Cancer Risks from Exposure to Radon in Homes

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Exposure to radon and its decay products in mines is a well recognized risk of lung cancer in miners. A large number of epidemiologic studies from various countries are quite consistent in this respect even it the magnitude of the risk differs according to exposure levels. Indoor radon became a concern in the 1970s and about a dozen studies have been conducted since 1979, mainly of the case–control design. From first being of a simple pilot character, the designs have become increasingly sophisticated, especially with regard to exposure assessment. Crude exposure estimates based on type of house, building material and geological features have been supplemented or replaced by quite extensive measurements. Still, exposure assessment remains a difficult and uncertain issue in these studies, most of which indicate a lung cancer risk from indoor radon. Also a recent large scale study has confirmed a lung cancer risk from indoor radon. More recently there are also some studies, mainly of the correlation type, suggesting other cancers also to be related to indoor radon, especially leukemia, kidney cancer, and malignant melanoma, and some other cancers as well. The data are less consistent and much more uncertain than for indoor radon and lung cancer, however; and there is no clear support from studies of miners in this respect. — Environ Health Perspect 103(Suppl 2):37–43 (1995)

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Introduction

In the 16th century, both Paracelsus and Agricola described a high mortality from pulmonary disorders among miners. In the retrospect, these observations have been taken to suggest a lung cancer risk from exposure to radon in mines. Other lung diseases might have been involved as well, however. In 1879, lung cancer was specifically reported to occur in excess among miners at Schneeberg in southeastern Germany (*1*). A few decades later a similar observation was made at Joachimsthal in Czechoslovakia (*2*). Since radioactive minerals were found in the mines, radioactivity was suggested to be responsible for the excess of lung cancer among those miners (*3*). More generally, however, the etiologic role of radon and its decay products for lung cancer was not well understood and agreed upon until the 1960s, when lung cancer cases appeared in U.S. uranium miners. Over the past 15 years, exposure to radon has become a public health concern, discovered to commonly occur also in dwellings, sometimes at fairly high concentrations.

Radon and Its Decay Products

The decay of uranium through radium is the source of radon (or more precisely, radon-222). Radon itself decays further into a series of radioactive isotopes of polonium, bismuth, and lead. The first four of these isotopes are referred to as short-lived radon progeny (or radon daughters) with half-lives from less than a millisecond up to almost 27 minutes. Like radon-222 itself, the decay products polonium-218 and polonium-214 emit alpha-particles. Another isotope, radon-220, or thoron, originates from the thorium decay chain, but the short-lived decay products are thought to be of less health interest.

The decay products of radon get electrically charged when created, and tend to attach to surfaces and dust particles in the air. Some also remain unattached. When the air is dusty, the unattached fraction tends to decrease. The unattached progeny is usually considered to be responsible for most of the α-irradiation delivered to the bronchial epithelium, at least in work situations with mouth-breathing, when this fraction is effectively deposited in the bronchia. Radon is not deposited so that the contribution of α-irradiation to the bronchial epithelium from the gas itself is relatively marginal. Some absoption of radon as well as the decay products takes place, however. Increasing interest is therefore also directed to the possibility of extrapulmonary cancer risks.

The α-particles travel less than 100 μm into the tissue, but their high energy causes an intense local ionization, damaging the tissue with a subsequent risk for cancer development. β- and γ-radiation are also present from some of the decay products but this much lower energy content compared to α-radiation makes the effect relatively marginal.

Units for Measuring Radon and Decay Products

Traditionally, and since the 1950s, the concentration of radon decay products, that is, radon daughters, or radon progeny, has been measured in working levels (WL) (*4*). One working level is defined as any combination of short-lived radon progeny in 1 l of air that will ultimately release 1.3×10^5 MeV of alpha energy by decay through polonium-214. In more recently introduced units, this amount of radon progeny may be taken as equivalent to 3700 Bq/m^3 EER (equilibrium equivalent radon) (*5*) or 2.08×10^{-5} J/m^3. The accumulated exposure to radiation is expressed in terms of working level months (WLM). In this context, one month refers to 170 hours of exposure. The corresponding SI-unit is the joule-hour per cubic meter, and 1 WLM is equal to 3.6×10^{-3} Jh/m^3 and may also be taken as 72 Bq-years/m^3.

Exposure to Radon and Radon Progeny

Very high levels of radon have occurred in uranium mines but quite often also in metal mines. For example, cumulated occupational exposures of as much as 3720 WLM were in the past obtained in uranium mines (*6*). Radon progeny concentrations in many nonuranium mines have also been relatively high, often about 1 WL or more, e.g., in hematite mines in West Cumberland, Great Britain (*7*). A cumulated annual exposure of 2.5 to 4.3 WLM was obtained by French miners during the late 1950s and 1960s but decreased to 1.6 – 3.2 WLM during the following decade (*8*).

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Both the use of stony building materials and ground conditions influence indoor concentrations of radon and its decay products. The leakage of radon from the ground shows great variations, and very high indoor concentrations can occur in one house but not in another even if located quite nearby. In general, the leakage of radon from the ground is usually more important than its emanation from stony building materials (*9*). Temperature, wind conditions, and air pressure, as well as behavioral factors influence ventilation and therefore the concentrations of radon and its decay products that may build up in a room. Efforts to improve insulation and preserve energy may have impaired the situation (*10–12*). Radon dissolves to some extent in water, which may serve as a carrier. Wet mines therefore tend to be high in radon, and to some extent, radon in tap water may contribute to the indoor concentrations.

The first measurements on indoor radon were made in Swedish dwellings in the 1950s (*13*). The levels found were in the range of 20 to 69 Bq/m^3. These observations seem not to have caused any concern from the health point of view, however. Recent measurements of indoor radon in Swedish homes have revealed higher levels. On an average, 122 Bq/m^3 were found in detached houses and 85 Bq/m^3 in apartments. Great variations occurred, however, that is, from 11 to 3300 Bq/m^3 (*14*). The differences found between the earlier and the more recently measured concentrations may suggest a general increase in the levels over time.

Indoor radon with concentrations in the range of 40 to 100 Bq/m^3 have been reported as an average from many countries, e.g. United States (*15*), Norway (*16*), Finland (*17*), Federal Republic of Germany (*18*), and elsewhere. Considerably higher levels like two or three thousand Bq/m^3, may occur in many houses, and this is about double the level tolerated in mines in most countries (about 1100 Bq/m^3 or 0.3 WL).

Lung Cancer in Miners

Many mining populations with exposure to radon and its decay products have been investigated, both by cohort and case–control studies. Some of the main results of these studies are summarized in Tables 1 and 2. There is a remarkable consistency between the results as always indicating an increased risk of lung cancer among the miners, even if the overall risk ratios range from about 1.5 to 15. It may be noted in this context also, that other malignant disorders than lung cancer have not yet been clearly demonstrated to depend on radon progeny exposure in mines. A few studies of miners have shown a tendency towards an excess of stomach cancer, however (*41*).

Other agents than radon and its decay products are present in the mine atmosphere and might be thought of as also responsible for the lung cancer risk among miners, for example, carcinogenic trace metals in the dust. Arsenic might have been present at low concentrations in some mines and clearly influenced the risk in one study (*33*). Asbestiform fibers have occurred in some Swedish mines at least, but are considered less likely to have played any substantial role for miners' lung cancer (*42*). Silica dust exposure is a probable cause of lung cancer, especially among silicotics (*43*). Such exposure does not seem to explain the lung cancer risk of miners, however, at least not to any greater extent (*44*). Furthermore, where the exposure to radon and its progeny has been very low as in coal (*45*), potash (*46*), and iron mining (*47*), little or no excess of lung cancer has been observed. Hence, taking these various studies together, the etiologic role of radon as a cause of lung cancer appears well supported through both positive and negative epidemiologic observations.

Assessing Exposure to Indoor Radon

Epidemiologic studies of indoor radon and lung cancer are demanding, especially regarding assessment of exposure. This aspect also includes the problem of obtaining proper contrasts in exposure. The main difficulties derive from the fact that some people spend most of their time at home, whereas others are more often out in the open air or have indoor activities elsewhere with subsequent exposure to different radon concentrations. Furthermore, almost everybody has lived in several houses with varying exposure levels. Only the exposure relating to the home environment would be possible to estimate reasonably accurately in retrospect, whereas exposure obtained in other houses can hardly be accounted for. No support in exposure assessment can be obtained from the individual since there is no perception of exposure to radon and its decay products. This could be a benefit from the validity point of view, however, since there remains little room for discussing recall bias in case–control studies of the health effects of indoor radon.

To assess exposure to indoor radon, current measurements in a number of subsequently used homes of an individual are useful and even necessary. Still, such measurements do not provide any particularly good estimate of an individual's accumulated exposure over many decades. A combination of measurements and judgments with regard to pertinent characteristics of a house might nevertheless be assumed to give a usable estimate of radon progeny exposure. Contrasts in exposure tend to level out, however, so that studies on indoor radon are inherently insensitive and likely to underestimate an effect.

Cellulose nitrate film strips have been used for measuring radon decay products (or indirectly radon, which nowadays is thought to be preferable to direct measurements of radon progeny). Such measurements also have been found to agree relatively well with the exposure estimates based on various characteristics of the houses and geological features likely to have determined indoor radon concentrations (*48*).

Studies of Indoor Radon and Lung Cancer

Several epidemiologic studies have been published since 1979 regarding indoor radon and the risk of lung cancer in the general population. With few exceptions, these studies have been of the case–control type. Most seem to suggest an effect of indoor radon with regard to lung cancer. The overall results of the various studies are summarized in table 3. Two studies of cohort character also have been reported (*61,62*), both being fairly inconclusive but showing tendencies consistent with an effect of indoor radon.

Some of the case–control studies provide little evidence of an effect, however. Especially a study from China on women has been taken to show no effect; but for small cell lung cancers, the odds ratio formally amounted to 1.7 (*58*). This study was conducted in an area with an unusually high risk of lung cancer in women. A high-risk background may therefore have masked an effect of radon. It is of interest too, that in some studies, a less clear effect has been obtained for smokers as well as for urban people (*48,57*).

A first report of a large-scale study on indoor radon and lung cancer in Sweden involved 1360 lung cancer cases and 2847 controls (*63*). Similar to earlier studies, it showed a moderate but significant effect of indoor radon on lung cancer with an odds ratio of 1.3 for a time-weighted exposure at 140 to 400 Bq/m^3 and 1.8 at levels above 400 Bq/m^3 of radon gas. Sleeping with an

open window eliminated the risk totally. It was estimated that some 9 to 16% of the annual lung cancer cases in Sweden were attributable to indoor radon.

The histologic types of the lung cancer cases usually have not been considered in the studies referred to. One of the studies considered specifically oat-cell and other anaplastic lung cancers in women, however. This study showed a clear association with indoor radon (*54*). Another study demonstrated an interesting predominance of squamous and small-cell carcinomas among those who had lived in non-wooden houses, which were likely to have had higher radon levels than wooden houses (*53*). Also the new large Swedish study showed the stronger effect for small-cell carcinomas (*63*). These findings agree with the relative excess of small cell undifferentiated lung cancers seen in the studies on uranium miners. Over time, however, the relative frequency of histologic types has become more normal in miners (*64–66*).

Some correlation studies relating to indoor radon also have indicated an association between lung cancer and indirect measures of potential radon emission from the ground. Some of these studies have utilized a contrast in exposure due to radon emanation from phosphate deposits that had been worked (*67*), or volcanic versus sedimentary structures (*68*). Another opportunity to find a contrasting exposure was obtained by comparison of lung cancer rates of the populations within and outside areas with high radon emanation from granite with increased radioactivity (*69*). The estimated average background γ-radiation per county has also been used as a proxy for potential indoor radon, since there tends to be a rather strong correlation between γ-radiation and emanation of radon from the ground (*70*). Some other more-or-less positive studies have been based on measurements of Ra-226 in water (*71*) or levels of radon in water and indoor air (*72*). Some correlation studies have come out negative with regard to lung cancer, for example, studies from Canada (*73*), China (*74*), and France (*75*).

The Combined Effect of Smoking and Radon

The effect of smoking and exposure to radon among miners has been more or less multiplicative in most studies with adequate data available (*35,66,76,77*). In a few studies there has been a merely additive relationship (*25,40,41*) and sometimes even less than an additive effect (*34,78*). The latter direction has also been observed regarding sputum cytology of uranium miners (*79*).

These rather inconsistent observations may be puzzling but could simply depend on the influence of smoking on the dose received by the epithelium. Smoking seems to increase mucous secretion in a dusty mine environment, causing productive cough (*80,81*). When the mucous sheath gets thicker, fewer α-particles are able to penetrate to the basal cells of the epithelium from which the cancer develops (*82*). An increase in thickness of the mucous layer of only about 10 μm would decrease the dose to the epithelium by some 50% (*83,84*). Furthermore, the clearance of deposited particles carrying radon decay products may also be influenced by smoking with consequences for the ultimate radiation dose delivered to the epithelium.

However, a synergism is likely to occur between chemicals in tobacco smoke and the actual dose of radiation to the epithelium. This way explains the more-or-less multiplicative interaction between smoking and radon progeny exposure seen in most of the studies of miners, especially from the more modern and presumably less dusty mines. Similarly, for indoor radon progeny and smoking, a synergistic effect has been indicated, although the combined effect has sometimes been weak (*48,54,56,57*). In the new large-scale Swedish study the combined effect appeared as clearly multiplicative, however (*63*).

Experimental data provide some support for the complex view given here on the interaction of smoking and radon progeny exposure in miners. When smoking and nonsmoking dogs were exposed to uranium ore dust and radon progeny, the smoking dogs were less affected by respiratory cancer than nonsmoking dogs (*85*). The reason was believed to be the relative protection offered by increased mucous secretion. On the other hand, experiments in rodents indicate that radon progeny exposure followed by exposure to cigarette smoke stimulated tumor development, whereas the reverse combination did not (*86*). Smoking may therefore play a complex role, exerting a carcinogenic effect but sometimes also reducing the dose to the epithelium by increasing mucous secretion. Such complex interaction might well explain the range of observed overall effects from multiplicative to less than additive.

The tendency of radon progeny to attach to environmental tobacco smoke and other particles in the air may imply further complexity of the interaction of smoking and radon progeny exposure. Airborne radioactivity tends to increase in the presence of tobacco smoke since there is less plating out of radon progeny on walls, furniture and other surfaces in the room (*87*). The fraction of unattached progeny tends to be reduced, however, while the attached fraction increases proportionally.

The implications for lung cancer risk in this respect is not very clear. Usually the lung cancer risk has been tied mainly to the unattached fraction, which is thought to contribute much more than the attached fraction of the radiation dose to the epithelium. However, the unattached radon progeny tends to be deposited in the nose to as much as about 50%, whereas the attached fraction is little affected by nose breathing (*88,89*). Attached radon progeny may therefore be deposited further down in bronchial regions with a thinner epithelium. Here the α-particles may be able to penetrate to the basal cells, from which cancer develops (*84*). The biologic net effect of the increased radioactivity of smoke-polluted indoor air and the subsequent change of the proportion of unattached to attached radon progeny is therefore not clear, nor are there any epidemiologic observations in this respect.

A Relation of Extra-Pulmonary Cancers to Indoor Radon?

A relatively recent report indicates a correlation between the incidence of myeloid leukemia, melanoma, cancer of the kidney, and certain childhood cancers and average radon exposure in the homes in a number of countries (*90*). Remarkably, however, lung cancer did not show any significant correlation, as would have been expected rather than the other positive correlations. Inhaled radon, finally reaching the bone marrow, was thought to have induced the myeloid leukemia through its further decay. The accumulation of airborne radon progeny on the skin and a filtering of radon progeny through the kidney were suggested to explain the correlations seen for melanoma and kidney cancer, respectively.

These observations have been criticized (*91*) but initiated a case–control study of 13 cancer forms in relation to residence in areas with different radon levels in the Viterbo province, Italy (*92*). Increased odds ratios between 2 and 3 were seen in the higher exposure categories for kidney cancer as well as for melanoma and

Table 1. Main results of some cohort studies of lung cancer in miners exposed to radon and radon progency (table expanded from NIOSH 1987)

Type of mining; country	Exposure or concentration, means	Person-years	Lung cancer deaths Observed	Expected	SMR	Reference and year
Metal, U.S.	0.05–0.40 WL	23,862	47	16.1	2.92	(*19*) 1963 (*20*) 1981
Uranium, U.S.	821 WLM	62,556	185	38.4	4.82	(*6*) 1971
Uranium, Czechoslovakia	289 WLM	56,955	211	42.7	4.96	(*21*) 1983 (*22*) 1978
Tin, U.K.	1.2–3.4 WL	27,631	28	13.27	2.11	(*23*) 1981
Iron, Sweden	0.5 WL	10,230	28	6.79	4.12	(*24*) 1984
Iron, Sweden	81.4 WLM	24,083	50	12.8	3.90	(*25*) 1984
Fluorspar, Canada	Up to 2040 WLM	37,730	104	24.38	4.27	(*26*) 1985
Uranium, Canada	40–90 WLM	202,795	82	56.9	1.44	(*27*) 1985
Uranium, Canada	17 WLM	118,341	65	34.24	1.90	(*28*) 1986
Iron, U.K.	0.02–3.2 WL	17,156	39	25.50	1.53	(*29*) 1988
Pyrite, Italy	0.12–0.36 WL	29,577	47	1.1	12.70	(*30*) 1989
Uranium non-smokers, U.S.	720 WLM	7,861	14	1.1	12.70	(*31*) 1989
Tin, U.K.	10 WLM/year for 30 years	?	15	3.4	447	(*32*) 1990
Tin, China	2.3–0.9 WL by time	175,143	981	_	Up to 1.8	(*33*) 1993

SMR, standardized mortality ratio.

myeloid leukemia. The dose–response trend and the odds ratio were significantly high only for kidney cancer, however. For lung cancer there was only a slightly increased risk in the intermediate exposure category. Confounding from smoking might have been negative, however, and was not controlled for in contrast to age, farming, and degree of urbanization.

Also two case–referent (case–control) studies on acute myeloid leukemia (AML) might be of some interest in this context (*93,94*). Various exposures were assessed by questionnaires, i.e., occupational exposures, leisure time activities, smoking, and medical care, particularly the use of drugs, X-ray treatment, and X-ray examinations. Residency was considered in terms of estimated γ-radiation from concrete and other stony building materials. An exposure–response relationship was obtained with an index for background γ-radiation. Indoor radon exposure was not thought of as important at the time of the study. Since there tends to be a correlation between radon emanation and gamma radiation from building materials, the possibility remains that indoor radon exposure could have played some etiologic role in this context.

In some of the aforementioned correlation studies there was also a relationship between radon exposure and other cancers, for example with regard to pancreatic cancer and male leukemia (*70*), bladder and breast cancer (*71*), reproductive cancer in males as well as for all cancers taken together (*72*). The mortality rate from stomach cancer was found to be increased in an area with uranium deposits in New Mexico (*95*). Uranium miners in west Bohemia also had an excess of stomach cancer along with liver, gallbladder, and extrahepatic bile duct cancers; only the latter was more clearly related to cumulative radon progeny exposure (*96*).

Table 2. Main results of some case-control studies of lung cancer in miners exposed to radon and radon progeny.

Type of mining, country	Exposure or concentration, means	Number of cases to controls	Number of exposed cases	Rate ratio, max	Reference and year
Zinc–lead, Sweden	1 WL	29/174	21	16.4	(*34*) 1978
Iron, Sweden	0.1–2.0 WL	604/(467×2+137)	20	7.3	(*35*) 1982
Iron, Sweden	0.3–1.0 WL	38/403	33	11.5	(*36*) 1983
Uranium, U.S.	30–2,698WLM	32/64	23	Infinite	(*37*) 1984
Uranium, U.S.	472 WLM in cases	65/230 (nested)	all	1.5% per WLM	(*38*) 1989
Tin, China	515 WLM in cases	107/107	7	(20.0)	(*39*) 1989
Tin, china	373 WLM	74/74	5	(13.2) 1.7% per WLM	(*40*) 1990 (subset of (*39*)

Table 3. Main results of case-control studies of lung cancer and exposure to indoor radon and progeny.

Reference and year	Number of cases to controls	Rate ratio	Remarks
(*49*) 1979	37/178	1.8	Significant trend; crude exposure assessment
(*50*) 1982	50/50	2.1	Published abstract only
(*51*) 1984	23/303	Up to 4.3	Special account for geology and Rn emanation
(*52*) 1984	Two sets of 30/30	___	Signigicantly high exposure for smoking cases
(*53*) 1987	604/(467 x 2 + 137)	Up to 2.0	For more than 20 years in nonwooden houses
(*54*) 1987	292/584	2.2	Women with oat cell cancer
(*55*) 1987	27/49	Up to 11.9	Rick of 11.9 for 10 WLM
(*48*) 1988	177/673	Up to 2,0	Clear effect for rual residents only
(*56*) 1989	210/209 + 191 (hosptl)	Up to 1.8 (in middle exp. category	Females; rate ratio 3.1 for small cell cancers
(*57*) 1989	433/402	Up to 1.7 (small cell)	Females in high risk area (China); slight effect for small-cell ca, only
(*58*) 1990	308/356	Up to 7.2	Females; little effect but in higher exposure category
(*59*) 1991	238/434	Up to 1.9	Lower effect at the highest exposure
(*60*) 1992 (= (*56*) but improved exposure)	210/209 + 191 (hosptl)	1.7 as average	Rate ratio up to 21.7 for smokers and high exposure

Conclusions

The results available so far from epidemiologic studies of indoor radon and lung cancer seem to agree fairly well with the data from miners. The conclusion therefore might be that indoor radon means a risk of lung cancer for the general population, but the quantitative aspect of this health hazard. However, considering the lifetime risk of lung cancer in miners, one extra death per 1000 miners has been proposed as possibly acceptable, which would permit only an exposure of about 0.1 WLM per year (*97*). The magnitude of the indoor radon problem might then be considered in relation to the fact that the average background exposure in the United States has been estimated to 0.2 WLM per year (and up to 0.4 WLM per year in the vicinity of radon- emitting ore bodies). Furthermore, there are indications that the exposure levels may be even considerably higher for large population sectors in many countries.

It is far from clear, however, if the increased cancer risks reported also for other sites than the lung can be attributed to radon and progeny or concomittant gamma radiation. Further interest into the effects of background radiation can be anticipated for the next decade or so and may finally bring clarity to this question.

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