Radon Occurrence and Health Risk*

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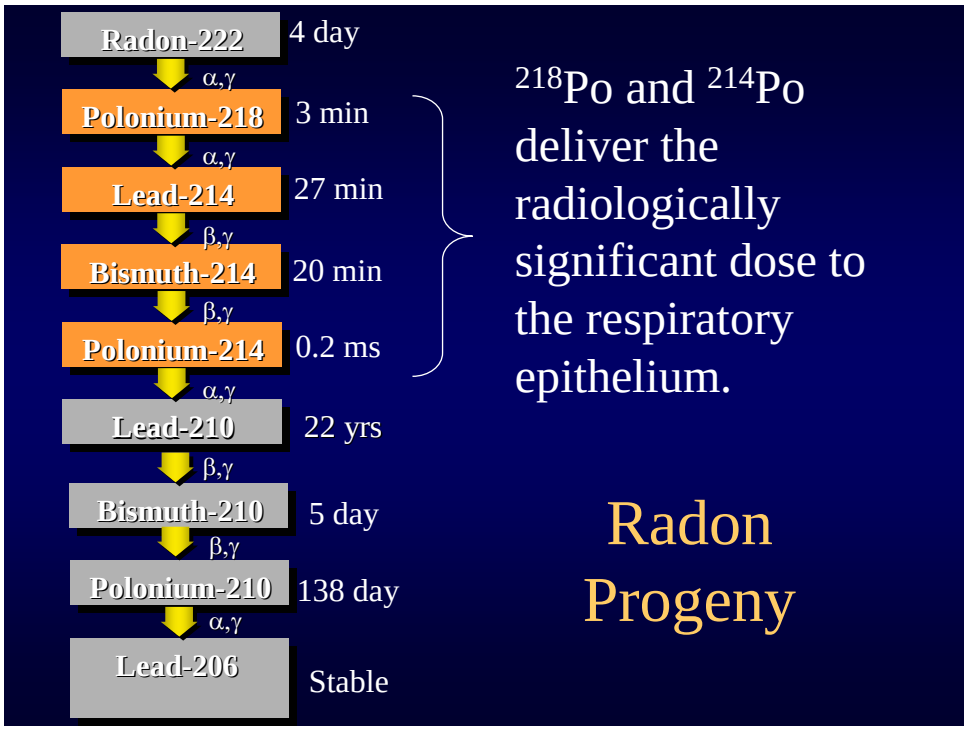
1. What are the chemical and radiologic characteristics of radon?

Radon is a colorless, odorless, tasteless, radioactive noble gas that generally lacks activity toward other chemical agents. However, radon occasionally forms clathrate compounds with some organic compounds and may form ionic or covalent bonds with highly reactive elements such as oxygen or fluorine. Radon is the heaviest noble gas and exhibits the highest boiling point, melting point, critical temperature, and critical pressure of all noble gases. Radon is highly soluble in nonpolar solvents and moderately soluble in cold water. Radon's isotopes, all of which are radioactive, include mass numbers ranging from 200-226. Radon-222 (^{222}Rn), formed in the ^{238}U decay chain, is the most important isotope because of its relatively long half-life of 3.82 days. The short half-life beta- and gamma-emitting decay products of ^{222}Rn achieve equilibrium with the parent isotope within several hours.

2. **Draw the ^{226}Ra decay chain. Include the half-life and major emission type of each decay product. Also, what are ^{222}Rn progeny and why are they important?**



Radon progeny is another name for the ^{222}Rn decay products or ^{222}Rn daughters. Radon progeny rather than ^{222}Rn gas deliver the actual radiation dose to lung tissues. The solid airborne ^{222}Rn progeny, particularly ^{218}Po, ^{214}Pb, and ^{214}Bi, are of health importance because they can be inspired and retained in the lung. The radiation released during the subsequent decay of the alpha-emitting decay products ^{218}Po and ^{214}Po delivers a radiologically significant dose to the respiratory epithelium. The ratio of progeny to ^{222}Rn gas ranges from 0.2-0.8 with a typical value of 0.4. The ratio between progeny and ^{222}Rn gas is called the equilibrium factor.

3. How does the behavior of ^{222}Rn progeny in air affect the dose delivered to lung tissues?

After decay of the ^{222}Rn gas, a high percentage of the decay products attaches to ambient aerosols. A small percentage of the decay products remain unattached; others increase their diameter through chemical and physical processes. The percent attachment depends on numerous factors, including the size and concentration of the airborne particles. The size and density of a particle determine its behavior in the respiratory tract. The unattached particle fraction with a 1-nm diameter is generally removed in the nose and mouth during breathing and has limited penetration of the bronchi. Maximal deposition occurs as the particles with diameters ranging from 3-10 nm increase their rate of penetration through the mouth and nose, ultimately depositing in the bronchial region. The deposition rate decreases for particles as their diameter increases toward 100 nm and larger because the particles are less able to diffuse to the airway

surface. However, particle deposition into the respiratory tract through impaction starts to increase again for particles above 500 nm. Larger particles with a diameter exceeding 3.5 μm deposit predominantly in the nose and mouth during inhalation and do not reach the sensitive respiratory epithelium.

4. List the physical and biologic factors that affect the dose delivered by ^{222}Rn progeny to the target cells in the respiratory epithelium.

- Aerosol size distribution
- Equilibrium between ^{222}Rn gas and ^{222}Rn progeny
- Respiratory rate
- Lung tidal volume
- Oral vs. nasal inhalation route
- Bronchial morphometry
- Clearance rate from lung
- Thickness of mucus in respiratory tract

5. When was the link between ^{222}Rn exposure and lung cancer first postulated?

As early as 1556, Agricola described high mortality rates from respiratory diseases among underground metal miners at Schneeberg in the Erz Mountains of Central Europe. Harting and Hesse first linked the high mortality rates at Schneeberg to lung cancer in 1879 on the basis of autopsy findings. In 1921 Margaret Uhlig suggested that radium emanation, later known as ^{222}Rn, may be the cause of the lung cancers. In 1939 Peller wrote the first review of mining-related cancers, which described the occurrence of lung cancers in Schneeberg and Joachimsthal miners. Finally, in the mid 1950s ^{222}Rn progeny inhalation rather than ^{222}Rn gas was implicated as the causative agent in the excessive lung cancer deaths noted for miners in both the United States and Europe.

6. What was the initial scientific evidence that ^{222}Rn progeny exposure causes lung cancer?

Laboratory animals exposed to high concentrations of ^{222}Rn progeny display lung carcinoma, emphysema, pulmonary fibrosis, and a shortened life span. However, the International Agency for Research on Cancer has classified ^{222}Rn as a human carcinogen primarily on the basis of findings in underground miners exposed to ^{222}Rn progeny. The lung cancer risk attributable to ^{222}Rn progeny exposure has been examined in over 20 different populations of underground miners, including uranium, fluorspar, shale, and metal miners from the United States, Canada, Australia, China, and Europe. The findings from these studies overwhelmingly document that ^{222}Rn progeny exposure causes lung cancers in miners. An analysis of the pooled data from 11 major studies involving 68,000 miners found that lung cancer was linearly related to ^{222}Rn progeny concentrations in underground mines and that overall about 40% of miners' lung cancers were attributable to ^{222}Rn progeny exposure. A subset analysis of the miner data suggests a synergistic (submultiplicative) effect for combined exposure to ^{222}Rn progeny and cigarette

smoke. Other factors possibly influencing the relationship between ^{222}Rn progeny exposure and lung cancer include age at exposure, age at risk, exposure rate, sex, other carcinogens, and nonspecific inflammation of the airways.

7. Does ^{222}Rn progeny exposure induce a specific subtype of lung cancer?

Lung cancer encompasses a clinically and histologically diverse group of carcinomas. The major histologic types of lung cancer include squamous cell carcinoma, small cell carcinoma, adenocarcinoma, and large cell carcinoma. Early findings from the miner epidemiologic studies noted a high frequency of small cell carcinoma in both smokers and nonsmokers. Recent findings from the miner data have indicated that all major subtypes have occurred in excess. The North American and European Pooled residential radon studies found an overall increase in all the histologic types so a specific histologic subtype of lung cancer has not been definitively associated with ^{222}Rn progeny exposure.

8. What units are used to express ^{222}Rn gas concentrations and ^{222}Rn progeny exposure?

The activity (rate of decay) of ^{222}Rn is expressed in units called curies. The Curie is based on the rate of decay of one gram of ^{226}Ra or 3.7 x 10^{10} disintegrations per second. The International System of Units (SI) measure of activity is Becquerels per cubic meter (Bq/m3). One Bq equals 1 disintegration per second. Historically, ^{222}Rn progeny exposure rates have been expressed as working levels (WLs); 1 WL equals any combination of short-lived ^{222}Rn progeny (^{218}Po, ^{214}Pb, ^{214}Bi, and ^{214}Po) in 1 liter of air that releases 1.3 x 105 MeV of potential alpha energy. The value of 1.3 x 10^{5} MeV derives from the energy produced by complete decay of the short-lived ^{222}Rn progeny in radioactive equilibrium with 100 pCi/L of ^{222}Rn. A unit that incorporates both dose and time is the working level month (WLM). Exposure to 1 WL for 1 working month (170 hours) equals 1 WLM cumulative exposure. The SI unit of cumulative exposure is expressed in joule-hours per cubic meter (Jh/m^3). One WLM is equivalent to 3.5 x 10-3 Jh/m^3.

9. List common occupations that have the potential for high ^{222}Rn progeny exposure.

- Mine workers, including uranium, hard rock, and vanadium
- Workers remediating radioactive contaminated sites, including uranium mill
- sites and mill tailings
- Workers at underground nuclear waste repositories
- Radon mitigation contractors and testers
- Employees of natural caves
- Phosphate fertilizer plant workers
- Oil refinery workers
- Utility tunnel workers
- Subway tunnel workers
- Construction excavators
- Power plant workers, including geothermal power and coal

- Employees of radon health mines
- Employees of radon balneotherapy spas (waterborne ^{222}Rn source)
- Water plant operators (waterborne ^{222}Rn source)
- Fish hatchery attendants (waterborne ^{222}Rn source)
- Employees who come in contact with technologically enhanced sources of
- naturally occurring radioactive materials
- Incidental exposure in almost any occupation from local geologic ^{222}Rn sources
- Farming (plowing), grading, etc.
- Employees of radon chambers
- Hospitals (radium implants, etc.)
- Academia and research facilities that use ^{222}Rn or ^{226}Ra.
- Antique stores and collectors (radium items)

10. What is the occupational exposure limit for ^{222}Rn?

The exposure limit varies by regulating agency and type of worker. The Miners Safety and Health Act (MSHA) covers underground miners, whereas the Occupational Safety and Health Act (OSHA) regulates exposure to ^{222}Rn gas and ^{222}Rn progeny for workers other than miners. The MSHA sets limits so that no employee can be exposed to air containing ^{222}Rn progeny in excess of 1.0 WL (100 pCi/L) in active work areas. The MSHA also limits annual exposure to ^{222}Rn progeny to less than 4 WLM per year. OSHA limits exposure to either 30 pCi/L or 0.33 WL based on continuous workplace exposure for 40 hours per week, 52 weeks per year. The Nuclear Regulatory Commission (NRC) and Department of Energy (DOE) generally exclude ^{222}Rn from their occupational exposure regulations. However, when the materials generating the ^{222}Rn are or were under the control of a licensee (e.g., uranium mill, in situ leach facility), dose limits are enforced. When ^{222}Rn progeny are present in equilibrium with ^{222}Rn gas, the derived air concentration (DAC) is 30 pCi/L or 0.33 WL and the annual limit on intake (ALI) is 4 WLM.

11. What is the principal site of ^{222}Rn exposure for most people?

The primary site of ^{222}Rn exposure for most people is the home. The average person in the United States receives over one-half their annual average radiation dose equivalent from ^{222}Rn progeny exposure. The ^{222}Rn progeny exposure imparts a greater effective dose equivalent to the average person than all other natural and man-made sources combined. The high percentage of radiation dose contributed by ^{222}Rn progeny is attributable to both the extended time spent in the home and the frequent occurrence of ^{222}Rn within the home. In some cases, long-term residents of homes with high ^{222}Rn concentrations exceed the cumulative ^{222}Rn progeny exposure noted for some underground miner cohorts.

12. What is the source of ^{222}Rn in homes?

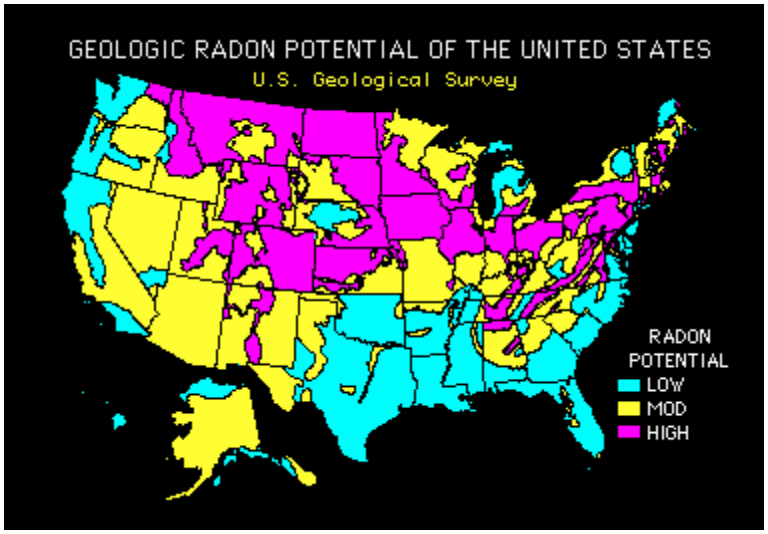
Radon-222 is present in the natural environment because of the radioactive decay of ^{238}U. Four intermediate decay products follow the decay of ^{238}U and precede ^{226}Ra, the direct source of ^{222}Rn. Because ^{222}Rn is a gas, it can readily travel through several meters of permeable soils before decaying. The major sources of ^{222}Rn in indoor air are (1) soil gas emanations from soils and rocks, (2) off-gassing of waterborne ^{222}Rn into indoor air, (3) building materials, and (4)

outdoor air. Of these four sources, soil gas represents the predominant source of indoor ^{222}Rn gas. The primary limiting factor for ^{222}Rn gas migration in the soil is its half-life of 3.8 days. Radon-222 gas enters the home from the soil through cracks in the home, including cracks in the home's foundation, loose-fitting pipe penetrations, sump openings, crawl spaces, and open top of block walls.

In the United States waterborne ^{222}Rn accounts for approximately 5% of the total indoor air ^{222}Rn concentrations for homes utilizing ground-water sources. Waterborne ^{222}Rn may account for a higher percentage of total indoor ^{222}Rn concentrations in some areas of the United States, such as Maine and New Hampshire, where waterborne ^{222}Rn concentrations occasionally exceed 1,000,000 pCi/L. The inhalation exposure from waterborne ^{222}Rn occurs when it off-gasses from the water supply during activities such as showering, washing clothes, and washing dishes. Researchers estimate that 10,000 pCi/L of ^{222}Rn in water contributes about 1 pCi/L of ^{222}Rn to the indoor air of a home. Building materials generally contribute only a small percentage of the indoor air ^{222}Rn concentrations. However, building materials may impart a greater ^{222}Rn contribution when waste products from uranium mining were used to make concrete, concrete blocks, or wallboard. In some areas with a high geologic ^{222}Rn source, outdoor ^{222}Rn gas concentrations exceed several pCi/L for short periods, depending on meteorological conditions.

13. Which areas of the United States have the greatest potential for elevated ^{222}Rn gas concentrations?

The United States Geological Survey Bureau assessed the potential for geologic ^{222}Rn using five main types of data: (1) geologic (litholithic); (2) aerial radiometric; (3) soil characteristics, including soil moisture and permeability; (4) indoor ^{222}Rn gas data from ^{222}Rn surveys; and (5) building architecture. The areas of the United States with high geologic ^{222}Rn potential are shown on the map below. From a limited national survey, the EPA has estimated an average outdoor ^{222}Rn concentration of 0.4 pCi/L for the United States. An average indoor ^{222}Rn concentration of 1.5 pCi/L has been estimated for single family homes from summary data from numerous state residential ^{222}Rn surveys. However, some yearly average outdoor ^{222}Rn concentrations in areas of high geologic radon potential (e.g. areas in Iowa) are equivalent to the national indoor average.



http://energy.cr.usgs.gov/radon/rnus.html

14. What has served as the basis for risk estimates for residential ^{222}Rn progeny exposure?

In 1998 the National Academy of Sciences (NAS) used data compiled from studies of underground miners exposed to ^{222}Rn progeny to project that 18,600 (range: 3,000-32,000) lung cancer deaths each year in the United States are attributable to residential ^{222}Rn progeny exposure. Therefore, numerous public health agencies rank residential ^{222}Rn exposure as the second leading cause of lung cancer after cigarette smoking. The authors of the NAS report cautioned that their approach to assessing risks posed by indoor ^{222}Rn exposure was subject to considerable uncertainty because of gaps in knowledge about the effects of low levels of exposure. Risk estimates derived from miners for the general population must be interpreted cautiously because of inherent differences between the two populations as well as differences between mine and home environments. In 2004 the U.S. EPA issued an updated report in which they attributed 21,000 lung cancer deaths each year to residential radon exposure.

15. What differences between mine and residential environments may limit the generalizability of risk estimates based on miner data?

a. Relatively higher ^{222}Rn gas concentrations in mines than in homes
b. Greater concentrations of airborne dust in mines than in homes
c. Particle diameter higher in mines than in homes
d. Different activity size distributions of radon progeny and rates of attachment for the two environments
e. Presence of other toxic pollutants in mine air, which may act as confounders
f. Age and sex differences between miners and the general population
g. Higher level of physical activity among miners, which affects respiration rates
h. Greater extent of oral vs. nasal breathing in the miners, which leads to increased deposition of larger particles into the lung

i. Different exposure patterns and rates for miners vs. the general population (miners have shorter-term high exposure compared with the lifelong lower concentration exposure for the general population)
j. Most miners were smokers compared with a minority of the general population
k. Even with these physical and biologic differences, the NAS estimated that the dose per unit ^{222}Rn concentration was essentially the same for mine and residential environments.

16. Is there direct evidence the prolonged exposure to residential ^{222}Rn increases lung cancer risk?

Large-scale case-control epidemiologic studies with enhanced dosimetry like the National Institutes of Environmental Health Sciences funded Iowa Radon Lung Cancer have documented a 50% increased risk of lung cancer even at a prolonged exposure at the U.S. EPA action level of 4 pCi/L (150 Bq/m^3). Both the North American and the European Pooled Residential Radon Studies also noted an increased risk of lung cancer for prolonged exposure even at 100 Bq/m^3.

17. How can a homeowner test for ^{222}Rn gas?

Short-term ^{222}Rn test gas kits, which use charcoal detectors, or longer-term test kits, which use an alpha track detector to provide a year-long average ^{222}Rn concentration, are available at most hardware or discount stores. Detectors can also often be purchased at discounted prices through local health departments.

18. What are the recommended residential ^{222}Rn exposure limits? Where can I get more information about ^{222}Rn?

The EPA recommends that ^{222}Rn gas concentrations not exceed a year-long average concentration of 4 pCi/L in any livable area of the home. Instructions for testing and interpreting residential ^{222}Rn concentrations are available from the EPA in "A Citizen's Guide to Radon: a "Guide to Protecting Yourself and Your Family from Radon," and "Radon, The Health Threat with a Simple Solution: A Physician's Guide." A list of state radon contacts who can provide information about testing and reducing the radon concentrations in the home is available from the EPA: http://www.epa.gov/radon/ .

19. Where do elevated waterborne ^{222}Rn concentrations occur?

The highest concentrations of waterborne ^{222}Rn are found in groundwater sources. Surface water sources have much lower concentrations. In addition, water distribution systems with historically high ^{226}Ra concentrations are a source of waterborne ^{222}Rn. Radium-226 adsorbed onto pipe scale within the water distribution system produces ^{222}Rn gas, which significantly increases waterborne ^{222}Rn concentrations by the time the water reaches the point of use.

20. Does ingestion of waterborne ^{222}Rn present a hazard?

The NAS estimates that 160 lung cancer deaths occur each year in the United States as a result of inhaling ^{222}Rn progeny produced by the decay of ^{222}Rn gas emanated from a waterborne source within the home. In comparison, the NAS estimates that 700 lung cancer deaths each year in the United States are attributable to natural outdoor exposure to ^{222}Rn progeny. The NAS also predicted that ingested radon causes about 20 deaths annually in the United States from stomach cancer. The EPA has previously proposed a waterborne ^{222}Rn standard of 300 pCi/L for public water supplies in the United States. The EPA is considering an alternative waterborne ^{222}Rn standard of 4,000 pCi/L for communities that make a concerted effort to reduce indoor ^{222}Rn gas concentrations.

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