CHAPTER 11

Radon Overview

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Radon is a naturally occurring radioactive gas produced by decay of radium. It is ubiquitous, appearing throughout the earth's crust wherever uranium and thorium are found. It is an element that is chemically inactive in the normal environment, not very soluble in water, and is only removed by radioactive decay. In the troposphere, its chemical inertness and predictable rate of disappearance make it an ideal tracer of atmospheric dynamics and air mass movement. Its source as an atmospheric tracer is well known, i.e., the continental land mass, and its loss by decay occurs at a fixed rate, giving it a mean lifetime of 5.5 d. Man and all other living things have evolved in an environment containing radon, so inhaling radon and radon decay products is an occurrence common to all.

Radon began to be recognized as a significant component of indoor air pollution only within the last two decades. It is now understood that inhaling radon decay products provides the dominant component of natural radiation exposure for the general public.[2] Indeed, radon now is considered, along with tobacco smoke, to be the most significant pollution hazard of indoor air.

What is the nature of this health hazard, and why are we just now "discovering" it?

The answer to the second question is that, while radon has been well known as a radiation health hazard for many years, only recently have we realized that it is concentrated in the indoor environment, particularly in private homes, and that these indoor concentrations can greatly exceed the radon concentration in outdoor air.

The answer to the first question requires an understanding of what radon is. When people speak of radon, they usually are referring to the isotope, ^{222}Rn, which is produced by decay of ^{226}Ra, which is a member of the naturally occurring

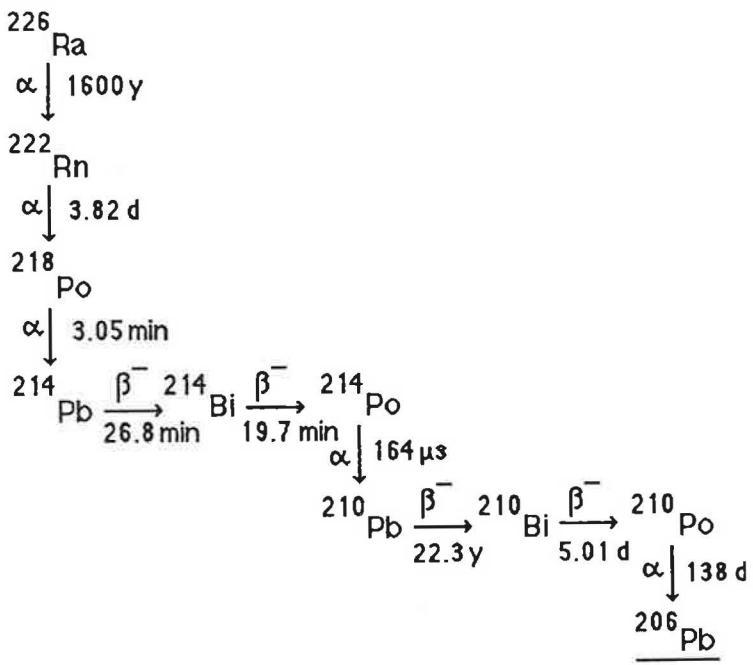

Figure 1. ^{226}Ra and progeny, part of the uranium decay series (main branch), showing decay modes and half-lives.

uranium decay chain originating with ^{238}U. This radon isotope is characterized as an alpha emitter with a half-life of 3.8 d and a mean life of 5.5 d. Its immediate decay product is ^{218}Po, half-life 3.05 min, which decays by alpha emission to produce ^{214}Pb. The decay series is depicted in Figure 1.

When ^{218}Po or one of its mass 214 progeny is inhaled, it is usually carried into the respiratory system in or on an aerosol particle. These particles most likely stick to the inner surfaces of the lungs or the bronchial passage and do not leave the body. Thus, as they undergo their natural process of radioactive decay, each particle of ionizing radiation emitted by the decaying isotopes causes radiation damage to the surrounding tissue. Since ^{210}Pb has a mean lifetime of 32 years, a long time can elapse between the time the radioisotope enters the body and its ultimate disappearance by radioactive decay. These longer-lived radon progeny can accumulate in the body over a long period of years before the results of the radiation exposure begin to be evident. Such radioisotopes often have time to move within the body to sites quite different from their original site. Radioactive lead, for example, might end up collecting in bone tissue if it is not retained in the lungs or bronchial passages.

Another radon isotope, not as well known and not as well studied as ^{222}Ra, is ^{220}Rn, commonly known as thoron since it is a member of the thorium decay series

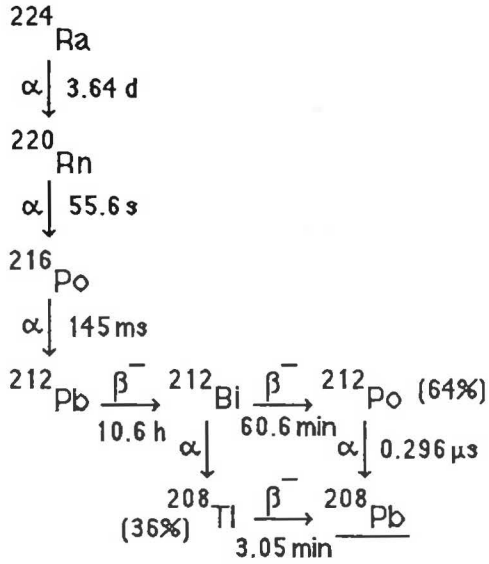

Figure 2. ^{224}Ra and progeny, part of the thorium decay series, showing decay modes and half-lives.

originating with ^{232}Th. Thoron has a half-life of only 56 s; therefore, it enters the atmosphere almost entirely by diffusion through the top few centimeters of soil.[3] Its decay by alpha particle emission produces ^{216}Po (half-life 150 msec) which immediately releases another alpha to produce ^{212}Pb (half-life 10.6 h), the longest lived radionuclide in the thoron decay chain (see Figure 2).

Both ordinary radon (^{222}Rn) and thoron can enter the indoor environment primarily through cracks in the floor and walls of the basement or any parts of the house in contact with the soil and rocks.[4] Entry can also occur via utilities, such as water, or building materials that contain traces of radium and uranium.

Radon can spread throughout a building, decaying and producing its aerosol-borne progeny everywhere. Thoron, because of its very short lifetime, is usually confined to the space where it first entered the building, but its ^{212}Pb and ^{212}Bi descendants can still be carried as aerosols into other parts of the building.

When inhaled, the mass-212 thoron daughters represent a radiation hazard to the body similar to the mass-214 radon progeny, except the delayed hazard represented by radon's mass-210 radioisotopes is non-existent in the case of thoron. This can be important, depending on how easily such progeny can diffuse through cell walls, enter the bloodstream, and be circulated to other sites within the body. There is an opportunity, especially for radon progeny, to cause radiation damage to other organs besides the lungs and bronchial passages. Thus, it might not be surprising to learn that radon exposure has been cited as the possible cause of other kinds of cancer than lung cancer.[4]

Now, let's take a closer look at the physics and chemistry that takes place when a radon or thoron atom undergoes radioactive decay. This decay is a very energetic event that results in the production of an atom different from the parent. The energy that is exchanged and the processes that occur while the new atom is adjusting to its environment can result in some unusual chemical and physical changes. When radioactive decay occurs in air, the new metal atom formed is likely to carry a positive charge and to be very reactive with any chemical species encountered. It is known that most of these radioactive products of radon decay end up as aerosol particles, i.e., submicroscopic particles that are light enough to remain airborne for long periods of time before eventually settling out on walls, floors, and other surfaces.

As a result of alpha decay, the ^{218}Po is very likely "born" with a high positive charge arising from Auger ionization and the severe perturbations caused by a sudden disruption of the nuclear charge with concomitant disturbances of the electronic orbitals of the atom. The ion also is born with lots of kinetic energy (~110 kev) because of conservation of momentum when the alpha particle is emitted.

After the ^{218}Po is born, it is a recoil nucleus that is positively charged. Thus, as a fast, heavy, charged particle, it causes ionization, excitation, and dissociation along its path in the medium in which it is produced. The recoil species regains electrons from the surroundings as it slows down near the end of its recoil path. It thus becomes thermalized, either as an ion or a neutral atom, at the end of a dense column of ion pairs and fragments with which it may react if it diffuses back in the direction that it came from. Experimental data show that about 88% of the ^{218}Po species reach the ends of their recoil paths and begin diffusing as singly charged cations. Neutralization of these cations can occur by reaction with a stray electron, by combination with a negatively charged ion, or by charge transfer involving a collision partner having a lower ionization potential than the recoil product.

Regardless of its state, whatever chemical product is formed is disrupted (destroyed) when the ^{218}Po species undergoes alpha decay to produce ^{214}Pb. The resulting lead isotope becomes another recoil ion with the same chances for neutralization or chemical combination as just described for polonium, except for minor differences in ionization potentials and recoil energies. The alpha decay process is so violent that one would not expect the final chemical state of ^{214}Pb to be at all related to the chemical state of its parent.

On the other hand, the same is not necessarily the case for ^{214}Bi produced by beta decay of ^{214}Pb. The beta decay process, while disruptive of the electronic orbitals surrounding the parent atom, is much less violent, kinetically. Recoil kinetic energies in beta decay can be zero.

The expected or predicted results for ^{214}Bi and ^{214}Po are that these species can remain on or within the aerosol particles in which they are produced. It should not be surprising, therefore, to find these radon progeny carried on larger aerosol particles than the ^{218}Po. It appears that these general expectations are borne out by experiment.

Chapters 12 and 13 in this book represent important efforts to describe the fate of ^{218}Po, both chemically and physically, in recognition of the fact that, without understanding these fundamental processes, we do not have a very good understanding of how to deal with these products of radon decay once they appear in the indoor air. The subsequent behavior of these short-lived radon progeny (and similar products of thoron decay) is dependent on their chemical and physical state.

Since the mass-214 lead and bismuth radionuclides have mean lifetimes on the order of approximately a half hour, there are numerous opportunities for things to happen to the aerosol particles bearing these isotopes before they decay. The principal hazard due to the presence of radon in indoor air is due to these aerosols that can be inhaled or ingested before the particles are deposited on the walls or other surfaces.

If these radionuclides are carried into the lungs or bronchial passages before their decay occurs, then their chemical and physical characterization is important in determining what happens in that new environment. If the particles are small enough and are not deposited in the respiratory system, they can be exhaled before they decay, much as would be expected for radon itself.

If the particles are not immediately exhaled, they will stick to the interior surfaces of the lungs or bronchial passages. Depending on whether or not the particles are water soluble they may dissolve and the radionuclide passed into the bloodstream. If the isotope survives long enough, it may be deposited somewhere else in the body before decay occurs. Regardless of where the radioisotope is when it decays, however, there is a significant risk of radiation damage to the surrounding cells or tissue. Damage to the body due to ionizing radiation can be carcinogenic; therefore, cancer is a risk whenever radionuclides of any kind decay inside the body.

If radon progeny are inhaled, there is a significant risk that they will initiate lesions that are malignant. In many instances, the radiation damage results only in destroying cells and producing mutations that are lethal to the cell (and thus not replicated) or producing nonmalignant and nonlethal mutations that the body can tolerate.

Thus, in order to address the hazards of indoor radon, it is necessary to understand how the radon progeny diffuse or are transported through the air; how they deposit on surfaces and, if inhaled or ingested, how they behave when deposited inside the body. In addition, in order to accurately assess the ultimate risk, it is necessary to understand how the ionizing radiation produced by radioactive decay of these particles interacts with the lungs or whatever tissue is near the site where decay occurs.

Chapter 14 addresses the question of sorption of radon on porous materials. The ability of radon to adsorb and then be desorbed before its decay may affect its abundance and transport in indoor air where there are lots of available surfaces for adsorption to occur. It is unlikely that radon atoms decaying on a wall will contribute to the indoor radon hazard unless some process occurs by which dust

particles containing the radioactive progeny can be released from the wall surface to become airborne.

Chapter 15 discusses the problems of determining the rate of emanation of radon from a surface. This may apply to the determination of the emanating power of various soils in contact with a basement, for example, or it might apply to the release of radon from surfaces of dry wall or other construction materials indoors. The ease with which radon is released from such surfaces determines their effectiveness as potential sources of indoor radon.

Finally, Chapter 16 addresses one of the problems of experimentally determining the amount of airborne radon progeny present. Specifically, the collection and counting efficiency of wire screens is discussed. Such screens have the capability of determining not only the total number of radon progeny collected, but also the distribution of particle sizes on which the radionuclides are carried. This information is useful in predicting dry deposition rates as well as determining rates of diffusion and probabilities of sticking inside the bronchial passages and lungs.

Thus, the section of this book devoted to indoor radon addresses the important scientific questions that must be understood in order to be able to proceed with the development of effective means of control and remediation.

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