

# *In Focus: Radon and lung cancer*

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## **ABSTRACT**

In the European Union lung cancer death is the most common cause (circa 20%) of total cancer deaths. For 2006 it is estimated that 236,000 lung cancer deaths occurred in the EU 25 with the majority of these being due to active cigarette smoking. From the pooling of 13 residential radon epidemiological studies in 9 EU countries it has been estimated that about 9% of lung cancer deaths may be due to radon exposure in the home. In this paper an account is given of the lung cancer risk estimates derived from these and other residential radon epidemiological studies. A summary account is also given of the mechanisms by which radon can cause lung cancer. Based on the epidemiological studies it is estimated that in 2006 in the EU 25 about 21,000 lung cancer deaths were due to radon exposure. The important role of smoking in radon related lung cancer is discussed. Also discussed are sources of indoor radon as well as practical strategies that may be adopted to reduce residential radon exposures and the associated lung cancer risks.

## **INTRODUCTION**

In the EU as in most developed regions of the world lung cancer is the most common cause of death from cancer. It is estimated that 19.7% of all cancer deaths in the EU in 2006 were due to lung cancer (1). The vast majority of these lung cancer deaths are attributable to cigarette smoking but residential radon studies estimate that radon exposure may be responsible for a not insignificant percentage of these deaths. The U.S. Surgeon General has cited radon to be the second cause of lung cancer after active smoking and radon has been classified as a Group 1 carcinogen by IARC (2, 3). It has been tentatively suggested and is being investigated that radon exposure may be associated with other health endpoints such as leukaemia in children or adults but at present the only health effect established for radon is that it does cause lung cancer (4).

In indoor air radon produces a series of short-lived decay products which may attach to aerosol particles present in the air or deposit on room surfaces. It is the inhalation and deposition of the airborne short-lived radon decay products which gives rise to irradiation by alpha particles of sensitive cells in lung tissue such as the basal cells of the bronchial epithelium (5). From considerations of their respective radioactive half

lives as well as their physical and chemical properties lung dosimetry models show that the radiation dose delivered to the lung is dominated by the alpha particles emitted by the short-lived radon decay products Po-218 ( $E_{\alpha} = 6.00$  MeV) and Po-214 ( $E_{\alpha} = 7.68$  MeV). Because these alpha particles have respective ranges of only 48  $\mu\text{m}$  and 71  $\mu\text{m}$  in tissue they deliver a high density of ionization damage to cells in these short distances. It is this lung dose that is considered to be the cause of radon induced lung cancer either on its own or jointly with tobacco smoke carcinogens. This is supported by animal studies. Due their respective size dependent spatial deposition patterns in the human respiratory tract radon decay products unattached to aerosols (the unattached fraction) deliver a greater alpha radiation dose to sensitive lung tissue in the bronchial region compared to those attached to aerosols (the attached fraction).

## **MINER AND RESIDENTIAL STUDIES OF RADON HEALTH EFFECTS**

There have been numerous studies over past decades into the effects of elevated radon exposure on underground miners both those in uranium mines and in other types of mines (5). Due to differences in study design and in particular to large errors in measuring radon and its decay products in these mines the lung cancer risk factor estimates from these studies cover a range of values. All of them, however, showed a clear dose-related increased risk due to radon exposure. Information on smoking status was available only for a fraction of miners of some of these studies. For smoker miners, the relative risk per unit radon exposure were found to be about 2–3 times higher than the relative risk for all the miners (6,7). This means that the combined risk of smoking and radon was found in these studies to be sub-multiplicative but to be more than additive, thus suggesting synergism between radon and tobacco smoke. In absolute terms the estimated risks per unit radon exposure to smokers was found to be greater than for non-smokers in the mining cohorts. Attempts have been made to transfer or apply the miner studies' risk factors to members of the public exposed to radon in their homes or to the general workforce in above ground workplaces, but this has proved to be somewhat problematic. This is primarily because the miner studies only give estimated risks for adult male miners whose breathing rates, lung morphometry, etc, differ from that of the general population. Moreover, miners were exposed to some more risk factors for lung cancer than are the general population in their homes. In addition aerosol characteristics, degree of equilibrium between radon and its decay products and other aspects of underground mines which influence radon progeny behaviour and consequent deposition pattern in the respiratory tract differ considerably from those present in homes. Nevertheless, Lubin et al. and the U.S. National Research Council BEIR VI Committee took data on residential radon exposure in the U.S. together with data on lung cancer mortality from 11 cohorts of underground miners and on this basis evaluated that the best estimate of the contribution from residential radon exposure to lung cancer deaths in the U.S. is about 10% or 15%, depending on the model used to fit miner data, with a 95% confidence interval of 3%-21% (7, 8). As stated above in this approach, there are many sources of uncertainty in extrapolating from the miner occupational studies to the public. An alternative approach to such use of miner studies or of the more theoretical approach of lung dosimetry modeling for estimating the radon lung cancer risk to the public has

been to directly determine the lung cancer risk from residential radon exposure studies.

Since the 1980s a number of case-control residential radon epidemiological studies have taken place in North America, in Europe and in China. A review of these can be found in (9). Some of the individual studies yielded results which were equivocal. A meta-analysis, however, of the summary odds ratios for these studies showed a slightly significant association between the lung cancer risk and residential radon exposure which was consistent with the results from the occupationally exposed miner studies (10). However, heterogeneity among these studies occurred, probably due to different control of confounding factors which cannot be controlled uniformly in a meta-analysis, whereas it can be done with a pooled analysis (11, 12).

More recently the results of a pooling of North American residential radon studies in a combined analysis of 7 North American case-control studies has been published (11). In this pooling study the radon measurements were based on long-term alpha track radon detectors placed in current and former homes of study subjects. Data was gathered on modifying factors, including age, sex, and smoking habits of the subjects. The study involved 3,662 cases of lung cancer and 4,966 controls. Collaborative analysis of individual data was carried out and data on each separate individual in the seven studies were collated centrally and analyzed with uniform methods.

The odds ratios for lung cancer was found to be increased with increasing radon exposure categories, with an odds ratio of 1.37 (95% CI = 0.98–1.92) for concentrations exceeding 200 Bq/m<sup>3</sup> relative to concentrations under 25 Bq/m<sup>3</sup>. Using a continuous linear model to fit data, the overall estimate of the excess odds ratio for lung cancer per 100 Bq/m<sup>3</sup> was 11%, which was slightly significant (95% CI = 0%–28%). No substantial differences was observed in the excess odds ratio by categories of cigarette smoking, number smoked per day, duration of smoking, or time since quitting. The data obtained in this pooling provides direct evidence of an association between residential radon exposure and lung cancer in keeping with extrapolation from the miner studies.

In Europe a similar pooling of residential radon studies has also taken place in recent years and, like their North American counterpart, has clearly demonstrated and estimated the lung cancer risks associated with radon exposure in homes. Moreover, due to the larger total study size and the higher radon exposure levels of the European studies, a higher statistical power and therefore smaller confidence intervals were obtained and further analyses were possible to be carried out. This collaborative analysis involved 13 European epidemiological studies from nine EU Member States (Austria, Czech Republic, Finland, France, Germany, Italy, Spain, Sweden and the United Kingdom) and included individual data on 7,148 lung cancer cases and 14,208 controls without lung cancer (12,13). Each of these European case-control studies of residential radon and lung cancer had over 150 people with lung cancer and 150 controls without lung cancer. These studies incorporated detailed smoking histories of all subjects and sought radon measurements in homes inhabited by these individuals during the past 15 years or more. As in the North American pooling study data on each separate individual in the thirteen European studies was analyzed with uniform methods and was collated centrally. Radon measurements were obtained from residences occupied during the 5-34 year period prior to lung cancer diagnosis or acceptance as a control.

In this collaborative study a proportionate increase in risk was found not to be strongly influenced by any one study. The dose-response relationship appeared linear with no evidence of a threshold, and a significant relation remained even among those whose average measured radon concentrations were below 200 Bq/m<sup>3</sup>. A non-regulatory Reference Level of 200 Bq/m<sup>3</sup> for residential radon has been in common use in some European countries for many years, originally recommended by the European Communities for future dwellings (14). The absolute risk to smokers and recent ex-smokers was not unexpectedly found to be much greater than that to lifelong non-smokers. This study has provided strong direct evidence of a statistically significant association of residential radon exposure and lung cancer, as predicted by extrapolation from the miner studies. The risk of lung cancer after stratification for study, age, sex, region of residence, and smoking increased by 8.4% (95% CI = 3.0%–15.8%) per 100 Bq/m<sup>3</sup> increase in measured radon concentration. No evidence was found that the excess relative risk varied with age, sex or smoking history. When corrections were applied to remove the bias arising from random uncertainties in radon exposure assessment, the dose-response relation was found to remain linear but increased twice in magnitude to 16% (95% CI = 5%–31%) per 100 Bq/m<sup>3</sup> increase of the estimated mean corrected radon concentration. While the estimated excess relative risks were independent of smoking status, in absolute terms the risks to smokers at any level of radon exposure were much greater than those to lifelong never smokers. For example, taking the risk to lifelong non-smokers exposed to a radon concentration of 0 Bq/m<sup>3</sup> to be 1.0 the relative risk for a habitual smoker of 15-24 cigarettes per day relative to this was estimated to be 25.8, 29.9 and 42.3 at radon concentrations of 0, 100 and 400 Bq/m<sup>3</sup> respectively. For lifelong non-smokers the corresponding risks are estimated to be 1.0, 1.2 and 1.6 respectively. While the very high risks for smokers exposed to radon may seem to indicate that the risk from radon exposure is only important for smokers this is not the case. Taking the absolute lifetime risk to 75 years of lung cancer for lifelong non-smokers not exposed to radon to be about 0.41% (or 1 in 250) then on the basis of the Darby et al study for continuous exposure to radon concentrations of 400 Bq/m<sup>3</sup> and 800 Bq/m<sup>3</sup> this risk will be increased by factors of about 1.6 and 2.3, respectively. In the latter case at 800 Bq/m<sup>3</sup> the estimated absolute risk to a lifelong non-smoker will have increased to 0.93% (or close to 1 in 100). Even allowing for the many uncertainties in such an estimate an involuntary risk of this magnitude of contracting a fatal cancer cannot reasonably be considered to be trivial.

In the context of radon and smoking it should be noted that an interaction between passive smoking and exposure to radon has also been estimated, although the combined risk would be much lower than for active smoking and with a larger confidence interval. Therefore, in this paper we will consider synergism between radon and active smoking, only. It should be noted that a pooling analysis of all the Chinese, North American and European studies which is presently underway is expected to be more informative than the previous regional ones.

In 2006 lung cancer was the most common cause of cancer death in Europe with an estimated 334,800 (19.7% of total) deaths (1). Its major cause is smoking but on the basis of the Darby et al study it is estimated that in Europe, exposure to radon in the home may account for about 9% (95% CI = 3%–17%) of deaths from lung cancer and 2% of all deaths from cancer (12,13). This major collaborative study of 13 residential radon epidemiological studies in 9 EU Member States therefore forms a very solid

basis for policy makers both at EU and Member State levels to formulate and develop effective radon risk management strategies.

## **ESTIMATING RADON RELATED LUNG CANCER DEATHS IN THE EU**

The collaborative pooled analyses of epidemiological studies in North America and in Europe have provided strong evidence that residential radon is an important cause of lung cancer. The European collaborative analysis in particular has quantified the radon related risk of lung cancer to smokers and former smokers relative to that of lifelong never smokers. This study gives a firm basis in principle for estimating the burden of radon related lung cancer deaths in the EU. The process of making a realistic estimate of this burden, however, requires the existence and availability of reliable data bases on indoor radon concentrations and also of smoking prevalence in all Member States.

It should be noted in Table 2 that mean indoor radon concentrations throughout the EU are quite variable. Large variability in indoor radon concentrations may also be present within individual countries. There are many contributory factors to such variability. As indoor radon in most houses originates in the soil or rock subjacent to the house the geological and soil characteristics in a region are a strong determinant of indoor radon levels. Building design, air-tightness of houses and also ventilation preferences of the occupants can also be major influences on the indoor radon level. These factors combined with the geographical distribution of the population in a country can also contribute to the variability. A good example is the UK where high indoor radon values are present in the Devon and Cornwall peninsula but the mean population weighted national indoor radon level at  $21.7 \text{ Bq/m}^3$  is one of the lowest in the EU. This is primarily due to the fact that a large fraction of the UK population lives in the London region which is mainly built on clay with low radon emanating and permeability characteristics.

In the case of smoking habits the data bases available also show there is considerable variability in smoking prevalence throughout the EU. As shown in Table 1 the percentage of adults who smoke in the EU ranges from 17.5% in Sweden to 45% in Greece (15). The EU average is 29% but despite wide variations in smoking prevalence among member states, the overall average for the 25 member states is broadly the same as it was before enlargement in 2004. While the average percentage of non-smoking adults in the EU can be taken from Table 2 to be 71% it should be noted that the non-smoking cohort is composed both of lifelong never smokers and former smokers. As the risk of radon related lung cancer is strongly influenced by smoking status and as the lung cancer risk decreases with time since quitting smoking in order to make a realistic estimate of radon related lung cancer incidence in the EU good information on former or ex-smokers is needed in addition to data on present active smokers (16). Where national data on former smokers is available it usually simply given as their percentage in the population with little or no additional information such as the time since they stopped active smoking or indeed the duration and extent of their previous active smoking habits. In spite of these and other limitations in the available radon and smoking data it is possible using the findings of the Darby et al collaborative study to

make an estimation of the lung cancer impact due to radon in the EU. As already stated above in this study it was estimated that in Europe, exposure to radon in the home accounts for about 9% of deaths from lung cancer and perhaps up to 2% of all deaths from cancer. More accurate estimates on the radon lung cancer burden in Europe are presently being made but are not yet completed. As lung cancer deaths in Europe are estimated to have been 334,800 in 2006 this implies that perhaps up to 30,000 of these deaths may have been caused by exposure to radon in the home (1). The corresponding estimated figures in 2006 for the EU 25 are 236,000 and about 21,000 respectively. In considering these putative radon related EU lung cancer deaths the following three important qualifying observations must be made:

- (1) The majority of these estimated radon related lung related cancer deaths occur in active smokers exposed to radon.
- (2) It should also be noted that, due to the near log-normal distribution of indoor radon levels found in all national surveys the majority of these deaths will occur to persons (both smokers and non-smokers) exposed to indoor radon levels well below the indoor radon Reference Level of 200 Bq/m<sup>3</sup> used in most European and EU countries.
- (3) Residential radon studies have shown that the risk of lung cancer due to the combined effects of smoking and radon exposure are much greater than the additive effect of both individual risks. Therefore in estimating the global lung cancer burden in a country or region good data is needed on not only the indoor radon distribution but also on smoking prevalence. As Table 2 shows smoking prevalence is quite variable throughout the EU. While the EU mean is 29% the percentage of active smokers ranges from 17.5% in Sweden to 45% in Greece.

These three observations have important implications for policy makers in the EU formulating policies and strategies aimed at managing the lung cancer risk from indoor radon.

Table 1. SMOKING PREVALENCE IN THE EUROPEAN UNION (EU 25)\*

EU Member State	Total % of Smokers	% of Men	% of Women	EU Member State	Total % of Smokers	% of Men	% of Women
Austria	29	32	26	Belgium	27.5	33	22
Cyprus	23.5	39	8	Czech Rep.	30.5	38	23
Denmark	27	30	24	Estonia	31.5	45	18
Finland	22.5	26	19	France	30.5	36	25
Germany	32.5	37	28	Greece	45	51	39
Hungary	35.5	42	29	Ireland	27	28	26
Italy	24	31	17	Latvia	31	49	13
Lithuania	28	44	12	Luxembourg	37.5	39	26
Malta	25.5	30	21	Netherlands	30	33	27
Poland	31	39	23	Portugal	20–23	31	9 ?
Slovakia	40	48	32	Slovenia	24	28	20
Spain	32	39	25	Sweden	17.5	16	19
UK	25	26	24	<b>EU Average</b>	<b>29</b>	<b>35</b>	<b>22</b>

\*(15)

## EXPOSURE TO RADON

There are a wide range of both passive and active radon measurement techniques available. As radon is a gas its concentration in a building can be quite variable both diurnally and seasonally due to changes in meteorological parameters, ventilation practices etc. Due to this variability it is generally the case that an assessment of radon exposure in a building is best achieved by making a long-term passive measurement of radon. Typically this is done using alpha track-etch detectors (17). In many EU Member States such long-term indoor radon measurements are usually made over a period of at least three months and preferably in the heating season when radon levels are usually at their highest. In these cases, the annual average can be obtained by applying seasonal correction factors. In some other EU Member States one-year measurements are preferred to obtain the annual average radon concentration. A common approach is to place one detector in the main living room of a house and a second one in the principal bedroom.

In most of the older EU Member States extensive and representative surveys of indoor radon have taken place while in many of the recent accession countries representative nationwide indoor radon surveys have yet to take place. Table 1 gives a summary of the indoor radon data in the EU 25 expressed in units of Bq/m<sup>3</sup>. Because of differences in the characteristics of these surveys it is not possible to calculate a population weighted EU average indoor radon concentration but it is probably close to 50 Bq/m<sup>3</sup>. The distribution of indoor radon in most countries approximates well to a log-normal distribution. While they are very rare a small number of homes with indoor radon levels of some tens of thousands of Bq/m<sup>3</sup> have been found in a number of countries.

Table 2. INDOOR RADON IN THE EUROPEAN UNION (EU 25) \*

EU Member State	Arithmetic Mean Bq/m <sup>3</sup>	Geometric Mean Bq/m <sup>3</sup>	EU Member State	Arithmetic Mean Bq/m <sup>3</sup>	Geometric Mean Bq/m <sup>3</sup>
Austria	102	n/a	Belgium	48	38
Cyprus	7	7	Czech Rep.	118	n/a
Denmark	53	29	Estonia	120	92
Finland	120	84	France	62	41
Germany	50	40	Greece	55	52
Hungary	107	82	Ireland	91	37
Italy	70	52	Latvia	n/a	n/a
Lithuania	32	22	Luxembourg	110	70
Malta	n/a	n/a	Netherlands	23	18
Poland	41	32	Portugal	62	45
Slovakia	87	n/a	Slovenia	87	60
Spain	45	42	Sweden	108	56
UK	22.7	9.7	<b>EU Average</b>	<b>n/a</b>	<b>n/a</b>

\* (18,19)

## SOURCES OF INDOOR RADON

Radon-222, commonly referred to as “radon”, is a chemically inert radioactive gas which is a member of the uranium-238 naturally occurring radioactive decay series. Its immediate parent in the decay series is radium-226. It is produced in most rocks and soils from which it may enter the indoor air of houses. There are a number of possible sources of indoor radon. The most important source for most buildings is soil gas infiltration. It is well established that this is driven by the positive pressure gradient that usually exists between the subjacent soil gas and the indoor air spaces of a building (20). In assessing the risk potential of soil for high indoor radon concentrations in future buildings the main determinants are the subjacent soil permeability, its radium-226 activity concentration and the associated concentration of radon in the soil gas. In some EU member states such as the Czech Republic and Sweden soil radon risk classification based on such soil characteristic is in use (21). In most EU member states, while soil and geological characteristics are taken into account, strategies to achieve low radon levels in future buildings in an area are largely based on surveys on indoor radon levels in existing buildings and on the use of radon proof construction technologies.

In general the contribution to indoor radon levels due to radon emanation from building materials is minor compared to the contribution from soil gas. There are exceptions to this, for example, in parts of Italy where high radium content volcanic tuff is used as a building material or in Sweden where alum shale containing elevated levels of radium has been used in the past as aggregate in aerated concrete products (17).

## RADON CONTROL OPTIONS

While exposure to indoor radon gives rise to a lung cancer risk this risk in principle can be controlled or reduced. At the level of an individual house it is technically feasible, in most cases, to ensure that the indoor radon level is kept at or brought down below a reference or action level set by the national radiation regulatory agencies. In principle the use of ventilation as a means to reduce indoor radon levels appears to be an obvious radon control strategy. It should be noted, however, in the majority of buildings with a radon problem the source of the radon is soil gas which enters the building by pressure driven flow. Therefore if ventilation is used care must be taken to ensure that the ventilation regime does not increase the pressure driven flow thus increasing indoor radon levels. A ventilation solution to an indoor radon problem also may carry an energy penalty. The preferred approaches to controlling indoor radon levels are active soil depressurization by means of sub-floor radon sumps coupled to extraction fans and/or the installation of radon impermeable barriers or membranes in the building foundations (22).

As already mentioned above the most common residential radon reference level being used in EU countries is  $200 \text{ Bq/m}^3$ . This reference level is a recommended value and is not a mandatory regulatory level unlike an Action Level such as  $400$  or  $500 \text{ Bq/m}^3$  for radon in workplaces set by some Member States in their implementation of the EU Basic Safety Standards Directive (23). In the case of an existing house found to be above such a reference level remedial action might involve the installation of a sub-floor sump coupled to an extractor fan or some other appropriate remedial technology, such as a radon membrane barrier, to reduce soil gas radon entry to the house living spaces (22). The cost of such remedial action will vary considerably from one house type to another but experience in some EU countries would indicate that remediation costs should be between € 500 and € 2000. In the case of future houses the incorporation of radon control building technologies into the construction is less costly than their retrofitting in existing houses and would represent a very small fraction of the cost of new house construction. The incorporation of such building technologies in all new houses is already part of the existing building codes in some EU member states such as Ireland (24). WHO Air Quality Guidelines for Europe also suggest that building codes should include sections to ensure that radon daughter levels do not exceed  $100 \text{ Bq/m}^3$  EER (Equilibrium Equivalent Radon concentration) which is similar to a radon concentration of about  $200\text{--}250 \text{ Bq/m}^3$  (25).

Apart from these building technology aspects there are a number of different strategies that can be adopted at a national level to control indoor radon with the objective reducing the lung cancer risk associated with long term radon exposure. These strategies may be divided into the following three principal categories:

(A) Identification of houses with high radon levels and the remediation of these houses. This is rather like the concept often used in radiation protection where a critical group of the most exposed persons is considered a protection priority and the main objective is to reduce *individual* high risks.

In most countries a house with an indoor radon level above  $1000 \text{ Bq/m}^3$  would be classified as “high” as the estimated lifetime lung cancer risk, even for a lifelong never smoker, would be considered unacceptable by most standards of health protection. On the basis of European national radon surveys which show that the

distribution approximates closely to a log-normal distribution the percentage of dwellings in most EU states likely to have a radon level above 1000 Bq/m<sup>3</sup> will be very low. For example in Ireland, where the mean indoor level is 91 Bq/m<sup>3</sup> it is estimated that in < 0.1% of houses is the radon level above 1000 Bq/m<sup>3</sup>. Obviously where high houses are found at random in an area householders should be strongly advised to take action and the competent regulatory agencies should carry out more detailed local surveys to find other high houses that may be present in the area. The problems and costs of finding all high houses on a national basis would not appear in most countries to be justified both from a practical perspective and also from a cost-benefit analysis perspective. On the other hand having a strategy to find high radon houses may be justified in a defined region known to have a high radon potential due to its geological and soil characteristics.

(B) As a consequence of the characteristics of log-normal distributions and the fact that national average indoor radon levels in the EU are mostly below 100 Bq/m<sup>3</sup> the best strategy in principle to reduce the *collective* risks, i.e. the radon related number of lung cancers in the population, should be to reduce the average indoor radon level in a country. For the existing housing stock this is not a practical or cost effective option. The reduction of radon levels in new build future houses by the introduction of appropriate radon preventative building regulations is perhaps therefore the only effective strategy that can over time effectively reduce the national risk from radon related lung cancer. In regions known to have a high radon potential particularly stringent radon prevention building regulations might be considered.

(C) Due to the demonstrated synergism between radon and smoking in terms of causing lung cancer a strategy that should be considered is to couple radon reduction strategies with national strategies aimed at reducing the consumption of cigarettes.

In most EU Member States where there are well developed radon control policies a mixture of the above strategy options (A) and (B) are usually in operation together with radon risk communication programmes. However, having a combined strategy of reducing smoking and radon exposure is presently not part of the public health programme in any EU Member State.

## CONCLUSION

It has been demonstrated by residential radon studies that exposure to radon increases the risk of lung cancer. Even though the estimated excess relative risk factor of 16% per 100 Bq/m<sup>3</sup> was found not to vary with age, sex or smoking history the absolute lung cancer risk associated with unit radon exposure is much greater for active smokers than for lifelong never smokers. In the EU it is estimated that radon related lung cancer deaths account for about 9% (95% CI = 3%–17%) of the total and similar estimates can be obtained from North American studies. Radon levels in homes are controllable by various building technology options such as the installation of active radon sumps and radon proof membranes in the foundations of houses. Coupled to the introduction of indoor radon control regulations there is a need at EU level to establish strict protocols and training programmes to ensure the effective use of these techniques. While radon levels in high radon homes should be reduced it is more cost-effective at a national level to adopt building regulation strategies aimed at reducing

the average radon levels in new houses below the current national average level. In the case of radon risk communication programmes, however, information on the exacerbation of the lung cancer risk in smokers by radon exposure should be emphasised.

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