Radon: An Overview of Health Effects

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Abbreviations

BEIR VI	US National Research Council's Biological Effects of Ionizing Radiation VI committee
IARC	International Agency for Research on Cancer
MCL	maximum contaminant level
NCRP	National Council on Radiation Protection & Measurements
NRRS	National Residential Radon Survey
OSHA	Occupational Safety and Health Administration
TMI	Three Mile Island
US EPA	United States Environmental Protection Agency
USGS	US Geological Survey
WHO	World Health Organization
WL	working level
WLM	working level month

Introduction

Protracted exposure to radon and its decay products is one of the greatest environmental health threats. In this article, radon is used to refer to the gas itself (radon-222) as well as its decay products. In cases where the text focuses specifically on the gas, it is referred to as radon-222. Radon is the second leading cause of lung cancer in North America and the leading cause of lung cancer for individuals who have never smoked. Overall, radon is also the leading environmental cause of cancer mortality in North America. It was also the first respiratory occupational carcinogen ever to be identified. The occurrence of radon-222 in homes has been known since the 1950s. In fact, surveys of homes built in alum shale-rich areas of Scandinavia identified elevated residential radon-222 concentrations as early as the 1970s.

Radon received heightened attention in the United States in 1984 when a construction engineer named Stanley Watras triggered a portal radiation monitor at the Limerick Nuclear Power Plant in Pennsylvania, the United States. The source of the radioactive contamination was found to be radon-222 decay products from his home that had attached to his clothes. Mr. Watras's home was found to have basement radon concentrations of 162 800 Bq m^{-3} (4400 pCi l^{-1}). The magnitude of the elevated radon-222 concentrations received worldwide attention, and a new environmental threat was discovered. Subsequent radon-222 surveys in various parts of the world have demonstrated that the potential for radon exposure is widespread. The International Agency for Research on Cancer (IARC) has concluded that radon is a Group 1 carcinogen. This article and the following articles on radon provide a general overview of its characteristics, sources, occurrences, and health effects, as well as guidance on both radon measurement and mitigation. Additional information on the health effects related to protracted radon exposure can be found in the 'Further Reading' and 'Relevant Websites' sections.

Radon-222 Characteristics

Chemical and Physical Data

Radon-222 is a noble gas, and like the other noble gases, it is tasteless, colorless, odorless, and generally chemically inert. However, radon has been observed to form clathrate compounds when the gas atoms are physically trapped within the molecular framework of organic compounds such as hydroquinone or phenol. Radon-222 also has the potential to form covalent or ionic bonds with highly reactive elements such as fluorine or oxygen. Like other noble gases, it is soluble in nonpolar solvents, with increasing solubility as the temperature decreases. It is also fairly soluble in water with solubility coefficients of 0.51 and 0.16 at 0 °C and 39 °C, respectively. This article focuses primarily on radon-222, although there are 35 known radon isotopes (all radioactive) with atomic mass numbers ranging from 195 to 229. Radon-222 has the following physical constants:

Atomic weight	222
Atomic number	86
Boiling point	− 62 °C (760 mmHg)
Critical pressure	62 atm
Critical temperature	104 °C
Density	9.73 g l^{-1} (0 °C, 760 mmHg)
Ionization potential	10.748 eV
Melting point	− 71 °C (760 mmHg)
Specific activity	1.5377×10^5 Ci g^{-1}

Radiological Data

Radon-222 exhibits the longest radiological half-life (3.82 days) of the radon isotopes. Radiation from the decay of radon-222 includes a 5489.7 keV alpha emission

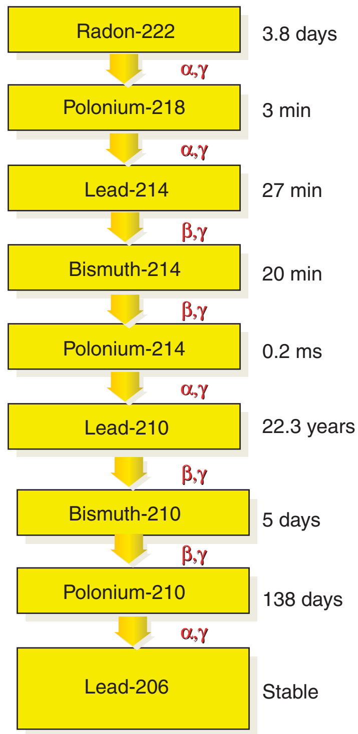

Figure 1 Radon-222 decay chain.

(intensity 99.92%), a 4987 keV alpha emission (intensity 0.08%), a 4827 keV alpha emission (intensity 0.0005%), and a 510 keV gamma emission (intensity 0.07%). Radon-222 decays into a series of solid decay products that achieve equilibrium with the parent within 4 h (Figure 1).

Radon-222 Sources

Radon-222 is part of the uranium-238 decay chain and is frequently generated close to uranium-238. Nonetheless, because several of the radionuclides (uranium-234, thorium-230, and radium-226) between uranium-238 and radon-222 have relatively long half-lives, these nuclides have the potential to migrate through soil formation, weathering, and other natural processes to distant locations away from the parent uranium-238. Once radon-222 is formed, its migration is limited mainly by its 3.8-day half-life. The primary sources of indoor radon are from ground (e.g., soils and rocks) source emanations, off-gassing of waterborne radon into a building, emanation from building materials, and entry of radon into a structure from outdoor air. In most cases, the primary source of indoor radon-222 is from ground sources underlying the structure. Radon-222 soil concentrations in the United States range from 1 to 3 μCi m^{-3} and exhibit mean exhalation rates from soil to air of 0.45 pCi $m^{-2} s^{-1}$. Alternatively, the oceans only contribute approximately 2% of the total input into atmosphere.

The contribution of waterborne radon to indoor air radon concentrations varies substantially both between and within countries. For example, for US homes whose occupants utilize groundwater, waterborne radon-222 accounts for approximately 5% of the total indoor residential radon-222 concentration. However, in some geographic areas of the United States (e.g., Maine and New Hampshire), the relative radon-222 contribution from groundwater sources, as compared to ground sources (e.g., soils and rocks), to indoor air can exceed 50%. The United States Environmental Protection Agency (US EPA) estimates, based on analyses performed by the academic community, that 10 000 pCi l^{-1} (370 000 Bq m^{-3}) of radon-222 in water contributes approximately 1 pCi l^{-1} (37 Bq m^{-3}) of radon-222 to the indoor air. Researchers report that the actual transfer rate may vary from 1000:1 to 100 000:1. Obviously, transfer rates for residential microclimates (e.g., bathroom and dishwashing in kitchen) will exhibit greater variation. The primary health risk related to waterborne radon-222 is breathing in radon-222 decay products that have been produced by the decay of radon-222 gas after off-gassing from water rather than ingesting waterborne radon-222.

Residential and Environmental Exposure to Radon

Although radon occurs naturally outdoors, current construction methods tend to increase, or enhance, the radon concentrations in homes and other built environments as compared to the relative radon concentrations outdoors. The worldwide average residential radon-222 concentration has been estimated to be 39 Bq m^{-3}. At this time, most countries have not performed detailed systematic surveys of residential radon-222 concentrations (see the 'Further Reading' and 'Relevant Websites' sections). To provide an example of the surveys taken by some countries, the US experience follows. In the late 1980s and early 1990s, the US EPA in cooperation with the individual states performed a systematic survey of residential radon-222 'screening' (assumed worst-case short-term radon test performed under 'closed-house' conditions) concentrations in the United States. The stated goals of the survey were to characterize the distribution of indoor radon-222 screening concentrations for each state willing to participate and identify geographic regions of elevated radon-222 concentrations within the states. The US Geological Survey (USGS) in cooperation with the US EPA has published a general geologic-based radon-222 prediction potential map for the United States that considered radon screening measurements as well as rock and soil units that exhibit similar radon generation and transport characteristics in its construction (see Figure 2). A US map displaying the radon zones based on screening measurements and other factors impacting

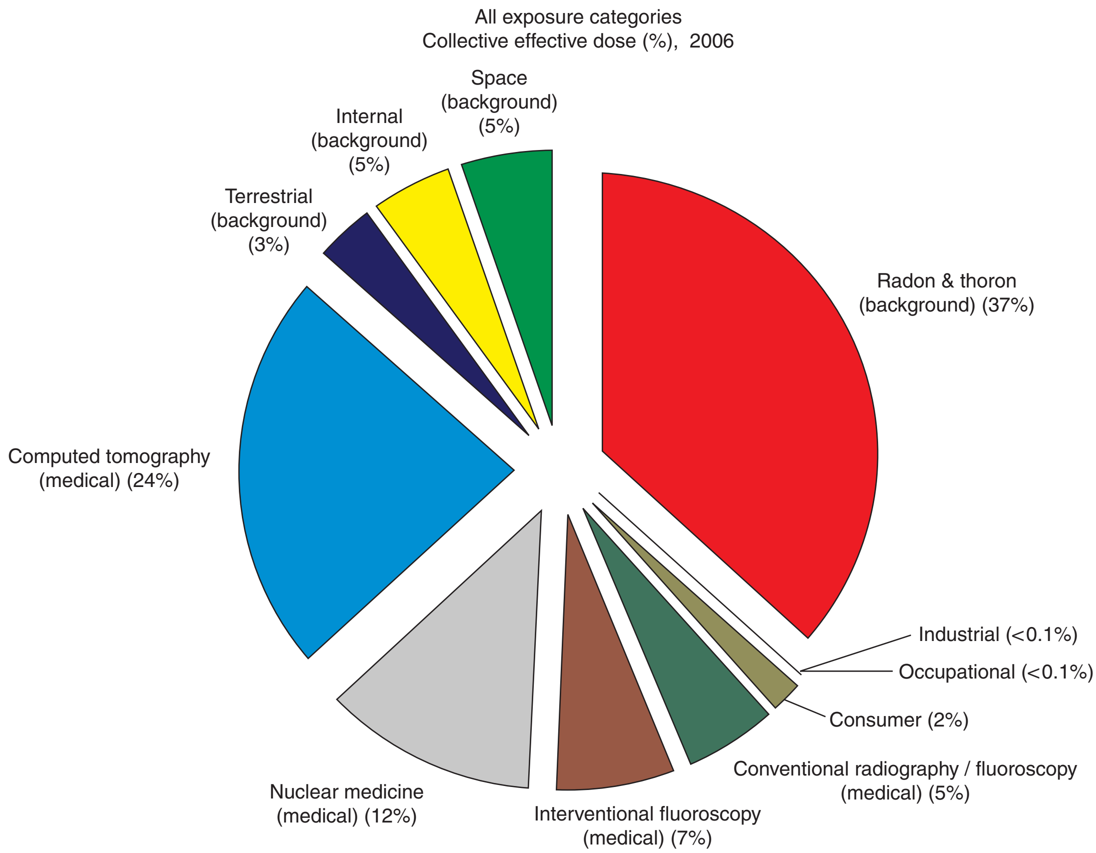

Figure 2 Percent contribution of various sources of exposure to the total collective effective dose and the total effective dose per individual in the 2006 US population. Reproduced from National Council on Radiation Protection and Measurements (NCRP) Report 160. Reprinted with permission of the National Council on Radiation Protection and Measurements, http://NCRPonline.org

potential radon exposure is also available (see US EPA links in the 'Relevant Websites' section). Because of the potential radon-222 variation, even between adjacent homes, prediction maps should not be used as a substitute for determining actual radon concentrations for a particular home in regard to health-based decisions (i.e., performing radon mitigation). The only way to know the radon-222 concentration within a specific home is to test that home for radon.

In the early 1990s, the US EPA performed the National Residential Radon Survey (NRRS) in an attempt to estimate the average radon concentrations of occupied homes over the 50 states. It randomly sampled approximately 7100 eligible homes from 125 counties nationwide using the framework of the NRRS. The yearlong arithmetic mean radon-222 concentration was $1.25\,\mathrm{pCi\,l^{-1}}$ ($46\,\mathrm{Bq\,m^{-3}}$), and the median radon-222 concentration was $0.67\,\mathrm{pCi\,l^{-1}}$ ($25\,\mathrm{Bq\,m^{-3}}$). Subsequent studies by the US EPA found that Iowa has both the highest mean screening radon-222 concentrations and the greatest percentage (71%) of screening radon-222 measurements over the US EPA's radon-222 action level of $4\,\mathrm{pCi\,l^{-1}}$ ($148\,\mathrm{Bq\,m^{-3}}$) in comparison to any other state surveyed in the United States. The US EPA also noted that the counties surrounding the Three Mile Island (TMI) Nuclear Plant in Pennsylvania have the highest regional radon-222 concentrations in the United States. It should be noted that the elevated radon concentrations in the region around the TMI have no relationship to the TMI Nuclear Plant. The US EPA also estimated that the mean yearly outdoor radon-222 concentration is $0.4\,\mathrm{pCi\,l^{-1}}$ ($15\,\mathrm{Bq\,m^{-3}}$). However, the mean yearly outdoor radon concentrations in some areas of the United States have been documented to exceed this concentration. For example, the mean yearly outdoor radon-222 concentration in Iowa is $0.8\,\mathrm{pCi\,l^{-1}}$ ($30\,\mathrm{Bq\,m^{-3}}$), with some areas of the state exhibiting mean yearlong outdoor radon-222 concentrations of $1.4\,\mathrm{pCi\,l^{-1}}$ ($52\,\mathrm{Bq\,m^{-3}}$).

Occupational Exposure to Radon

Systematic surveys of workplace radon-222 concentrations are almost nonexistent. The degree of workplace-related radon exposure for uranium and hard rock miners is better documented, but the potential for exposure to radon in almost any occupational setting is significant (see Table 1). International cooperation is needed to assess workplace exposures and to develop guidance for workplace-related exposures. To date, the World Health Organization's (WHO) initiatives on radon (see the 'Further Reading' and 'Relevant Websites' sections) have limited their guidance to residential radon exposures.

Radon Exposure Guidelines

Residential Radon Exposure Guidelines

In 1987, the National Council on Radiation Protection & Measurements (NCRP) estimated that radon contributed 55% of the overall radiation exposure for the average person in the United States. Although radon exposure has not decreased for the average person, the NCRP estimates that as of 2006, the percentage of the overall radiation exposure attributable to radon has dropped to 37% for the average US population because of the increased use of computed tomography and nuclear medicine (see NCRP information in the 'Relevant Websites' section) (Figure 3).

Table 1 Occupations with potential for radon 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 radon-222 source)
- Water plant operators (waterborne radon-222 source)
- Fish hatchery attendants (waterborne radon-222 source)
- Employees who come in contact with technologically enhanced sources of naturally occurring radioactive materials
- Agricultural exposures (e.g., plowing)
- Incidental exposure in almost any occupation from local geologic radon-222 sources

Many countries have adopted guidelines and reference levels to promote awareness and institute guidance regarding limiting radon exposure. As per WHO guidance, a reference level is the annual mean residential radon concentration above which it is strongly recommended, or in some countries legally required, to reduce the radon concentration. It is important to note that the reference level does not define a safe concentration of radon but rather represents a level of risk that a country deems unacceptable for protracted exposure. Radon mitigation is often recommended for homes above the reference level. In fact, in some countries such as the Czech Republic, Sweden, and Switzerland, it is compulsory to reduce radon concentrations that exceed a certain reference level. As noted in the WHO's International Radon Project guidance (see the 'Further Reading' and 'Relevant Websites' sections), of the 36 WHO member countries surveyed, almost all countries had adopted a reference level between 100 and 400 $Bq\,m^{-3}$ for existing homes. If radon concentrations exceeded these reference levels, radon mitigation was either recommended or required. In addition, some countries have also set radon concentration targets for new building construction at levels below the reference levels for existing homes.

Some countries prefer to use the term 'action level,' rather than 'reference level.' The action level is the point at which remediation work to reduce radon

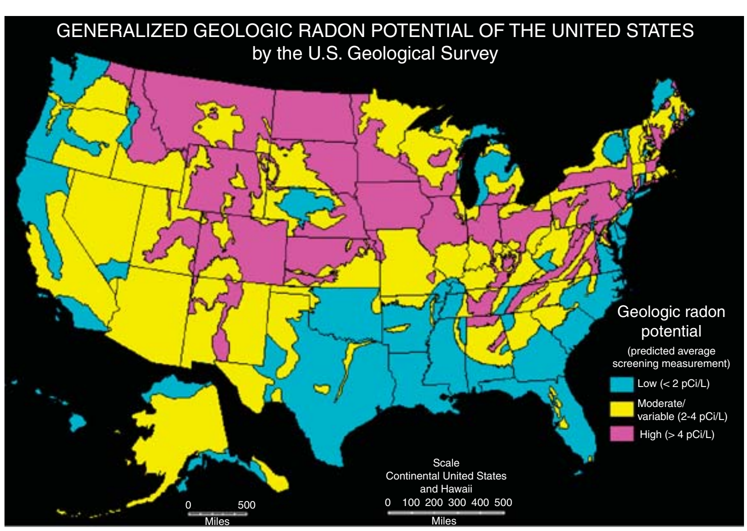

Figure 3 Generalized geologic radon potential of the USA.

concentrations, such as radon mitigation, is recommended. Unfortunately, many householders continue to perceive that the action level is the maximum concentration at which protracted exposure was safe, even though governmental agencies often indicated that there was increased risk to radon exposure even below the action level. For example, the US EPA has established $4\,\mathrm{pCi\,l^{-1}}$ ($148\,\mathrm{Bq\,m^{-3}}$) as the radon-222 action level for the United States. It is important to note that this is not a standard and is not a health-based guideline. The US EPA recommends performing a short-term radon-222 test and a repeated test if the reported radon-222 concentration was $4\,\mathrm{pCi\,l^{-1}}$ ($148\,\mathrm{Bq\,m^{-3}}$) or higher. It recommends taking a long-term test to obtain a better estimate of the long-term radon-222 concentration or another short-term test if the results are needed quickly. It indicates that the higher the initial short-term test, the more certain you can be that a second short-term test should be performed rather than waiting for the results of a long-term test. It further recommends that if the first short-term radon-222 test exceeds $8\,\mathrm{pCi\,l^{-1}}$ ($296\,\mathrm{Bq\,m^{-3}}$), another short-term test should be performed immediately. The US EPA guidance also directs householders to lower the radon-222 concentrations in the home if the yearlong radon test exceeds $4\,\mathrm{pCi\,l^{-1}}$ ($148\,\mathrm{Bq\,m^{-3}}$) or if the average of the first and second short-term radon test exceeds $4\,\mathrm{pCi\,l^{-1}}$ ($148\,\mathrm{Bq\,m^{-3}}$). Although $4\,\mathrm{pCi\,l^{-1}}$ ($148\,\mathrm{Bq\,m^{-3}}$) is the action level in the United States, the Congress has set a long-term goal that indoor radon-222 concentrations be reduced to outdoor concentrations. As pointed out by the US EPA in its Citizen's Guide to Radon, "the EPA believes that any radon exposure carries some risk – no level of radon is safe." Fortunately, with current mitigation technology, the radon-222 in the vast majority of homes can be mitigated to below $2\,\mathrm{pCi\,l^{-1}}$ ($74\,\mathrm{Bq\,m^{-3}}$). Most other countries rely on long-term radon measurements to make decisions regarding the need for radon mitigation. The WHO has provided guidance for member countries in regard to establishing an appropriate radon-222 reference level (see the 'Further Reading' and 'Relevant Websites' sections).

Occupational Radon Exposure Guidelines

The findings from the North American and European pooled residential radon epidemiologic studies have provided stimulus for some countries to review their occupational exposure guidelines for radon. However, the occupational guidelines for most countries regarding radon exposure fail to reflect the documented effects posed by protracted radon exposure. For example, the US Occupational Safety and Health Administration's (OSHA) adult exposure limit, which has not been updated since 1971, is $100\,\mathrm{pCi\,l^{-1}}$ ($3700\,\mathrm{Bq\,m^{-3}}$) averaged over a 40 h work week.

Waterborne Radon Exposure Guidelines

Because the primary risk posed by radon results not from waterborne radon, but rather when it becomes airborne (e.g., via showering), most countries have not performed systematic surveys of waterborne radon or established guidelines for exposure. In the United States, the EPA proposed a maximum contaminant level (MCL) for waterborne radon-222 of $300\,\mathrm{pCi\,l^{-1}}$, for public water supplies based on an excess lifetime cancer risk of 2×10^{-4}, in 1991 that was later withdrawn in 1997. The US EPA subsequently proposed a multimedia approach for reducing radon-related lung cancer in 1999 that focused on reducing higher waterborne radon-222 concentrations, linked with reducing indoor air concentrations. States where multimedia radon-222 mitigation programs were implemented would be permitted to have a $4000\,\mathrm{pCi\,l^{-1}}$ limit for waterborne radon-222. The proposal has not been finalized as of 2009. In addition, private well water sources are not regulated.

Radon Health Effects

Introduction

As radon-222 gas undergoes radioactive decay (**Figure 1**), a large percentage of the decay products, which are solid, attach to ambient aerosols. Although some of the decay products remain unattached, others increase their size through various physical and chemical processes. The attachment rate depends on numerous factors such as the size and concentration of ambient particles. Deposition of radon decay products in the lung also depends on numerous factors, including the particle size, breathing frequency, tidal volume, and lung volume. As mentioned previously, once inhaled and deposited, alpha particles released during the decay of polonium-218 and polonium-214 deliver the majority of radiation dose to the sensitive lung epithelium. Irradiated cells may undergo DNA breaks, accurate repair, inaccurate repair, gene mutations, apoptosis, chromosomal change, and genetic instability. Alpha particles are unique among environmental carcinogens in their ability to produce double-strand DNA breaks. The resulting DNA breakage and rejoining may produce various outcomes, including the insertion, deletion, or rearrangement of genetic material. Exposure to radon can also produce reactive oxygen intermediates that can affect the stability of p53 and produce oxidative damage to the DNA. The cells that have sustained genetic damage, and do not die, may become cancerous. In fact, cancer is thought to be monoclonal in nature (i.e., cancer can originate from a single cell that has completed the process of malignant transformation). Because even a single alpha particle can cause significant genetic damage to the cell, it is unlikely that there is a threshold for radon-induced lung cancer.

Radon-related lung cancer is one of the most intensively studied areas of occupational epidemiology. In the book *De Re Metallica* (Of Metal Matters), published in 1556, Georgius Agricola describes the occurrence of high mortality rates of respiratory diseases in underground metal miners at Schneeberg in the Erz Mountains of Central Europe. The terms describing the widespread diseases of miners in this area, 'Bergsucht' (miners' plague) and 'Bergkrankeit' (miners' disease), received even wider usage because of the publication of a book by Paracelsus (a Swiss physician, chemist, and alchemist who is often credited as one of the fathers of modern medicine), around this same period, entitled *On the Miners' Plague and Other Miners Illnesses*. However, it was not until 1879 that Härting and Hesse linked the high mortality rates, based on over 20 autopsy findings, at Schneeberg to lung cancer. Friedrich E. Dorn, a German scientist, discovered radon-222 in 1900, which he called 'radium emanation.' However, a year before, a British scientist named R. B. Owens and a New Zealand scientist named Ernest Rutherford discovered radon-220. Years later, H. E. Müeller in 1913 and Margaret Uhlig in 1921 are credited with causally relating the lung cancer occurrence with 'radium emanations.' Eighteen years later, in 1939, Sigismund Peller, an internist and medical statistician, published the first review of mining-related cancers in which he described the occurrence of lung cancers among the Schneeberg and Joachimsthal miners. In the mid-1950s, radon progeny inhalation, rather than radon-222 gas, was finally implicated as the causative agent in miner-related lung cancer mortality.

Radon and Lung Cancer: Miner-Based Epidemiology

Overwhelming evidence supports the link between radon exposure in miners and the increased risk of lung cancer. The miner-based study findings also suggest a linear dose response for radon exposure. Findings from studies of radon-exposed underground miners have provided the primary foundation for both the occupational risk estimates as well as the projected risk estimates for residential exposure. The US National Research Council's Biological Effects of Ionizing Radiation (BEIR) VI committee pooled data from 11 miner-based studies, which included approximately 68 000 men (over 2700 of the men had died of lung cancer). The BEIR VI committee also presented results for a population of miners with mean radon exposures of 15 working level months (WLMs), which is similar to a 25-year residential radon exposure at 4 $pCi\,l^{-1}$ (148 $Bq\,m^{-3}$). Every retrospective cohort epidemiologic study of radon-exposed underground miners reported increased risk for lung cancer, including the studies with ranges of cumulative exposures that overlapped with the cumulative exposures occurring in the residential setting (see the 'Further Reading' and 'Relevant Websites' sections).

Projections to Residential Radon Exposure Lung Cancer Risk Based on Miner Data

Cumulative exposure assessments for the miner studies were expressed in the units of WLM. A working level (WL) is any combination of radon decay products in 1 l of air that will result in the ultimate emission of 1.3×10^5 MeV of potential alpha energy. A WL is equal to the alpha energy released by the decay of short-lived radon daughter products in equilibrium with 100 $pCi\,l^{-1}$ (3700 $Bq\,m^{-3}$) of radon-222. A WLM is defined as the cumulative exposure received by breathing air containing radon decay products at a concentration of 1 WL for 170 h. Under assumed standard conditions in a home for 25 years, radon concentrations of 1 $pCi\,l^{-1}$ (37 $Bq\,m^{-3}$) and 4 $pCi\,l^{-1}$ (148 $Bq\,m^{-3}$) would result in 3 and 15 WLMs of exposure, respectively, to radon and its decay products. The mean exposure for the miners examined by the BEIR VI committee was 164 WLMs.

In developing the risk estimates for residential radon exposures (i.e., extrapolation or interpolation of the miner-based data to the residential setting), the BEIR VI committee assumed that the risk of developing radon-induced lung cancer increases linearly as the exposure increases and that even very low exposures carry some risk (e.g., no threshold for risk). The BEIR VI committee's projected central risk estimates, based on two different models, attributed approximately 15 400 or 21 800 total radon-related lung cancer deaths each year in the United States. Subsequently, the US EPA updated the BEIR VI risk estimates for the United States, based on 1995 mortality data, stating that approximately 21 100 (14.4%) of the 146 400 lung cancer deaths in the United States were radon related. If one ranks the estimated 2008 US mortality for radon-induced lung cancer as compared to other common types of cancer from all causes, the risk of lung cancer posed by radon exceeds the total mortality for several other types of cancers (Table 2). In fact, radon is considered the number one health risk in the home, and comparative human health-based risk assessments performed by numerous governmental agencies have also consistently ranked radon among the most important environmental health risks facing their country.

Among individuals who never smoked, the US EPA estimated that 26% of lung cancer deaths were radon related. Because of the log normal distribution of radon, the majority of radon-induced lung cancers for many countries occur below the radon action or reference levels adopted by that country. For example, the BEIR VI committee estimated that two-thirds of the radon-related deaths in the United States occur below the EPA's action level of 4 $pCi\,l^{-1}$ (148 $Bq\,m^{-3}$). In fact, to reduce the

Table 2 All cause estimated 2008 US cancer mortality by selected cancer types as compared to estimated radon-induced lung cancer mortality

Cancer type	*Estimated deaths*
1. Lung and bronchus	161 840
2. Colon and rectum	49 960
3. Breast	40 930
4. Pancreas	34 290
5. Prostate	28 660
6. Leukemia	21 710
Radon-induced lung	21 000
7. Non-Hodgkin lymphoma	19 160
8. Liver and bile duct	18 410
9. Ovary	15 520
10. Esophagus	14 280
11. Urinary bladder	14 100
12. Kidney and renal pelvis	13 010
13. Stomach	10 880
14. Myeloma	10 690
15. Melanoma	8 420

overall number of radon-attributable lung cancer deaths in the United States by 50%, radon concentrations in all homes in the country would have to be reduced to below a yearly average of $2\,\mathrm{pCi\,l^{-1}}$ ($74\,\mathrm{Bq\,m^{-3}}$) (see the 'Further Reading' and 'Relevant Websites' sections).

Radon and Lung Cancer: Residential Radon Epidemiologic Studies

Because of the inherent population differences between miners and the general population, as well as differences between the mine and residential depositional environment, many scientists questioned whether the risk estimates derived from miners could be extrapolated to the general population. These differences included (1) the relatively higher radon concentrations in mines versus the home; (2) greater concentrations of airborne dust in the mines versus the home that affect both the activity size distribution of the radon progeny and the rate of attachment; (3) the presence of other toxicants in the mine air that may act as confounders; (4) age and gender differences between miners and the general population; (5) the higher level of physical activity among the miners that affects respiration rates; (6) differences in exposure rates and duration; (7) differences in the equilibrium ratio between radon and its decay products; and (8) the greater percentage of oral versus nasal breathing in miners that tends to increase the deposition of larger sized particles into the lung. In the initial attempts to examine the association between average radon exposure based on geographic measurements and lung cancer mortality, numerous ecologic studies (i.e., geographical correlation studies) were conducted. These ecologic studies examined the correlation between aggregate, and often surrogate (e.g., building materials and geology), measures of radon exposure and geographic-based lung cancer mortality rates. However, the usefulness of this study design is limited to hypothesis generation since the ecologic design cannot adequately adjust for possible confounding, effect modification, and bias. The uncertainties mentioned earlier increased the need to perform case-control epidemiologic studies worldwide to directly evaluate the risk of lung cancer posed by residential radon exposure.

Since the 1980s, 22 major residential radon (primarily case-control) epidemiologic studies examining the relationship between protracted residential radon exposure and lung cancer have been performed worldwide. Seven of the studies were performed in North America, 13 in Europe, and two in China. The studies had mixed results. To improve the power to examine the relationship between radon exposure and lung cancer, three collaborative pooling efforts were initiated: a European, North American, and Chinese pooled analysis. The pooled studies reported an increased lung cancer risk at $100\,\mathrm{Bq\,m^{-3}}$ ($2.7\,\mathrm{pCi\,l^{-1}}$) of 8% (95% CI: 3–16%), 11% (95% CI: 0–28%), and 13% (95% CI: 1–36%) for the European, North American, and Chinese pooled analyses, respectively. These risk estimates were similar to the increased risk of 12% (2–25%) at $100\,\mathrm{Bq\,m^{-3}}$ extrapolated from miner data.

The specific methodology used by the various studies to calculate retrospective radon exposure is critical to accurately assessing risk. When the pooled analyses incorporated more stringent methods to model radon exposure, higher risk estimates were observed. If the retrospective exposure misclassification does not differ systematically between cases and controls, results tend to be biased toward finding no association. In fact, empiric models based on some of the more rigorous case-control epidemiologic residential radon studies have clearly demonstrated that studies with improved retrospective radon exposure estimates were more likely to detect an association between protracted residential radon exposure and lung cancer. These findings suggest that the pooled residential radon studies likely underestimate the true lung cancer risk posed by residential radon exposure (see the 'Further Reading' and 'Relevant Websites' sections).

Individual Susceptibility to Radon-Induced Lung Cancer

Not surprisingly, because of the submultiplicative association between radon and smoking, individuals who smoke or who have smoked are at an increased risk of developing radon-induced lung cancer. In addition, although data are lacking, it is very likely that individuals who are exposed to other lung carcinogens (e.g., environmental tobacco smoke, nickel, and radiation from medical procedures) or mixtures of toxicants also have increased susceptibility to radon-induced lung cancer. Furthermore, certain genetic predispositions (e.g., individuals who lack GSTM1) may also predispose individuals to increased risk from protracted radon exposure.

Radon Exposure and Other Adverse Health Outcomes

Possible adverse health outcomes related to radon exposure other than lung cancer have been suggested by various epidemiologic studies and include leukemia, stomach cancer, liver cancer, and pancreatic cancer. However, to date, the evidence to support an association between radon and cancer, other than lung cancer, is not convincing. Further work is needed in this area since few studies have been performed to directly assess possible links between radon exposure and cancer incidence or between radon exposure and noncancer outcomes.

See also: Assessing Human Exposure to Environmental Toxicants, Assessment of Human Exposure to Air Pollution, Cellular Stress Responses to DNA Damage: An Intracellular Balance between Life, Senescence, and Death, Children's Environmental Health: General Overview, Cigarette Smoke, DNA Damage Repair, and Human Health, Clinical Consequences of Radiation Exposure, Environmental Carcinogens and Regulation, Environmental Lung Cancer Epidemiology, Exposure Guidelines and Radon Policy, Exposure Reconstruction Using Space-Time Information Technology, Exposure Science: Contaminant Mixtures, Exposure Science: Ingestion, Exposure Science: Inhalation, Exposure Science: Monitoring Environmental Contaminants, Exposure Science: Pharmacokinetic Modeling, Fate and Transport: Geostatistics and Environmental Contaminants, Frequency and Timing of Environmental Exposure, Genome Effects and Mutational Risk of Radiation, Indoor Radon Prevention and Mitigation, Measurement of Air Pollutants, Mineral and Fuel Extraction: Health Consequences, Models of Human Exposure to Environmental Contaminants, Perceptions and Physiological Responses to Indoor Air Quality, PM2.5 Sources and Their Effects on Human Health in China: Case Report, Productivity and Health Effects of High Indoor Air Quality, Radon Measurement, Residential Radon Levels Around the World, Sick Building Syndrome, Ventilation.

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