Chapter 8.3 Radon

General description

Radon is a radioactive noble gas. Of all radon isotopes only two, radon-222 (radon) and radon-220 (thoron) occur in significant amounts indoors. Radon-222 is one of the decay products of uranium-238 (half-life 4.5×10^9 years) and radon-220, a nuclide thorium-232 decay (half-life 1.4×10^{10} years). The mother nuclides are radium-226 and radium-224, respectively.

Thoron has a half-life of only 55 seconds, which mostly results in low levels indoors *(1)*. Recent measurements in some countries have shown, however, that in certain situations the doses from thoron and its progeny are significant and comparable to those from radon *(2–4)*.

Radon decays into radioactive metal ions by alpha radiation. The most important of the radon decay products in the present context are the alpha emitters polonium-218 (half-life 3.05 min) and polonium-214 (half-life 1.5×10^{-4} seconds). Immediately after the decay, the progeny are unattached, but a large portion is soon attached to particles in the air or to surfaces. The short-lived progeny collectively have a half-life of about half an hour. The first long-lived progeny, lead-210, has a half-life of 22.3 years.

Sources

Uranium is present in the earth's crust and radon occurs in building materials, groundwater and natural gas. Nevertheless, the ground is the major radon source. The lower air pressure indoors gives rise to a pressure-driven flow of radon-rich soil air into the indoor environment through cracks in the bottom slab and cellar walls. Certain rocks and soil, such as some granites and shales, contain more uranium than others. However, ground with moderate contents of uranium and/or radium can also give high indoor radon concentrations *(5,6)*. The inflow depends largely on the building construction and the permeability of the ground materials.

Building materials made from soil (e.g. clay bricks) or rock always contain uranium and radium. The content is usually low, but some materials may have high concentrations of radium-226, for example alum shale concrete and building materials made of volcanic tuff, gypsum waste, etc. The radon concentration can reach several thousand bequerels per litre (Bq/l) in water from drilled wells in regions with granite rock, for example in Finland and Sweden *(7)*. This contributes to indoor radon and to exposure via ingestion.

Occurrence in air

The radon concentration in outdoor air is higher over large continents than over sea. During temperature inversions (a reversal of the normal atmospheric temperature gradient), levels may reach hundreds of Bq/m^3 over regions with enhanced concentrations of uranium and radium in the ground *(8,9)*. The radon concentration outdoors is usually about 10 Bq/m^3 as an annual average *(1)*.

Indoor radon concentrations depend on the house construction and the underlying soil.

Together with climatic factors and human habits, this leads to variations in radon levels by hour, day, season and year *(6)*. Furthermore, the concentration varies between buildings and rooms, and within rooms. When the major radon source is the ground, rooms in basements or in contact with the ground have higher radon concentrations than rooms on higher floors. Within rooms the radon concentrations may vary, for example near inlet or outlet ducts, and near points of high inflow of radon from the ground, building materials or radon-rich water.

The distribution of radon concentrations in dwellings is approximately log-normal, with a tendency for high concentrations to lie above those predicted by this distribution *(10)*. The geometric mean and geometric standard deviation describe the distribution, while the arithmetic mean is often used to estimate the average probability of detrimental health effects. The United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) has estimated the worldwide, population-weighted values of these parameters for dwellings to be 25 Bq/m^3, 2.5 Bq/m^3 and 40 Bq/m^3, respectively *(1)*.

Radon concentrations in dwellings differ between countries because of differences in geology and climate, in construction materials and techniques, and in domestic customs. The arithmetic means for countries vary from 12 to 140 Bq/m^3 *(1)*. Radon concentrations for some European countries are given in Table 1. More than 10% of the dwellings in certain countries such as Finland and Sweden have levels exceeding 200 Bq/m^3. Systematic investigations of above-ground workplaces are still rare, with the exception of public buildings such as schools.

Conversion factors

Large-scale measurements of radon are made by determining the activity of radon gas. The equilibrium factor between the radon progeny and the radon gas depends on several factors, principally the aerosol concentration and air exchange rate. Measurements in several countries have shown equilibrium factors in dwellings of between 0.2 and 0.8 *(1)*. UNSCEAR and the International Commission on Radiological Protection *(25)* have adopted a typical worldwide equilibrium factor of 0.4.

The SI unit for activity concentration is the bequerel (Bq), which is one radioactive decay per second. (The old unit is the curie (Ci), with one pCi/l equivalent to 37 Bq/m^3.) There are several measures for radon progeny. The equilibrium equivalent concentration of radon is the activity concentration of radon, in equilibrium with its short-lived progeny, that would have the same potential alpha energy concentration as the existing non-equilibrium mixture given in Bq/m^3. The SI measure for radon progeny is the concentration of short-lived radon progeny in air in terms of the alpha energy released during complete decay through polonium-214 given in joule-hours per cubic metre (J·h/m^3).

The cumulative exposure is given as Bq·h/m^3, Bq·y/m^3 (the average radon concentration at 100% occupancy), J·h/m^3 or J·y/m^3. The potential alpha energy exposure of workers is often expressed in terms of the working level month (WLM). 1 WL was originally defined as the concentration of potential alpha energy associated with the radon progeny in equilibrium with 100 pCi/l (3700 Bq/m^3). Since the quantity was introduced for specifying occupational exposure, one month was taken to be 170 hours.

Table 1. Radon levels in dwellings of some European countries

Country	Number of houses sampled	Period and duration of exposure	Sample characteristics	Radon concentration (Bq/m^3)					Reference
				Average	Geometric mean	Geometric mean SD[a]	Percentage over 200 Bq/m^3	Percentage over 400 Bq/m^3	
Belgium	300	1984–1990 3 months to 1 year	population-based (selected acquaintances)	48	37	1.9	1.7	0.3	[b]
Czechoslovakia	1200	1982 random grab sampling	–	140	–	–	–	–	*(1)*
Denmark	496	1985–1986 6 months	random	47	29	2.2	2.2	< 0.4	*(12)*
Finland	3074	1990–1991 1 year	random	123	84	2.1	12.3	3.6	*(13)*
France	1548	1982–1991 3 months (using open alpha track detectors)	biased (not stratified)	85	52	2.3	7.1	2.3	*(14)*
Germany	7500	1978–1984 3 months 1991–1993 1 year	random	50	40	–	1.5–2.5	0.5–1	*(15,16)*
Greece	73	1988 6 months	–	52	–	–	–	–	*(1)*
Hungary	122	1985–1987 2.5 years	preliminary survey	55	42 (median)	–	–	–	[c]
Ireland	1259	1985–1989 6 months	random	60	34	2.5	3.8	1.6	*(17)*
Italy	4866	1989–1994	stratified random	75	62	2.0	4.8	1.0	*(18)*

Country	Number of houses sampled	Period and duration of exposure	Sample characteristics	Radon concentration (Bq/m^3)					Reference
				Average	Geometric mean	Geometric mean SD[a]	Percentage over 200 Bq/m^3	Percentage over 400 Bq/m^3	
		1 year							
Luxembourg	2500	1991	–	–	65	–	–	–	*(1)*
Netherlands	1000	1982–1984 1 year	random	29	24 (median)	1.6	–	–	*(1,19)*
Norway	7525	1987–1989 6 months	random	60	32	–	5.0	1.6	*(20)*
Portugal	4200	1989–1990 1–3 months	volunteers in a selected group (high school students)	81	37	–	8.6	2.6	*(21)*
Spain	1555–2000	winter of 1988– 1989 grab sampling	random	86	41–43	2.6–3.7	–	4	*(1,22)*
Sweden	1360	1982–1992 3 months in heating season	random	108	56	–	14	4.8	*(10)*
Switzerland	1540	1982–1990 3 months (mainly in winter)	biased (not stratified)	70	–	–	5.0	–	*(23)*
United Kingdom	2093	1986–1987 1 year	random	20.5	15	2.2	0.5	0.2	*(24)*

[a] SD = Standard deviation.

[b] A. Poffjin, personal communication.

[c] L. Sztanyik & I. Nikl, personal communication.

Source: Bochicchio et al. *(11)*.

Analytical methods

Time-integrated, continuous recording or instantaneous measurements of radon, usually the activity of radon gas, can be made. Owing to the large variability of indoor radon concentrations, both diurnally and seasonally, measurements integrated over at least three months are required when the aim is to determine the annual average. As a rule, such systems are based on solid-state nuclear track detectors *(26).* International comparisons between laboratories in Europe measuring radon in the air, have been organized by the Commission of the European Communities and by the National Radiological Protection Board in the United Kingdom *(27).*

For epidemiological studies, retrospective measurement methods are desirable. Such methods have been developed but not sufficiently evaluated *(28).* A continuous recording method is useful in choosing the best mitigation method. The origin of the radon inflow can be determined by short-term measurements in several rooms and at various locations within rooms.

Routes of exposure

The highest exposure usually results from inhalation of radon and its progeny. High levels of radon in domestic water might also give high exposures through the intake of water taken directly from the tap *(29).* Radon dissolves in water, but is easily removed when the water is heated or used for other purposes. Radium and the long-lived radon progeny contained in food contribute to exposure by ingestion. However, the consumption of foodstuffs with high concentrations is generally low.

Underground miners have historically been exposed to very high concentrations of radon, especially in uranium mines. Nowadays the levels in most mines have been reduced substantially. The general population in several regions is exposed to high concentrations of radon due to the geology, building construction and domestic habits. Indoor concentrations of 10 000 Bq/m^3 and higher have been found in dwellings, for example in Finland *(30)*, Sweden *(31)* and Germany *(32).* Other buildings may also have high concentrations.

Toxicokinetics

Absoption and doses

Following inhalation, some of the radon progeny are deposited and become attached to the bronchial and bronchiolar epithelium. Deposited activity will irradiate the cells with alpha particles. The most critical cells are currently considered to be the basal and secretory cells in the bronchial epithelium *(33).* A small portion of deposited activity is spread by the body fluids to other organs. Unattached progeny are largely filtered by the nose and, to some degree, in the larynx region. Ingested radon and the other radionuclides in the uranium chain may also be taken up by the gastrointestinal tract and distributed to other organs *(29,34).* Most of the ingested radon in water is transferred to the blood.

The effective half-life for irradiation of the bronchial epithelium by radon progeny following inhalation exposure is about 30 minutes. From the lymph and blood, radon and other nuclides in the uranium chain are distributed to other organs in the body. The highest inhalation

exposures are given to the bronchial epithelium and the effective dose to the other organs represents only a small proportion of the total effective dose *(25)*.

The dose to the human lung from inhalation of radon and its progeny is not included in the latest lung model developed by ICRP *(35)*. The results of epidemiological studies of miners, supported by data from epidemiological studies in dwellings, are considered to be more reliable. Conversion conventions are used for comparing the effective dose from exposure to sources of radiation other than radon inhalation. ICRP has calculated these to be 1.1 mSv per mJ·h/m^3 at home and 1.4 mSv per mJ·h/m^3 at work *(25)* for the ICRP reference population. An exposure to 100 Bq/m^3 in dwellings with 80% occupancy would give 1.8 mSv/year.

Biomarkers of exposure

A correlation of the activity of lead-210 in bone with the cumulated radon exposure from inhalation of radon has been suggested *(36,37)*. Lead-210 is incorporated in bone and has a biological half-life of 10–15 years. Its physical half-life is 22.2 years. Lead-210 in the bones of miners can be derived from the decay of radon gas and radon progeny and also from uranium dust.

In vivo gamma spectrometric measurements of lead-210 in skull bones have been made in miners exposed to very high radon levels *(38)*. Owing to the insensitivity of the method and the influence of other sources of lead-210 intake (water, food, etc.) such measurements have not been feasible for persons exposed to radon in dwellings. This also holds true for *in vitro* measurements in bone *(36)*. In addition, there are practical and ethical problems in sampling for *in vitro* measurements.

Exposure to ionizing radiation increases the number of chromosome aberrations in human blood lymphocytes *(39)*. This increase mainly reflects the last year of exposure, owing to repair mechanisms, and limits the usefulness of this parameter as a marker of long-term exposure.

Health effects

Effects on experimental animals and *in vitro* test systems

Studies in experimental animals show that lung cancer may be induced by exposures to radon progeny as low as about 20 WLM *(40),* corresponding to a lifetime (70-year) exposure of about 60 Bq/m^3. Most experiments have been conducted on male rats; overall, the risk estimates are comparable to those obtained from studies in miners. There is evidence of an inverse dose–rate effect at high concentrations (>100 WL), but this effect was not seen at low levels in one experiment *(41)*. Exposure to tobacco smoke following completed exposure to radon progeny seems to result in a potentiating effect *(42)*. Associated exposure to other factors, however, such as tobacco smoke or uranium ore dust, is not required for carcinoma induction by radon progeny. There is some evidence of increased tumour rates at other sites than the respiratory tract in laboratory animals exposed to radon progeny *(41,43)*, but the findings are not consistent.

Molecular and cellular radiobiology have contributed to the understanding of mechanisms involved in carcinogenesis caused by ionizing radiation. Alpha irradiation can induce mutations and other types of DNA damage, which may affect oncogenes and tumour suppressor genes, leading to impaired cellular growth control *(43)*. This may result in

neoplasm formation. Cancer induction in rats exposed to radon progeny by inhalation can be fitted to a model based on inactivation of both alleles of tumour suppressor genes *(44)*. This may point to a mechanism for cancer induction by radon progeny relevant to human exposure.

Effects on humans – lung cancer

Epidemiological studies on underground miners provide strong evidence that inhalation of radon progeny is associated with an increased risk of lung cancer. A linear relative risk model seems to fit the data over a wide range of exposures *(25,45)*. Combined analyses of the miner cohorts have indicated excess relative risks per WLM of about 0.5–1.5%. The excess relative risk per WLM decreased with attained age and time since exposure. Over a broad range of total cumulative dose, a higher lung cancer risk was associated with underground exposures received at low rates, suggesting that either low exposure rates, long duration of exposure or both may be especially hazardous. Data were too sparse to determine whether this "inverse dose rate effect" applied to the ranges of exposures found in most homes. Most of the miners were smokers, but a clear increase in lung cancer risk with exposure was also demonstrated among the nonsmokers. The joint effect of radon exposure and smoking showed no clear pattern in the different miner cohorts, although the data were consistent with an interaction between additive and multiplicative. There is some evidence that lung tumours in miners exposed to radon progeny have a characteristic mutational pattern in the p53 tumour suppressor gene *(46,47)*, but these data need confirmation.

Lung cancer risks related to residential radon exposure have been investigated with different epidemiological designs *(48)*. Ecological studies comparing aggregated data on residential radon concentrations and lung cancer rates in geographical areas are unsuitable for risk assessment, mainly because of imprecise exposure estimates and poor control of confounding factors. The most relevant evidence comes from epidemiological investigations using individual data on exposure and health effects, mainly case–control studies. Early case–control studies based the exposure estimation on residential characteristics and/or geological information, sometimes supplemented with radon measurements. These studies are not useful for quantitative risk assessment, primarily because of crude exposure data and limited size.

A few recent case–control studies based the exposure assessment on radon measurements in the homes of study subjects, covering residential periods of about 10–30 years. Four of the studies come from Scandinavia, three from North America and one from China. Axelson et al. *(49)* conducted radon progeny measurements in 142 lung cancer patients and 264 controls with 30 years or more of residency in areas with uranium-rich alum shale deposits in southern Sweden. In rural areas, the relative risk (RR) of lung cancer was 1.4 (95% confidence interval, CI: 0.6–3.2) comparing those with radon progeny levels above 150 Bq/m^3 and below 50 Bq/m^3. The corresponding RR in urban areas was 0.8 (95% CI, 0.2–3.0). The authors cautioned that the crude classification of smoking may have led to residual confounding.

Pershagen et al. *(50)* carried out a study in Stockholm on 210 female lung cancer patients and 400 controls. An RR of 1.7 (95% CI, 1.0–2.9) was associated with a time-weighted average radon exposure exceeding 150 Bq/m^3 compared to an exposure of up to 75 Bq/m^3. Positive trends were observed correlating the RR of lung cancer with estimated cumulative radon exposure. Adjustment for the percentage of time actually spent in the home reduced the evidence of a dose–response relationship. The size of the study was too small to allow meaningful analyses of interactions between radon exposure and smoking.

A nationwide study of 1360 lung cancer patients, 1424 population controls and 1360 controls matched on vital status (excluding deaths from smoking-related diseases) was also carried out in Sweden *(51)*. Radon measurements were performed over a three-month period during the heating season in 8992 dwellings occupied for more than two years by study subjects. The RR of lung cancer increased with both estimated time-weighted exposure and cumulative exposure. A linear RR model appeared consistent with the data (Fig. 1). The RRs for time-weighted exposures of 140– 400 Bq/m^3 and >400 Bq/m^3 compared to <50 Bq/m^3 were 1.3 (95% CI, 1.1–1.6) and 1.8 (1.1–2.9), respectively. A supra-additive interaction between radon exposure and smoking was indicated (P = 0.02) and the data were consistent with a multiplicative effect of the two factors.

Fig. 1. Relative risk for lung cancer related to estimated time-weighted average residential radon exposure in the nationwide Swedish study [a]

[a] Bars indicate 5% confidence intervals.

Source: Pershagen et al. *(51)*.

A study of men from a rural area in Finland, including 238 lung cancer patients and 434 controls, found no statistically significant relationship between indoor radon exposure and the risk of lung cancer *(52)*. The exposure–response curve initially showed an increase but then deviated downward at the higher exposure levels. The RR in the highest quintile of radon exposure (= 265 Bq/m^3) was 1.23 (95% CI, 0.71– 2.13) compared to the lowest quintile (< 109 Bq/m^3). A stronger effect of radon was suggested in heavy smokers. The authors noted that the results did not conflict with risk estimates based on the miner studies.

A study in women from New Jersey included 433 lung cancer patients and 402 controls *(53)*. The RR was 4.2 (90% CI, 0.99–7.5) comparing those with indoor radon concentrations of at least 4j pCi/l (148 Bq/m3) and below 1 pCi/l (37 Bq/m^3). The strongest relation to radon appeared among smokers of fewer than 15 cigarettes per day, but the interpretation of the data was difficult because of small numbers, particularly in the highest exposure category.

Alavanja et al. *(54)* conducted a study among nonsmoking women in Missouri, which included 538 lung cancer patients and 1183 controls; 30% of the patients and 17% of the controls were former smokers. An RR for lung cancer of 1.20 (95% CI, 0.9–1.7) was observed in the highest quintile of radon exposure (time-weighted average radon level exceeding 2.46 pCi/l or 91 Bq/m^3) in comparison to the lowest quintile (radon levels up to 0.79 pCi/l or 29 Bq/m^3). A positive exposure–response trend was suggested for the adenocarcinoma cell type, with an RR of 1.66 in the highest exposure category (95% CI, 1.0–2.6).

A study in Winnipeg, Canada included 738 lung cancer patients and 738 controls *(55)*. When the exposure assessment covered the period 5–30 years prior to enrolment in the study, RRs of 0.97 (95% CI, 0.81–1.15) and 0.93 (0.71–1.11) per unit of cumulative radon exposure were observed for bedroom (3750 Bq/m^3·years) and basement (5000 Bq/m^3·years) measurements, respectively. Restricting the analysis to subjects with at least 75% coverage with radon measurements, the corresponding RRs were 1.18 (0.95–1.46) and 1.12 (0.90–1.41), respectively. The authors cautioned that the imputation of missing radon values might have introduced a downward bias in the risk estimates.

Blot et al. *(56)* performed a case–control study among women in Shenyang, China, an area with particularly high lung cancer rates in females. The study included 308 lung cancer patients and 356 controls. No association between radon and lung cancer was found. The relative risk was 0.7 (no confidence interval given) in those with residential radon levels of 8 pCi/l (296 Bq/m^3) and higher, compared to subjects with up to 1.9 pCi/l (70 Bq/m^3). It should be noted that this study population had very high indoor particulate exposure, quite different from those in the other study populations described above, and this may have reduced the bronchial dose from radon.

An overall assessment of the epidemiological evidence on residential radon exposure and lung cancer shows that most studies lacked an adequate statistical power to detect effects of a size consistent with linear extrapolation of exposure–response relationships observed in miners. For example, residential mobility tends to reduce the variance of exposure within a population and thereby increase the sample size requirements. The largest and most conclusive residential study showed an association between estimated radon exposure and lung cancer risk that was in general agreement with most other residential studies as well as with risk estimates derived from underground miner studies. Several extensive epidemiological studies on residential radon exposure and lung cancer are currently under way, and will strengthen the basis for risk estimation over the next few years.

Other cancers

A few ecological studies show correlations between domestic radon levels and cancer of specific sites *(48)*, but no clear pattern has emerged. Darby et al. *(57)* performed an analysis of cancer mortality in 11 miner cohorts. For all cancer other than lung cancer the total number of deaths observed was close to the number expected from mortality rates in the areas surrounding the mines (RR, 1.01; 95% CI, 0.95–1.07, based on 1179 deaths) and mortality did not increase with increasing cumulative exposure to radon. Among individual categories of cancer examined, mortality was significantly increased for cancers of the stomach (RR, 1.33; 95% CI, 1.16–1.52) and liver (RR, 1.73; 95% CI, 1.29–2.28). For leukaemia, mortality was increased in the period less than 10 years since starting work (RR, 1.93; 95% CI, 1.19–2.95) but not subsequently. For none of these diseases was mortality significantly related to cumulative exposure to radon decay products. Among the remaining individual categories of cancer other than lung cancer, mortality was related to cumulative exposure only for cancer of the pancreas, and in the period less than 10 years since start of employment for other and unspecified cancers (likely to include lung cancer metastases). Overall, the study provides evidence that high concentrations of radon in air do not cause a material risk of mortality from cancers other than lung cancer.

Evaluation of human health risks

Exposure evaluation

Exposure to radon and radon progeny is the dominating source of exposure to ionizing radiation in most countries. The radon levels vary considerably between dwellings, and depend primarily on inflow of soil gas and the type of building material. Arithmetic mean concentrations in European countries range from about 20 Bq/m^3 to 100 Bq/m^3, with even higher levels in some regions. The geometric mean concentrations are generally about 20–50% lower because of the skewed distribution of radon levels.

Health risk evaluation

A few recent case–control studies provide evidence on lung cancer risks related to residential radon exposure. In general, the exposure assessment was based on radon measurements in homes of the people being studied, covering residential periods of about 10–30 years *(48)*. Some of the studies indicate increased relative risks for lung cancer by estimated time-weighted residential radon level or cumulative exposure, but the picture is not fully coherent. It should be realized, however, that most studies lacked an adequate statistical power. The largest of the studies, with analyses over the biggest range of exposure, showed a clear increase in risk with estimated exposure to radon, which appeared consistent with a linear relative risk model *(51)*. The interaction between radon exposure and smoking with regard to lung cancer exceeded additivity and was close to a multiplicative effect.

To date, risk estimation for residential radon exposure has often been based on extrapolation of findings in underground miners. Several circumstances make such estimates uncertain for the general population, however, including the possible influence of other exposure factors in the mines and differences in age, sex, size distribution of aerosols, the attached fraction of radon progeny, breathing rate and route *(43,58)*. Furthermore, the relevance is not fully understood of the apparent inverse effect of exposure rate observed in miners and the possible difference in relative risk estimates for nonsmokers and smokers *(45)*.

It is of interest to compare risk estimates based on the nationwide Swedish study on residential radon exposure and lung cancer *(51)* with those obtained from miners. Fig. 2 shows the estimated attributable proportion of lung cancer related to residential radon, using risk estimates from the Swedish study and assuming a linear relative risk model. Imprecision in the exposure estimation leads to attenuation of the exposure–response relationship, and it has been indicated that this may have led to an underestimation of the risk by a factor of up to about 2 *(59)*. It is suggested that the true values lie between the unadjusted and adjusted estimates.

Fig. 2 also gives estimates of attributable proportion based on extrapolations from underground miners, after adjusting for dosimetric differences between mines and homes. As an example the radon concentration distribution in western Germany, with an arithmetic mean of 50 Bq/m^3, leads to an attributable proportion of 7% (95% CI, 1–29%) using the model in Lubin et al. *(45)* and 6% (2–17%) using that of the National Academy of Sciences *(43)*. Corresponding values based on the Swedish residential study are 5% and 9%, respectively, without and with adjustment for exposure misclassification.

Fig. 2. Estimated attributable proportion of lung cancer related to residential radon exposure based on the national Swedish study and extrapolations from miners

Table 2 shows population risk estimates under three different assumptions with regard to population exposure, taken to represent long-term residential exposure in European countries with relatively high, medium and low residential radon concentrations. The estimated attributable proportion of lung cancer related to residential radon exposure ranges from 2–5% in low-exposure areas to 9–17% in high-exposure areas.

Table 2 also shows estimated excess lifetime deaths from lung cancer related to residential

radon. Assuming that lung cancer deaths constitute 3% of total deaths, it is estimated that around 600–1500 excess lung cancer deaths occur per million people exposed on average to 25 Bq/m^3 over their lifetime. For an average exposure of 100 Bq/m^3, the corresponding estimate ranges from 2700 to 5100 excess lung cancer deaths per million people exposed.

Table 2. Attributable proportion of lung cancer related to long-term residential radon exposure in regions with high, medium and low indoor concentrations [a]

	Exposure category		
	High	**Medium**	**Low**
Radon concentration			
Arithmetic mean (Bq/m^3)	100	50	25
> 200 Bq/m^3	15%	1.5%	0.75%
> 400 Bq/m^3	5%	0.5%	0.25%
Proportion of all lung cancers attributable to the exposure			
Total	9–17% [b]	5–9%	2–5%
> 200 Bq/m^3	4–6%	0.4–0.6%	0.2–0.3%
> 400 Bq/m^3	2–3%	0.2–0.3%	0.1–0.15%
Excess lifetime lung cancer deaths (per million) [c]			
Total	2700–5100	1500–2700	600–1500
> 200 Bq/m^3	1200–1800	120–180	60–90
> 400 Bq/m^3	600–900	60–90	30–45

[a] A linear relative risk model is assumed and a multiplicative interaction between radon and other risk factors for lung cancer, including smoking.
[b] The range in estimated attributable proportion is based on assessment of the uncertainty due to imprecision in exposure estimates of the observed exposure–response relationship *(6)*.
[c] It is assumed that lung cancer deaths constitute 3% of total deaths.

Source: Pershagen et al. *(51)*.

Guidelines

Radon is a known human carcinogen (classified by IARC as Group 1 *(60)*) with genotoxic action. No safe level of exposure can be determined. Quantitative risk estimates may be obtained from a recent large residential study, which are in general agreement with a linear extrapolation of risks observed in miners. The risk estimates obtained in the studies conducted among miners and the recent study from Sweden would correspond to a unit risk of approximately 3–6 × 10^{-5} per Bq/m^3, assuming a lifetime risk of lung cancer of 3%. This means that a person living in an average European house with 50 Bq/m^3 has a lifetime excess lung cancer risk of 1.5–3 × 10^{-3}. Similarly, a person living in a house with a high radon concentration of 1000 Bq/m^3 has a lifetime excess lung cancer risk of 30–60 × 10^{-3} (3–6%), implying a doubling of background lung cancer risk.

Current levels of radon in dwellings and other buildings are of public health concern. A lifetime lung cancer risk below about 1 × 10^{-4} cannot be expected to be achievable because

the natural concentration of radon in ambient outdoor air is about 10 Bq/m^3. No guideline value for radon concentration is recommended. Nevertheless, the risk can be reduced effectively based on procedures that include optimization and evaluation of available control techniques. In general, simple remedial measures should be considered for buildings with radon progeny concentrations of more than 100 Bq/m^3 equilibrium equivalent radon as an annual average, with a view to reducing such concentrations wherever possible.

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