

2. RELEVANCE TO PUBLIC HEALTH

2.1 BACKGROUND AND ENVIRONMENTAL EXPOSURES TO RADON IN THE UNITED STATES

Radon is a noble gas formed from the natural radioactive decay of uranium (U) and thorium (Th), natural components of the earth's crust, which decay to radium (Ra) and then to radon (Rn). Decay chains include ^{226}Ra and ^{222}Rn for ^{238}U ; ^{223}Ra and ^{219}Rn for ^{235}U ; and ^{224}Ra and ^{220}Rn for ^{232}Th . As radium decays, radon is formed and released into pores in the soil. Fissures and pores in the substrate allow the radon to migrate to the surface, where it can be released to the air. Radon may also be released into surface and groundwater from the surrounding soil. Though radon is chemically inert, it decays by normal radioactive processes to other radon progeny. The alpha emitting progeny of radon (primarily polonium isotopes ^{218}Po and ^{214}Po) are the ones that can damage the lungs and potentially cause cancer.

Radon may be useful in helping to detect seismic activity, for radiation therapy (as a decay product of ^{223}Ra), as a tracer for leak detection, for flow rate measurements, in radiography, and is used in some chemical laboratory research. It can also be used in the exploration of petroleum or uranium, as a tracer in the identification of NAPL (non-aqueous phase liquid) contamination of the subsurface, in atmospheric transport studies, and as a radiation standard for calibrating radon monitoring equipment in support of environmental surveys of homes and other buildings.

The primary source of radon is its precursors in soil where it is formed and released. On a global scale, it is estimated that 2,400 million curies of radon are released from soil annually. Groundwater provides a secondary source of radon, with an estimated 500 million curies released globally per year. Additional sources of radon include surface water, metal mines (uranium, phosphorus, tin, silver, gold, etc.), coal residues and combustion products, natural gas, and building materials. Global radon releases from oceans, phosphate residues, uranium mill tailings, coal residues, natural gas emissions, coal combustion, and human exhalation are estimated at 34, 3, 2, 0.02, 0.01, 0.009, and 0.00001 millions of curies per year, respectively. Geology, soil moisture conditions, and meteorological conditions can affect the amount of radon released from soil.

The primary pathway for human exposure to radon is inhalation, both indoors and outdoors. Ambient outdoor levels are the result of radon emanating from soil or released from coal, oil, or gas power plants, which can vary temporally and spatially. Outdoor radon levels are typically much lower than indoor radon levels. Soil gas intrusion into buildings accounts for the majority of indoor radon. However,

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indoor radon also can originate from water used for domestic purposes, outdoor air, and building materials.

Exposure to high concentrations can occur in any location with geologic radon sources. Relatively high-level occupational exposure can occur through employment at underground mines (uranium, phosphorus, tin, silver, gold, hard rock, and vanadium), sites contaminated with radon precursors (radium, uranium, or thorium), natural caverns, phosphate fertilizer plants, oil refineries, utility and subway tunnels, excavators, power plants, natural gas and oil piping facilities, “health” mines and spas, fish hatcheries, and, historically, hospitals that used radium needles for therapy.

2.2 SUMMARY OF HEALTH EFFECTS

The most compelling evidence of radon-induced health effects in humans derives from numerous studies of underground miners, particularly uranium miners exposed in the middle part of the twentieth century in the United States and several European countries. These cohort mortality studies typically involved long-term estimates of exposure to high levels of radon based on available measurements in the working environment and contained inherent uncertainty due to confounding factors such as smoking status and coexposure to known or suspected carcinogens (diesel exhaust, arsenic, and silica dust). Nevertheless, the results consistently demonstrate increased risk of lung cancer with increasing exposure to radon in the working environment. The mining cohorts have been followed for several decades or more. Continued follow-up and refined assessments of the most widely-studied mining cohorts have resulted in improved exposure estimates (except for silica dust, which was not considered) and more complete categorization of individuals according to cause of death, mining history, and smoking status. Assessments did not account for actual confounding due to exposure to silica dust (which has since been identified as a known human carcinogen), nor did they necessarily include adjustments for potential confounding exposures to arsenic and diesel exhaust, although considerations for arsenic were made in several studies. One in-depth analysis included assessment of results pooled from 11 of the most widely-studied mining cohorts using the most recent and comprehensive follow-up results available at the time for each individual cohort. The results provide evidence for increasing risk of lung cancer mortality with increasing cumulative exposure to radon and its progeny, and the risk is significantly increased when there is coexposure to cigarette smoke, arsenic, or silica dust.

Reported associations between radon and lung cancer in the mining cohorts raised concern regarding the potential health effects of radon in homes, particularly at levels lower than those experienced in

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mining cohorts. Numerous residential case-control studies of lung cancer have been performed in the United States and in many other countries, including Canada, China, Finland, Germany, Sweden, and the United Kingdom. Some of these studies reported positive or weakly positive associations between lung cancer risk and residential indoor radon concentrations, whereas significant associations were not observed in others. One recent residential case-control study reported a borderline statistically significant negative association between lung cancer risk and exposure to radon at levels in the range of 25–150 Bq/m³ (1.4–4.1 pCi/L), which are near or below the 4.0 pCi/L EPA action limit. Numbers of cases and controls in the individual residential case-control studies limited the statistical power to identify a significant association between radon exposure and an adverse health outcome such as lung cancer. In order to increase the statistical power, investigators involved in most of the studies pooled the results in three separate assessments that included: (1) a combined analysis of 2 China case-control studies, (2) a combined analysis of 7 North American case-control studies, and (3) a combined analysis of 13 European case-control studies. In addition, an overall assessment of the China, North American, and European analyses was conducted by the United Kingdom. Independent results of the pooled analyses provide convincing evidence of an association between residential radon and lung cancer risk in cigarette smokers and recent ex-smokers as demonstrated by increased lung cancer risk with increasing cumulative exposure. The risk to nonsmokers was found to be 25-fold lower. Thus, the risk of radon-induced lung cancer decreases more by reducing or stopping smoking than by reducing residential radon concentration, and both can be used in conjunction for further risk reduction. Collectively, these studies show appreciable health hazard from residential radon, particularly for smokers and recent ex-smokers. An overall pooling of the China, North American, and European case-control studies is in progress.

Associations between radon and health effects other than lung cancer have been made by some investigators. Excess mortality from noncancer diseases reported in some of the mining cohorts include all noncancer respiratory diseases, pneumoconiosis, emphysema, interstitial pneumonitis, other (unspecified) chronic obstructive respiratory diseases, and tuberculosis. However, confounding factors such as exposure to crystalline silica dust and other respiratory toxicants, smoking history, and work experience were likely major contributors to mortalities from noncancer respiratory diseases. Alterations in respiratory function in U.S. uranium miners have been reported. Analyses among U.S. uranium miners indicated a loss of pulmonary function associated with increasing cumulative exposure to radon and radon progeny and with the duration of underground mining. Evaluations of these respiratory end points did not include adjustment for effects of other mine pollutants, such as ore crystalline silica and diesel engine exhaust particles, which were not recognized as human carcinogens at the time the studies were conducted.

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Some information is available regarding lung cancer in animals exposed to radon and its progeny at concentrations considered relevant to human health. Significantly increased incidences of lung tumors were reported in rats repeatedly exposed to radon and its progeny at cumulative exposures as low as 20–50 Working Level Months (WLM). These results are consistent with the demonstrated associations between lung cancer risk and exposure to radon and radon progeny in occupationally-exposed miners and residentially-exposed individuals.

2.3 MINIMAL RISK LEVELS (MRLs)

Inhalation MRLs

No acute-, intermediate-, or chronic-duration inhalation MRLs were derived for radon due to a lack of suitable human or animal data regarding health effects following inhalation exposure to radon and its progeny. The strongest evidence for radon exposure-response and radiation dose-response relationships in humans is for lung cancer; however, cancer is not an appropriate end point for MRL derivation. Nonneoplastic lesions have been reported in animals exposed to radon and its progeny for acute, intermediate, and chronic exposure durations; however, these effects were consistently observed only at lethal or near lethal exposure levels, which were several orders of magnitude higher than those associated with lung cancer in chronically-exposed humans.

Oral MRLs

No acute-, intermediate-, or chronic-duration oral MRLs were derived for radon due to a lack of suitable human or animal data regarding health effects following oral exposure to radon and its progeny. Available human data are limited. In an ecological study, radon levels were measured in 2,000 public and private wells in 14 counties in Maine (Hess et al. 1983). The county averages were compared to cancer rate by county to determine any degree of correlation. Significant correlation was reported for all lung cancer and all cancers combined, when both sexes were combined, and for lung tumors in females. Confounding factors (e.g., smoking) were not considered in this analysis. In addition, exposure to radon in these water supplies could have been by the inhalation route as well as the oral route. No significant associations were observed between cases of bladder or kidney cancer, relative to controls, where mean concentrations of radon in the drinking water were 170, 140, and 130 Bq/L in bladder cancer cases, kidney cancer cases, and controls, respectively (Kurttio et al. 2006). The U.K. Health Protection Agency

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(HPA 2009) reviewed available studies that assessed possible associations between radon and cancer end points and concluded that there is insufficient evidence to suggest that radon is associated with increased risk of cancer at sites other than the lung.