

Save Our Sievert!

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Abstract

The protection quantity *effective dose* was devised by the International Commission on Radiological Protection (ICRP) as a measure of radiation detriment which takes into account the different sensitivities of different organs and tissues to the induction by radiation of stochastic effects. However, in the case of exposure to radon and its decay products, effective dose is used differently. Without being explicitly acknowledged, effective dose is used as a measure of detriment from the combined effects of two separate carcinogenic agents: radiation (from radon progeny) and tobacco smoke. With recent epidemiological studies now able to estimate the risk of lung cancer as a function of both exposure to radon progeny and exposure to tobacco smoke, it has become clear that the major contributor to this hybrid form of effective dose is tobacco smoke. In the absence of smoking, the dose conversion convention – from radon progeny exposure to effective dose – would be several times smaller than the value recommended by ICRP. In order to make clear the true origins of risk, either the hybrid nature of ‘effective dose’ in the context of exposure to radon should be made explicit, or – the preferred solution – the conversion from radon and radon progeny exposure to effective dose should be based on risk to never-smokers. The latter approach preserves the quantity effective dose as a measure of radiation detriment. Contrary to some perceptions, it does no harm to the system of protection recommended by ICRP. And it avoids a gross misunderstanding that can result in poor decision making when implementing the principles of justification and of optimization of protection.

Key Words

Radon; radon progeny; effective dose; sievert; dose conversion convention.

Introduction

It is well established that ionizing radiation is a carcinogenic agent. The protection quantity *effective dose* was devised by the International Commission on Radiological Protection (ICRP) as a measure of *radiation detriment* which primarily reflects the risk of fatal cancer¹. But in the case of lung cancer and exposure to radon and radon progeny in air, ionizing radiation is not the only carcinogen of relevance. Tobacco smoke is another carcinogenic agent that leads to lung cancer and it is a much more powerful one than radon². When people are exposed to both radon and tobacco smoke, both carcinogens contribute to the overall risk of lung cancer:

$$\text{risk of lung cancer} = f(\text{smoking; radon}).$$

In quantifying the risk, both the level of smoking and the exposure to radon need to be specified. Except for never-smokers, the risk is a combined risk from exposure to radon and from exposure to tobacco smoke. For the population as a whole, the average risk includes a significant component – in fact a dominant component – due to smoking.

Current recommendations (ICRP 1993) for estimating effective dose from exposure to radon make use of a dose conversion convention which assumes a linear relationship between dose and risk. Consequently, when using population average values, the smoking component of the combined risk from radon and smoking is carried over into the quantity effective dose. This means that effective dose calculated for exposure to radon becomes a measure of the combined detriment from tobacco

¹ ‘Effective dose is a measure of dose designed to reflect the amount of radiation detriment likely to result from the dose’: IAEA Safety Glossary, International Atomic Energy Agency, Vienna, 2007.

² For simplicity, ‘radon’ is used here to mean radon-222 and/or radon progeny, unless the context implies the former only.

smoke and radiation, rather than a measure of radiation detriment. And since the smoking component is dominant, this hybrid form of effective dose is a highly inaccurate indicator of radiation detriment.

If effective dose is to be retained as a genuine measure of radiation detriment, then the dose calculated for exposure to radon should be based on the risk for never-smokers. This eliminates the component due to tobacco smoke. For the same reason, if the dose conversion convention is replaced by a dose coefficient obtained from dosimetric modelling, the modelling should use input parameters appropriate for never-smokers.

Discussion

Risk evidence from residential radon studies

Epidemiological studies show that radon and tobacco smoke interact synergistically. In particular, the well-known European pooled residential radon study (Darby *et al* 2006) suggests that the excess relative risk from radon is largely independent of smoking status, which implies that the radon and tobacco smoke risks combine multiplicatively. At its simplest, this can be represented as follows:

$$\Delta r_c = r_b \times ERR_{Rn} \times RR_{smk} \times \Delta c_{Rn} \quad \dots \text{Eq.1}$$

where Δr_c is the increment in cumulative risk of fatal lung cancer arising from an increment in indoor radon concentration Δc_{Rn} , and where ERR_{Rn} is the excess relative risk from radon per unit radon concentration, RR_{smk} is the relative risk from smoking, and r_b is the baseline risk of lung cancer in the population.

The relative risk from smoking increases dramatically with level of smoking, as indicated in Table 1.

Table 1 - Distribution of male controls and relative risk by smoking status in the European pooled residential radon study (Darby *et al* 2006)

Smoking status	Number of controls	Proportion of controls (%)	Relative risk
Never-smoker	2888	27.8	1.0
Current smoker	<15/day	1075	10.3
	15-24/day	1144	11.0
	≥ 25/day	473	4.6
Ex-smoker	<10y	1176	11.3
	≥ 10y	3133	30.2
Other ¹	499	4.8	8.3
Total	10388		

¹Occasional smokers and smokers of pipes, cigars, etc.

For male current smokers of more than 15 cigarettes per day – a significant proportion (16%) of the controls – the risk of fatal lung cancer is about 30 times the risk for never-smokers.

Eq.1 and the pattern of smoking shown in Table 1 can be used to estimate the average risk of lung cancer in the population of controls, as shown in Table 2, where values of r_b (0.59%) and ERR_{Rn} (0.00084 per Bq m⁻³ for measured radon) are taken from Darby *et al* (2006) for cumulative risk to age 80. For this cohort of controls, the average relative risk from smoking for men is about 10 times the risk for never-smokers, and for women about 3 times the risk for never-smokers (data from Table 3 of Darby *et al*, 2006). If these values were used for a population with equal numbers of males and females, the average relative risk from smoking would be 6.6 times the risk for never-smokers. This value has been used to calculate the cumulative risk values in the bottom row of Table 2.

Table 2 - Calculated values¹ of incremental cumulative risk, Δr_c (%), to age 80 for men by smoking status and by increment in measured indoor radon concentration, based on the European pooled residential radon study.

Smoking status	Radon concentration (Bq m ⁻³)				
	0	40	100	200	400 ²
Never-smoker	0	0.02	0.05	0.1	0.2
Ex-smoker $\geq 10y$	0	0.10	0.25	0.5	1.0
Smoker $<15/day$	0	0.26	0.65	1.3	2.6
Ex-smoker $<10y$	0	0.41	1.0	2.1	4.1
Smoker 15-24/day	0	0.51	1.3	2.6	5.1
Smoker $\geq 25/day$	0	0.78	2.0	3.9	7.8
'Population average' ³	0	0.13	0.33	0.65	1.3

¹Smoker and ex-smoker values calculated as never-smoker values multiplied by smoking relative risk from Table 1. This gives slightly different results from those calculated separately by smoking status and radon concentration, as in Darby *et al*, 2006.

²About 96% of homes in the European pooled residential radon study had measured radon concentrations less than 400 Bq m⁻³.

³Over both sexes, but using the baseline risk for males (see text for explanation of averaging).

It can be seen that, to the extent that the population of male controls in the European pooled study represents other populations, the 'population average' incremental cumulative risk can be attributed about 15% to radon and about 85% to smoking (1 : 5.6).

To the extent that cumulative risk to an attained age of 80 can be considered comparable with lifetime excess absolute risk³, it can also be seen that the lifetime excess absolute risk (LEAR) from radon recommended by ICRP is mostly tobacco smoke risk, since it is based on population-average data, including smoking. On the basis of the indoor radon studies, the currently recommended value of $5 \times 10^{-4}/WLM$ would be in the region of $1 \times 10^{-4}/WLM$ in the absence of smoking.

It is disappointing that neither the ICRP Statement on Radon nor ICRP Publication 115 (ICRP 2011) make it clear that the recommended nominal risk coefficient for radon is actually mostly attributable to tobacco smoke. This omission makes the ICRP recommendation misleading. To the unaware reader it appears that the entirety of the risk is attributable to radon.

It is self-evident that both tobacco smoke and radon must be present to create a combined risk. But the fact that radon is present does not mean that all of the combined quantitative risk should be attributed to radon, any more than it should be all attributed to smoking. The two carcinogenic agents each contribute, and the proportion in which they contribute determines the attribution of risk. The tobacco smoke contribution dominates the combined risk.

³Given conventional assumptions about hours of exposure (7000h/year at home) and radon progeny disequilibrium ($F=0.4$), exposure to 1 Bq m⁻³ radon-222 for a year corresponds to 4.4×10^{-3} WLM (ICRP 1993). Given also the convention of summing over 30 years ending 5 years before the attained age, exposure to 100 Bq m⁻³ indoor radon to attained age corresponds to 13 WLM. The population average incremental risk from Table 2 is 0.33% per 100 Bq m⁻³. This then corresponds to a risk of $0.0033/13 = 2.5 \times 10^{-4}/WLM$. This is in fair agreement with both the previously recommended nominal risk coefficient of $2.8 \times 10^{-4}/WLM$ (ICRP 1993) and the recently recommended value of $5 \times 10^{-4}/WLM$ (ICRP 2011).

Risk evidence from miner studies

Epidemiological studies of miners also provide data on the risks of lung cancer from inhaled radon progeny and historically have been used to calculate the LEAR. These generally are less precise in analysing the effect of smoking, but they nevertheless suggest that for men the risk for ever-smokers is about 10 times the risk for never-smokers (Chambers and Stager 2011). The relative risk for ever-smokers (all except life-long non-smokers) in the pooled European study male controls cohort is about 14 – not very different from the miner results. This suggests that if it were possible to further sub-divide the miner ever-smoker cohort, a similar pattern of relative risk as a function of smoking would be seen to that observed in the residential radon study.

The miner whole-cohort relative risk (ever-smokers plus never-smokers relative to never-smokers) is about 7.5 times for men and 3.5 times for women (Chambers and Stager 2011), suggesting a ‘population average’ of 5.5 for equal numbers of men and women – very similar to the value of 6.6 obtained above for the controls cohort in the European pooled residential radon study. This ought not to be too surprising as both values ultimately derive from the smoking pattern of the underlying reference populations used to calculate long-term or lifetime absolute risk from the relative risks derived from epidemiology.

Implications for a dose conversion convention

If the recommended LEAR for radon were used to derive a dose conversion convention, as has been done to date, the resulting effective dose per unit exposure would mostly reflect the risk from tobacco smoke averaged over the pattern of smoking in the population. That is, in retrospect, it can be seen that the currently recommended value of 5 mSv/WLM (ICRP 1993) mostly reflects tobacco smoke risk and that it thereby turns tobacco smoke risk into effective dose. It wrongly assigns tobacco smoke detriment as radiation detriment. From both the residential radon studies and the miner studies it can be seen that, in the absence of smoking, the current dose conversion convention would be 5 or 6 times smaller; that is, in the region of 1 mSv/WLM. This would more correctly have reflected the radiation detriment component of the combined detriment.

New ICRP recommendations

The ICRP has published new recommendations for a nominal probability coefficient (LEAR) for radon and radon progeny-induced lung cancer (ICRP 2011). The newly recommended value is 5×10^{-4} per WLM, almost twice the value of the previous recommendation of 2.8×10^{-4} per WLM (ICRP 1993). The increase arises from the inclusion of more recent data in the analysis and from a focus on lower cumulative doses in the exposed cohorts. However, the recommendation continues the mis-attribution of risk from tobacco smoke to radon. If the risk is to be risk from radon, rather than risk from tobacco smoke and radon combined, the nominal probability coefficient would lie in the region of 1×10^{-4} per WLM.

Similarly, if a dose conversion convention were to be derived using the new recommendations, it would increase by a factor of about 2.4 over the current value. Aligning risks (for adults):

$$\frac{2.8 \times 10^{-4} \text{ per WLM (ICRP 1993)}}{5.6 \times 10^{-5} \text{ per mSv (ICRP 1991)}} = 5 \text{ mSv per WLM} \quad \dots \text{Eq.2}$$

becomes:

$$\frac{5 \times 10^{-4} \text{ per WLM (ICRP 2011)}}{4.2 \times 10^{-5} \text{ per mSv (ICRP 2007)}} = 12 \text{ mSv per WLM} \quad \dots \text{Eq.3}$$

However, this includes the overestimation by 5 or 6 times through including tobacco smoke risk.

If effective dose is to be a measure of radiation detriment, rather than of tobacco smoke detriment and radiation detriment combined, then the dose conversion convention derived from recent data would be in the region of 2 mSv per WLM.

ICRP has stated (ICRP 2011) that it intends in future to replace the dose conversion convention with a dose coefficient derived from dosimetric modelling of alpha particle deposition in the respiratory tract from inhaled radon progeny. Current modelling estimates suggest values of 12 mSv/WLM in mining environments and 14 mSv/WLM in homes (Harrison and Marsh 2011). The coincidence of these values with the updated (hypothetical) dose conversion convention above (Eq.3) has encouraged ICRP to believe that it is appropriate to switch from epidemiology to dosimetry in making recommendations for a dose coefficient.

There is a very large discrepancy between the modelled values and the smoke-free value of around 2 mSv per WLM from epidemiology. ICRP appears to believe that the modelled values take smoking into account largely through the tissue weighting factor for lung, and that the apparent agreement between modelling and epidemiology (Eq.3) is genuine. If true, this would confirm that ICRP uses a hybrid effective dose or 'smoking effective dose' in the case of exposure to radon and radon progeny. The underlying dosimetric model adopted by ICRP is then one in which, at a fixed level of exposure to radon, 'effective dose' increases and decreases with level of smoking. That is, for a fixed radiation detriment, the effective dose varies with smoking status. Adopting a nominal value for the dose coefficient simply locks in a particular level of smoking; it does not alter the inappropriateness of the underlying model if effective dose is to be a measure of radiation detriment.

An additional complication arises if the effect of smoking is assumed to be taken into account through the tissue weighting factor for lung (0.12). In the absence of other biokinetic and physiological differences between smokers and non-smokers, it would imply that the tissue weighting factor for never-smokers would be 5 or 6 times smaller (about 0.02) and for continuing smokers of 15 or more cigarettes a day, about 5 times greater (about 0.6). In fact, there are of course morphological and physiological differences between smokers and non-smokers. Baias *et al* 2010 have investigated the effect of these on dosimetric modelling, and find that they account for no more than factor of about 2 in estimates of dose. Thus very large variations in the tissue weighting factor are still needed to explain the different risks for smokers and non-smokers; variations of a magnitude that appear inconsistent with the current derivation of tissue weighting factors (ICRP 2007).

Effective dose and the system of radiation protection

It has been suggested that using the hybrid form of effective dose – smoking effective dose – is necessary for implementing the ICRP system of radiation protection in a manner that provides protection for everyone in a population, smokers and non-smokers alike. But this confuses two distinct objectives. One is to establish safe living and working conditions for people who may be exposed to radon. The other is to accurately assess effective doses. A safe environment may be established on the basis of risk of harm. The combined risk from tobacco smoke and radon may be used to provide protection for the population as a whole, through optimization of protection and the application of appropriate reference levels and derived constraints. There is no need to convert the combined risk into dose.

For example, a commonly suggested maximum reference level for existing exposure situations is 10 mSv per year. For radon in homes, using the nominal risk coefficient⁴ of 5.7×10^{-5} per mSv, this corresponds to a risk of 5.7×10^{-4} per year. Using a population average LEAR value of 5×10^{-4} per WLM implies an exposure reference level of $5.7/5 = 1.1$ WLM in a year. An average radon progeny concentration of 0.027 WL is then implied for an exposure period of 7000 hours per year. This corresponds to 260 Bq m⁻³ of radon-222 and, when rounded, suggests a maximum reference level of 300 Bq m⁻³ for dwellings.

⁴For a population of all ages (ICRP 2011).

On the other hand, for those circumstances in which it is necessary to assign radiation doses to individuals, the assessment of effective dose should be based on radiation detriment, and should not be multiplied by a factor of 5 or 6 to include (a population average) tobacco smoke detriment. An individual's dose record should show true radiation doses. If necessary, the overall nominal risk⁵ for an individual could then be assessed on the basis of his or her smoking status.

Assigning risk to the wrong causative agent leads to poor decision making. In optimizing protection, all relevant risks, costs and benefits should be taken into account to determine the optimum approach to protection. The objective is to provide the best protection from harm that can be achieved in the prevailing circumstances. This objective is compromised if one of the contributing factors is in error. Similarly, in making decisions about the justification of a proposed planned exposure situation (radiation practice), risks and benefits need to be accurately assessed. Over-estimating radiation risk by including – without explanation – the risk from another carcinogen in radiation dose would have a significant effect on perceptions of acceptability of proposed activities.

The use of smoking effective dose also creates difficulties for the future. When reviewing recorded doses, it will not be clear how much of the combined detriment is due to radiation and how much to tobacco smoke. Further, a dose conversion based on smoking prevalence is unstable: as the prevalence of smoking continues to decrease over time, revisions of the conversion coefficient would be needed to take this into account.

Conclusions

It is now clear that there has been – and at the present time continues to be – a misrepresentation of the risk of harm from exposure to radon and radon progeny, and an inappropriate use of the quantity effective dose. The misrepresentation arises from the allocation to radon of all of the combined risk from tobacco smoke and radon when most of the magnitude of that risk (it would seem in the region of 85%) is due to tobacco smoke. The inappropriate use of effective dose arises from the conflict between its definition and intended use as a measure of radiation detriment and its actual current use as a measure of tobacco smoke detriment and radiation detriment combined, with the dominant component of the combined detriment being tobacco smoke detriment.

This unfortunate situation can be easily remedied. The definition of effective dose as a measure of radiation detriment should be confirmed, and effective doses from exposure to radon and radon progeny should be estimated without any contribution from tobacco smoke detriment. Recorded doses will then provide a true indication of radiation detriment. At the same time, the combined risk from radon and a population average level of smoking should be used to establish reference levels and derived constraints for living and working environments in order to provide protection for the population as a whole, including smokers and non-smokers.

Epidemiological studies suggest that the dose conversion convention for radon progeny should be in the region of 2 mSv/WLM (0.6 mSv/mJ h m⁻³) for adults at work. The corresponding value for one year's exposure to radon-222 in dwellings⁶ is about 0.7 mSv per 100 Bq m⁻³.

It is interesting to note that the new recommendation of the ICRP in its Statement on Radon (ICRP 2011) for a maximum reference level of 300 Bq m⁻³ for radon-222 in dwellings corresponds to an effective dose of about 2 mSv per year. At this level, the risk of fatal lung cancer for a never-smoker would be about 10⁻⁴ per year. For smokers, the risk would be several times higher. For an indoor radon concentration at the global average level of 40 Bq m⁻³, the effective dose is about

⁵ Such a risk estimate remains a nominal risk – based on risk factors appropriate for the reference individual (ICRP 2002) and smoking status – not a personal risk estimate taking into account a particular person's physiological characteristics.

⁶Using a nominal risk coefficient of 5.7 x 10⁻⁵ per mSv (ICRP 2011) for a population of all ages, and the conventional assumptions given in footnote 3.

0.3 mSv per year. This is comparable to the global average dose from the ingestion pathway, and slightly less than the doses from either cosmic rays or terrestrial radiation (UNSCEAR 2008). It is clear that radon is not the threat that it is often made out to be; the major culprit is tobacco smoke.

It is anticipated that ICRP will soon recommend values for dose coefficients for radon in various exposure circumstances. It has a choice: continue to use a 'smoking effective dose' dominated by tobacco smoke detriment, or use an effective dose that measures radiation detriment. This paper recommends the latter course.

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