, Azaredo and Lipsztein (1991) measured an average of 10 mBq d^{-1} in nonoccupationally exposed smokers and 5.2 mBq d^{-1} in nonsmokers. Bioassay for ^{210}Po in feces is another possible intake detection method, but it is even more complicated to interpret than urine because it depends on the route of exposure (ingestion, inhalation, wound) and the levels of dietary intake of ^{210}Po. In addition, the sample processing is more complex and tedious. Dry ashing is out due to the high temperatures needed.

As mentioned previously, ^{210}Po does emit a strong (0.802 MeV) gamma ray, but at a low fractional abundance. However, if the level of intake is high enough, then the ^{210}Po can be measured in vivo. I asked Mac Ennis, manager of our in vivo facility here at Los Alamos National Laboratory, what our minimal detectable activity would be for a 30-minute count using standard germa-

nium detectors. His reply was about 1.9 MBq. So, individuals who have had intakes for which acute health effects are of concern could be monitored quickly using a good in vivo measurement facility.

What are the important routes of human exposure?

Without going into a short course on internal dosimetry, I can summarize the following: There are several portals of entry for radioactive materials, including ^{210}Po. These are ingestion, inhalation, absorption through intact skin, wounds, and intravenous and intraperitoneal injection. The latter two tend to be restricted to medical procedures, whereas the first four are usually associated with unintentional intakes such as might occur in an occupational setting. For a given radionuclide, the distribution of radiation dose will depend on not only the route of exposure, but the in vivo solubility of the exposure material. Restricting our discussion now to alpha-emitting radionuclides, ^{210}Po in particular, the more insoluble the exposure material, the greater will be the dose to the tissue that constitutes the portal of entry (that is, the gastrointestinal [GI] tract for ingestion, respiratory tract for inhalation, skin and underlying soft tissue for wound site). In cases where the radionuclide does not reach the blood in large amounts or takes a long time to do so, the doses to the portal tissues can predominate. Conversely, if the exposure material is soluble, then the critical target organs and tissues are the systemic sites of deposition following absorption to blood.

Which route might be the most damaging will therefore depend on the amount and in vivo properties of the ^{210}Po intake material as well as the radiosensitivity of the target tissues. For example, let's assume that a subject ingested 1 MBq ^{210}Po in a form that was not very soluble ($f_1 = 0.1$ = the fraction of radionuclide in the GI tract that is absorbed into blood). Ninety percent of the ^{210}Po would traverse the GI tract and be excreted in feces and the remainder would be absorbed to blood. Using the ^{210}Po model for adult members of the public in ICRP Report 67 (1993), the calculated absorbed doses two days after intake would be 1.75 mGy for the lower large intestine wall (highest dose to the GI tract components), 3.5 mGy to the kidneys, 1.8 mGy to the liver, 0.7 mGy to the red bone marrow, and 3.0 mGy to the spleen; at 30 days, the doses respectively would be 3.7, 54, 28, 11, and 47 mGy. So, the dose to the GI tract is delivered rapidly, whereas the doses to the systemic organs accumulate more with time after intake and are significantly larger than the dose to the GI tract within a week after exposure.

ICRP Report 67 assumes an f_1 of 0.5 for ^{210}Po incorporated into food. In this case, the systemic organ doses would increase roughly in proportion to the increase in the f_1 value, and the GI tract dose would decrease, but not linearly with the increase in f_1 (this is because a fraction of the dose to the GI tract components is received from ^{210}Po circulating in blood).

Could a significant intake of ^{210}Po be inhaled? I think the answer is yes. Tobacco smokers inhale and deposit ^{210}Po every time they smoke because both ^{210}Po and ^{210}Pb are attached to or incorporated in the tobacco leaves (for more information see, for example, National Council on Radiation Protection and Measurements Report 95). About 1.8 mBq ^{210}Po is inhaled with each cigarette, and about 20 percent of it deposits in the lung. So, ^{210}Po is volatile enough to be entrained with the cigarette smoke. With the very high specific activity of ^{210}Po, it would probably not be difficult to "spike" a cigarette, or several, and use this route to poison an individual. However, I don't know how much of the source ^{210}Po spiked into a cigarette would actually be volatilized and entrained by the smoke particles. And there would be the nasty issue of contaminating environmental surfaces with ^{210}Po from exhaled smoke, cigarette ash, and sidestream smoke.

So, assuming an inhalation route of intake, then the primary determinant of the radiation dose pattern will be the in vivo solubility of the inhaled ^{210}Po. ICRP Publication 71 (1995) lists polonium dose coefficients of Types F, M, and S for members of the public. Similar as with the ingestion case described above, my colleague Luiz Bertelli and I calculated the doses that would result from an inhalation intake of 1 MBq ^{210}Po. At 30 days after intake, the doses for an inhaled Type F compound would be 3.8 mGy for the lung, 137 mGy for the kidney, 71 mGy for the liver, 28 mGy for the red bone marrow, and 118 mGy for the spleen. In comparison, for a Type S compound, the 30-day absorbed doses would be, for the same tissues, 873, 1.5, 0.8, 0.3, and 1.3 mGy respectively. So the in vivo solubility greatly affects the partitioning of dose between the lung and systemic target organs. It appears from media reports that if inhalation was the route of exposure for the Litvinenko case then the ^{210}Po was in relatively soluble form.

To what parts of the body does it go?

Apart from the portal organs, which depend on the route of exposure, the systemic target organs for ^{210}Po are relatively well known, as there have been a significant number of published animal studies on inhaled, ingested, and injected ^{210}Po and absorption through skin. For those wishing to learn more about these experiments, the following references are suggested: Fink (1950), Moroz and Parfenov (1972), and Stannard (1988). Of particular value is the series of 26 papers that was published as a supplement to *Radiation Research*

and summarized the extensive series of experiments conducted at the University of Rochester in the 1940s to 1960s (Stannard and Casarett 1964).

From the American and Russian injection studies, it was learned that the principal organs in which ^{210}Po deposited were, in order of fraction of injected dose, spleen, kidney, lymph nodes, blood cells, liver, and bone marrow, with smaller amounts measured in lung, plasma, skin, testis, muscle, and brain (Fink 1950, and reproduced in Stannard 1988). In rodents, fecal excretion rates were typically 10 times greater than that for urine.

Two mechanisms were found to be important in determining the tissue distribution of ^{210}Po. The first was the tendency for ^{210}Po to hydrolyze and form colloids as solution pH was raised toward neutral. Thus, the distribution patterns observed in the injection studies were influenced significantly by the colloidal nature of the polonium, such that tissues rich in reticuloendothelial or phagocytic cells took up the majority of the ^{210}Po. These organs include the spleen, lymph nodes, liver, and bone marrow. The second mechanism, which explained the avid binding of ^{210}Po to red blood cells, was due to specific binding of polonium atoms to the globin portion of the hemoglobin molecule.

Results from the animal ingestion studies indicated that the distribution pattern was different from that shown with direct injection, in that less ^{210}Po was measured in the reticuloendothelial tissues, and more in the blood. In fact, the blood had the highest concentration and accounted for ¼ to ½ the administered dose at 10 days after gavage (Stannard 1988). This was attributed to differences in the chemical form of ^{210}Po in the GI tract, which probably influenced both the rate at which the polonium atoms were absorbed to blood and also their chemical form. Interestingly, the differences in ^{210}Po distribution between injected and ingested forms was less apparent than with inhalation.

What are the primary health effects of a large exposure? What symptoms would be expected after a large exposure?

A lot of work has been done on the acute effects of ^{210}Po exposure in experimental animals at many different research institutes, primarily in the United States and Russia. The best description and compilation of those results can be found in Newell Stannard's classic book on radioactivity and health (1988). I will summarize some of the important findings here, but for brevity will not include the individual reference citations. These are provided in Stannard (1988).

- Acute lethality from intravenously injected ^{210}Po, in terms of LD_{50} in 20 days, was about 2.6 MBq kg^{-1} body mass in dogs, cats, and rabbits and about 1.5 MBq kg^{-1} for rodents. As the time after exposure was increased, the lethal dose decreased, for example, the 40-day LD_{50} in rats was 1.0 MBq kg^{-1}. Lethality studies were conducted in rats at the University of Rochester out to 300+ days, 500 days in mice at Argonne National Laboratory (ANL), and 600 days at Mound Laboratory. They found that life-shortening continued to occur at these longer times. At ANL, only doses ≤5.6 MBq kg^{-1} in mice showed no lethality to 500 days.
- The tissue damage caused by the alpha-particle radiation of ^{210}Po appears to be in large measure irreparable and irreversible; consequently, total absorbed dose appears to be the best predictor of acute effects. This was demonstrated in studies in which ^{210}Po was administered either as a single injection or as multiple injections spaced out in time to give a more-or-less constant dose rate.
- The different tissue distributions of ^{210}Po that result from different routes of administration did not result in different LD_{50} values. This suggests that the dose patterns to the various soft tissue targets from ^{210}Po sufficiently mimic a "whole-body" radiation pattern that the response becomes organismic. It also suggests that the dose imparted by ^{210}Po in circulating red cells—a prime deposition site—may serve to "homogenize" the dose pattern and concomitant response.
- The primary pathological effects from acute high-dose radiation from ^{210}Po are related to the hematological system, including bone marrow, and the testis and included hypoplasia, atrophy, and hemorrhage. Thus, the symptoms that might arise in a highly exposed individual would mimic those from acute whole-body irradiation.

How would the health effects vary with exposure level?

Having looked at potential dose distributions for ingested and inhaled ^{210}Po, one must now consider how the various irradiated organs would be affected by a given radiation dose. For members of the public who may have had casual contact with a ^{210}Po-contaminated environment (for example, an airplane, restaurant, or bar) and a small intake, the health endpoint of concern would be the lifetime stochastic risk, mainly for late-occurring cancer. The radiation protection guidance from ICRP and other agencies applies directly to these types of intakes and resultant doses. So the published radiation and tissue-weighting factors of ICRP and the standard linear no-threshold dose response model can be applied to the doses calculated from ^{210}Po intake to obtain a committed effective dose, and hence a risk.

However, for the case in which an individual has had a very large intake, such as that surmised for Litvinenko, the projected health impact is not late-occurring cancer, but early-occurring tissue damage and death, that is,

nonstochastic or deterministic risk. Most of us have never had to deal with this type of scenario with internally deposited radionuclides, except in studies with laboratory animals. However, Bobby Scott, PhD, has published a number of papers on this subject including a recent one on ^{239}Pu (Scott and Peterson 2003) and it is expected that he is soon doing the same for ^{210}Po.

Are there any effective treatments after an internal intake? To be effective when would these have to be administered? Are there side effects?

There do appear to be reasonably effective chelating agents for the removal or decorporation of systemically distributed ^{210}Po. But the results of experimental studies do not leave us with a clear picture of what the best therapeutic regimen would be.

To begin, polonium tends to bind most favorably with molecules having sulphydryl groups, as opposed to carboxyl, catechol, or pyridininone groups. Thus, chelators such as EDTA (ethylenediaminetetraacetic acid) and DTPA (diethylenetriaminepentaacetic acid) would not be useful for ^{210}Po decorporation. The Comission of the European Communities-Department of Energy guidebook for the treatment of internal radionuclide contamination (Gerber and Thomas 1992) recommends the use of BAL (British Anti-Lewisite, 2,3-dimercaptopropanol), a lipid-soluble molecule that was developed for treating poisoning with the arsenical gas Lewisite, which was developed between World Wars I and II. It is used for acute poisoning with arsenic, mercury, gold, and lead, as well as antimony, bismuth, chromium, copper, nickel, tungsten, or zinc. Although it has a long history of human use, it does have disadvantages, for example, a low therapeutic index or margin of safety, painful intramuscular injections, and a number of adverse effects.

Two other compounds that are chemical derivatives of BAL, DMPS (2,3-dimercaptopropane sulphonate) and DMSA (meso-dimercapto succinic acid), have also been shown to decorporate ^{210}Po in experimental animals (Volf et al. 1995). Both chelators have been used in humans for heavy metal decorporation (primarily mercury and lead). Both are as effective in removing polonium as is BAL, and with fewer side effects. But none of these compounds has been used yet in humans for polonium decorporation.

Another compound, HOEtTTC (N'N-di[2-hydroxyethyl]ethylene-diamine-NN-biscarbodithioate), which is a derivative of DDTC (diethyldithiocarbamate), has been used to reduce the lethality of ^{210}Po in rats (Rencova et al. 1997). At a ^{210}Po dose that caused 100 percent lethality in 44 days (1.45 MBq kg^{-1}), HOEtTTC produced 90 percent survival for the 150-day length of the study. This compound presently has only been used in animals, but does show promise for future use, provided that adequate pharmacological studies are done.

One aspect of treatment must be emphasized. All the experiments with ^{210}Po decorporation have been done under idealized experimental conditions in which the chelating agent is given within minutes to hours of contamination. This is not realistic in the case of contamination of a member of the public. It will take time to (1) identify that radioactive material has been used, (2) identify what that substance is, and (3) be able to administer the right drug. The longer the delay between the contaminating event and the initiation of therapy, the more radiation dose will have already been delivered, and the less effective will be the treatment in removing ^{210}Po from the body.

What effects might be seen in someone who receives a smaller exposure?

As intakes and doses from ^{210}Po decrease, the risks for acute effects also decrease. As the doses decrease, the time of onset of deterministic disease increases. The animal studies showed that this endpoint could still be observed at least to 500 days after exposure. As the dose decreases even more, then the deterministic risk effectively goes away, and risks for late-occurring cancers replace them, although there is probably an overlap of stochastic and nonstochastic risks over a certain dose range. This overlap has been observed with other radionuclides.

The types of tumors noted in the various published studies in the United States (summarized in Stannard 1988) and Russia (Moroz and Parfenov 1972) showed with reasonable consistency that the tissues that had the combination of highest doses and greatest radiosensitivity had the highest tumor incidences. These included, in general, soft tissue sarcomas and carcinomas, including lymphoma, kidney tumor, mammary tumor, and some reproductive organ tumors. Leukemia incidence in the Rochester studies was low. Again the relative importance of direct deposition and retention of ^{210}Po in specific organs and tissues versus dose received from ^{210}Po in blood cells has not been resolved.

How does one do dosimetry for an exposed person?

Internal dose assessments for persons exposed to ^{210}Po by various routes of exposure would be done the same way that they are done for exposure to other radionuclides. First, if a person was considered at risk for a ^{210}Po intake, then a urine bioassay sample should be

obtained. An interview with the individual would provide information for estimating the probability of intake. Was the person ever in a contaminated environment? How contaminated? When? If there was a possibility of a large intake, then an in vivo measurement might be warranted.

Once the measurement data are acquired, QA'd, and recorded, the dose assessment would be done using the standard ICRP biokinetic models—systemic, inhalation, ingestion as appropriate. If the result of the assessment yields a significant positive dose, then one might consider using a more current systemic polonium model than that of ICRP Publication 30. This newer model, developed by Leggett and Eckerman (2001), is a contemporary recycling model that takes into account all available data, including measurements from Mound workers who were exposed to ^{210}Po. The new model appears to give dose coefficients that are about twice as large as those given by ICRP 30.

If Newell Stannard were still living, what information would you like to get from him about polonium?

Bill Bair related that fortunately Newell recorded much information in his book, *Radioactivity and Health*. He also added some of his personal experiences in his memoirs. His *Radiation Research* supplement and his chapter in BEIR IV are good sources of information. Of course he could add many stories about working with ^{210}Po in the lab of a University Medical School in a large residential area of Rochester.

When I visited with Pat Durbin recently, she mentioned that she thought that Newell would have been saddened that a radioactive material had been used to kill a fellow human. I think she is spot on, as Newell was one of the kindest, most compassionate individuals I have ever met.

Has the recent interest in ^{210}Po taught us anything new about the physical, chemical, and/or biological properties of ^{210}Po?

Bill Bair offered this observation: "I believe it will when we learn the details. It is still a mystery to me how so much ^{210}Po was introduced into the man's body, when you consider that only about 10 percent is absorbed from the gut. Exposing him to an aerosol seems unlikely. Was it injected? If the autopsy collected tissue samples, we might learn more about its distribution; also, if they did autoradiography, we might get some info on polonium-radiocolloids or lack thereof. Considering how difficult it is to contain ^{210}Po, it will be of interest to learn just how widespread the contamination was. Further, there are reports that urine samples from other contaminated individuals contained ^{210}Po. It is not clear how much, but it does raise the question about how it got into their bodies, considering that, as far as I know, it is not readily absorbed through the skin."

What properties of ^{210}Po make it interesting in a who-done-it scenario?

The perceived difficulty in getting a large amount of ^{210}Po, mainly because it needs to be obtained from reactor facilities set up for activation work, gives a nice spin pointing at organizations—rogue countries, organized crime, sophisticated terrorists—that sort of thing. Regarding the material properties, ^{210}Po has very high specific activity, meaning that very little mass is needed to get MBq or GBq levels of activity. The radionuclide has a strong gamma ray, but at a low abundance, so little shielding would be needed for transport. The acute radiation effects resemble those from whole-body penetrating radiation, which gives a well-understood endpoint, particularly for lethality. On the downside, as the Litvinenko case has demonstrated, ^{210}Po gets around, that is, it is hard to contain when handling large amounts.

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Raymond Guilmette, PhD, has been studying the metabolism, biokinetics, dosimetry, and biological effects of internally deposited radionuclides for over 35 years, beginning with his thesis work at New York University studying the decorporation of americum in baboons using DTPA and currently as the team leader for internal dosimetry at Los Alamos National Laboratory. In the interim, he spent 23 years at the Lovelace Respiratory Research Institute, where he was involved in the lifespan plutonium dose-response studies as well as mechanistic studies on the retention of particles in the respiratory tract. He has been keenly interested in decorporation therapy since his early days as a researcher and continues to maintain contact with those active in internal emitter biology. He has also been an active contributor to several NCRP and ICRP committee activities.