Risks from Radon

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Abstract: In its draft report "Radiological Protection against Radon Exposure" (October 2011), the ICRP has reduced the upper reference level for radon gas in dwellings to 300 Bq m^{-3}. There is no specific reference level recommended for workplaces. A risk coefficient of $8x10^{-10}$ per Bq h m^{-3} is recommended without reference to smoking habits. On the basis of these figures:

- The estimated risk of fatal cancer from exposure to radon at home could be greater than the observed risk of accidental death from travelling by car, which would be surprising.
- The estimated risk of lung cancer from radon could be greater than the observed risk of lung cancer from all causes, which is actually known to be dominated by smoking.

The author is not aware of any direct evidence of risks from inhaling radon in Australian dwellings, 99% of which have radon levels below 50 Bq m^{-3}. Evidence available from other countries shows that:

- Effects on the incidences of lung cancer are uncertain at radon levels below 100 Bq m^{-3}.
- The estimation of risks at levels below 200 Bq m^{-3} depends on extrapolation from risks observed at higher levels.
- Risks to non-smokers from radon are much less (perhaps 25 times less) than risks to smokers.

It is concluded that the ICRP draft report and radon policies in the US and UK have the potential to cause unwarranted concern. Some people may be made to feel they need to spend money modifying their homes and workplaces to protect occupants from exposure to radon when there is no compelling reason to show that this is necessary. The vast majority of non-smokers do not need to be protected from radon.

Key words: Radon, risk, smoking, ICRP recommendations, remediation of dwellings

1. Introduction

Radon is a radioactive noble gas, present in the air we breathe due to radioactive decay of naturally occurring uranium and thorium. There are several isotopes of all the elements involved. The decay chain:

$$^{238}U\ (4.47x10^{9}\ y) \rightarrow {}^{234}Th\ (24.1\ d\ ) \rightarrow {}^{234}Pa\ (6.75\ h) \rightarrow {}^{234}U\ (2.46x10^{5}\ y)$$
$$\rightarrow {}^{230}Th\ (7.54x10^{4}\ y) \rightarrow {}^{226}Ra\ (1600\ y) \rightarrow {}^{222}Rn\ (3.82\ d)$$ [see reference 1]

is the most important.

Clearly, radon has always been a part of our environment, throughout evolution, and it is a fundamental principle of evolutionary biology that species adapt to their environments [2]. The amount of radioactivity in the world, including radon, is decaying and has been greater during the earlier stages of evolution than it is today. Currently, the level of radon outdoors, in the open air, ranges from approximately 1 to more than 100 Bq m^{-3} [3].

Human behaviour – particularly living in buildings – has modified our exposure to radon. Because of the ubiquity of uranium and thorium in the earth's crust, radon emanates from the earth beneath buildings and from many building materials, and enters buildings dissolved in water supplies. Despite the short half-lives of its isotopes (see figure 1), it concentrates in enclosed spaces (including the caves in which some of our ancestors lived). Due to poor ventilation of some homes in cold climates, the popularity of basements as living spaces (e.g. in the US) and

some other features of modern living, concentrations of radon in some homes in areas of high natural background radioactivity have become extremely high. Levels have been dangerously high in underground uranium mines (and some other mines) that were not properly ventilated [4].

Figure 1 shows that the radioactive "daughters" from the decay of radon (gas) are all solid [1]. When decay occurs in the bronchus or lung, short-lived nuclides (initially polonium, decaying to radioactive isotopes of lead and bismuth) tend to deposit on the surface of the mucous membrane. A healthy mucous membrane provides some protection to underlying tissue. This protection may be compromised if the mucous membrane is damaged, e.g. by tobacco smoke.

Smoking is the major aspect of modern human behaviour which has modified the risk from exposure to radon. Where the risk of lung cancer due to radon is discernible, it appears to be very much higher for regular smokers than for non-smokers [5,6].

Figure 1: The radioactive daughters of radon isotopes

^{222}Rn	^{219}Rn	^{220}Rn
α ↓ half-life = 3.82 d	α ↓ half-life = 3.96 s	α ↓ half-life = 55.6 s
^{218}Po	^{215}Po	^{216}Po
α ↓ half-life = 3.11 m	α ↓ half-life = 1.78 ms	α ↓ half-life = 0.14 s
^{214}Pb	^{211}Pb	^{212}Pb
β ↓ half-life = 26.8 m	β ↓ half-life = 36.1 m	β ↓ half-life = 10.6 h
^{214}Bi	^{211}Bi	^{212}Bi
β ↓ half-life = 19.8 m	α ↓ half-life = 2.14 m	β ↓ half-life = 60.5 m
^{214}Po	^{207}Tl	^{212}Po
α ↓ half-life = 164 μs	β ↓ half-life = 4.77 m	α ↓ half-life = 0.30 μs
^{210}Pb	^{207}Pb (stable)	^{208}Pb (stable)
β ↓ half-life = 22.3 y		
^{210}Bi		
β ↓ half-life = 5.01 d		
^{210}Po		
α ↓ half-life = 138 d		
^{206}Pb (stable)		

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2. Dosimetry and Risk

As already discussed, it is the decay products of radon rather than radon itself that create the potential for tissue damage. This has given rise to the unit of "working level month" (WLM) in mining, as follows [7].

One WL is the concentration of radon decay products in air which has the same ultimately-delivered alpha energy as 100 pCi L^{-1} (3700 Bq m^{-3}) of radon in equilibrium with its short lived daughters. A WLM is the time integrated exposure from breathing one WL for 170 hours, the notional working hours in a month. There has been a longstanding "convention" that 4 WLM is epidemiologically equivalent to 20 mSv, the occupational dose limit. Hence, 4 WLM y^{-1} has been the occupational exposure limit for miners and is generally equivalent approximately to 2000 hr y^{-1} exposure to 3000 Bq m^{-3}. If the radon concentration cannot be reduced below the workplace action level of (say) 1000 Bq m^{-3}, the operation would become subject to radiation protection regulation. In practice, uranium and mineral sand mining are regulated in Australia in any case.

However, since the energy is delivered to the bronchial cells by decay of the daughters, not the radon atoms, there is no exact equivalence to measured radon concentration. For modern practice in underground uranium mining, active ventilation is used to bring fresh air to the workforce and to remove the radon from work areas. This means that the equilibrium factor in work areas is generally very low (less than 0.2 and frequently less than 0.1) and can be quite variable depending on the ventilation system. Most uranium operations directly measure radon decay products rather than radon, as this is far more relevant for the calculation of dose. For operational mine sites in Australia the use of radon action levels is inappropriate due to the active ventilation and low equilibrium factor [8].

The situation of residential exposure is different from that in mines because the concentration of the decay products in air is likely to be closer to equilibrium with the radon than it is in properly ventilated mines. The situation outdoors is different again because meteorological conditions are likely to disturb the equilibrium.

3. Recommendations of the ICRP

The International Commission on Radiological Protection (ICRP), in its draft report "Radiological Protection against Radon Exposure" (October 2011), has revised the upper value of the reference level for radon gas in dwellings (i.e. where action would almost certainly be warranted to reduce exposure) from 600 Bq m^{-3} to 300 Bq m^{-3}. There is no specific reference level recommended for workplaces (previously 1000 Bq m^{-3}). National authorities are advised to set working levels in accordance with ALARA.

The Commission recommends a risk coefficient of 8×10^{-10} per Bq h m^{-3} for exposure of a population of all ages to radon-222 in equilibrium with its progeny, without reference to smoking habits. This translates into the estimated risks given in table 1 for workplaces and dwellings that comply with the recommended reference levels.

The figures in table 1 may be compared with the figures for other risks given in table 2. These are figures reported in 1989 by the author of the present paper [12], updated in the light of more recent figures for incidences of cancer and road traffic fatalities published by the Australian Bureau of Statistics [13]. When these risks were evaluated in 1989:

- cancer death rates recorded in Australia were somewhat lower than those in the US and about half those in Britain;
- the risk from road traffic accidents in Australia was consistently lower than that in the US and higher than that in Britain (which appears still to be the case).

Table 1: Risks estimated using the risk coefficient of $8x10^{-10}$ per Bq h m^{-3} recommended by the ICRP:
• for typical workplace occupancy;
• for persons who spend most of their time at home; and
• for persons who spend all of their time outdoors

Place	Concentration of radon, Bq m^{-3}	Estimated nominal risk rate, per person per year
Workplace with high level of radon	900	$150x10^{-5}$
Workplace with a lower level of radon	100	$16x10^{-5}$
Dwelling with high but compliant level of radon	250	$\sim 130x10^{-5}$
Dwelling with the mean level of radon in Australia [14]	11	$\sim 6x10^{-5}$
Dwelling with the mean level of radon in Britain [15]	21	$\sim 10x10^{-5}$
Dwelling with the mean level of radon in areas targeted for remediation in Britain [15]	64	$\sim 40x10^{-5}$
Dwelling with the mean level of radon in 27 countries of the European Union [15]	55	$\sim 30x10^{-5}$
Outdoors in the open air, worldwide [3]	About 1 to more than 100	$\sim 0.7x10^{-5}$ to $>70x10^{-5}$

Hence, the implications of the ICRP draft report (October 2011) are that:

- The estimated risk of death from exposure to radon at work and at home could be ten times greater than the observed risk of travelling by car (e.g. between home and work). This would be a surprising conclusion.
- The estimated risk of lung cancer from radon could be two to three times greater than the observed risk of lung cancer from all causes, which is actually known to be dominated by smoking.

The circumstances for these implications to apply would be extreme but circumstances could readily be encountered in which the estimated and observed risks would be of the same orders of magnitude. Even this would be surprising.

Table 1 shows the rate at which people are estimated to incur fatal cancer whereas table 2 shows the rate at which they actually die from various causes. Those who incur fatal cancer from radon

may actually die from some other cause. However, the comparison suggests that the risk coefficient recommended in the ICRP draft report (October 2011) leads to overestimation of the risks from radon.

Table 2: Risks to Individuals from Some Familiar Hazards [12,15]

Nature of the Risk	Risk rate, per person per year
Cancer from all causes in Australia [13]	180×10^{-5}
Lung cancer from all causes in Australia [13]	37×10^{-5}
Smoking 20 cigarettes per day* [12] • all health effects* • all cancers* • lung cancers*	 $\sim 500 \times 10^{-5}$ $\sim 200 \times 10^{-5}$ $\sim 100 \times 10^{-5}$
Drinking alcohol (average for all drinkers)* [12] • all health effects* • alcoholism and alcoholic cirrhosis*	 $\sim 40 \times 10^{-5}$ $\sim 10 \times 10^{-5}$
Road traffic accidents [13] • Australia • France • UK • USA	 8×10^{-5} 9×10^{-5} 6×10^{-5} 14×10^{-5}
Accidents at home in the UK and USA [12]	10×10^{-5}
Drowning (all situations) in Australia [12]	2×10^{-5}

* These are estimated risks from smoking or drinking alcohol (estimated to one significant figure) and are risks only to those who do smoke or drink. Other risks are averages for the whole population, derived from official statistics on the causes of death.

4. Studies of radon health effects

Studies of miners, who worked in underground uranium mines before the problem of radon was properly recognised, have shown that exposure to high concentrations of radon caused increases in the incidence of lung cancer [4]. The correlation between excess lung cancers and exposure to radon was most evident for lifetime exposures above 100 WLM in underground mines and there was evidence of higher risks to smokers than to non-smokers.

Exposure to radon in homes is usually at much lower levels than in underground mines. Therefore, numerous studies of domestic environments have since been carried out. Many of these studies [5,9,10,16,17] are predicated upon (or purport to confirm) the assumption of a linear relationship between risk and radon level without a threshold, i.e. conforming with the linear no-threshold (LNT) model of risk estimation.

The LNT model is recommended by the International Commission on Radiological Protection (ICRP) [11] for use in the practice of radiation protection in general, not just for the case of radon. It is based on risks of cancer observed at high levels of instantaneous exposure to atomic bomb survivors, not chronic exposures such as exposure to natural radiation. There is considerable evidence (summarised in refs. [18-20]) that no risk exists at low doses and that exposure to low levels of radiation is necessary for normal life and health. Roger Clarke, a former chairman of the ICRP, describes the effects of low doses as "irrelevant" [21].

In a geographically based study, Cohen [22] reported lower recorded incidences of lung cancer in the US in areas where measured levels of domestic radon were higher, up to about 200 Bq m^{-3} (i.e. a negative correlation). He could not suggest any explanation for this finding "other than failure of the LNT theory of carcinogenesis". There has been a great deal of controversy surrounding this matter. Puskin [23] and others claim that Cohen's finding has been explained by the finding that people living in high-radon counties (in the US) smoke less than those living in low-radon counties and *vice versa*. Cohen *et al* recognise the latter finding but do not accept it as a compete explanation [24,25]. Geographical studies of other parts of the world [26] have not conclusively found major adverse effects of radon.

Raw data obtained in a cohort study by Turner *et al* [17] showed a trend similar to Cohen's finding, for domestic radon levels up to about 150 Bq m^{-3}. With adjustments for smoking and other factors considered relevant, this trend disappeared and there was no significant effect of radon concentration at all for levels up to about 100 Bq m^{-3}. At higher levels, the incidence of lung cancer was significantly increased. The authors concluded that there was no significant departure from a linear relationship at any level of radon.

Case-control studies, e.g. references [5,9,10,16], have shown "a statistically significant increased risk of lung cancer at average radon concentrations above 100 Bq m^{-3}" [27]. The relationship has been extrapolated in these references as being linear down to the lowest concentrations of radon in homes. However, for levels less than about 100 Bq m^{-3}, effects on the incidence of lung cancer are uncertain.

It should be noted here that the concentration of radon outdoors has a typical level of 20 Bq m^{-3} and ranges up to more than 100 Bq m^{-3} [3].

On the basis of a linear extrapolation, figures presented in ref. [5] show that:

- In the absence of radon, the absolute risks to non-smokers and to cigarette smokers respectively would be 0.41% and 10.1% by age 75. In other words, there is a 0.41% risk of lung cancer which has nothing to do with radon or tobacco smoke, and an additional 9.69% from smoking.
- The incremental risk to non-smokers from radon is about 0.06% per 100 Bq m^{-3} up to 800 Bq m^{-3}.

- Hence, at about 680 Bq m^{-3}, the risk to non-smokers from radon is about the same as the risk from other causes. At lower radon levels, a case of lung cancer to a non-smoker would be more likely to have some cause other than radon (nearly seven times more likely at 100 Bq m^{-3}).
- The incremental risk to smokers from radon is about 1.5% per 100 Bq m^{-3} up to 800 Bq m^{-3} (25 times greater than for non-smokers).
- Hence, at 100 Bq m^{-3} (which is just above the mean value of radon concentration in this study), the risk to non-smokers from radon is 0.06% and the total risk to smokers from radon and smoking combined is 11.2% (nearly 190 times greater).

It therefore seems inappropriate to talk simply of a risk from radon in homes. The risk is from smoking, compounded by a synergistic effect of radon for smokers. **Considering also the uncertainties in the data**, there appears not to be a significant risk to non-smokers from radon, at least up to several hundred Bq m^{-3}.

5. Remediation of Dwellings

The specification of reference levels and action levels for radon gas in dwellings by the ICRP, and in some jurisdictions including Australia, implies that remediation can and should be undertaken if the level is too high. A number of publications deal with this matter, for example refs. [15,28,29].

In Britain [15], detailed consideration with costing has been given to remedial measures such as:

- the addition of active ventilation in existing buildings; and
- membranes in the foundations of new building built in areas where radon levels are expected to be high, and sumps to which active ventilation can be applied if this is found to be necessary.

In ref. [15], estimates of the numbers of deaths from radon related lung cancers are based on the proportionality of risk to radon level (essentially the LNT model) and are used to evaluate the cost-effectiveness of remedial measures. Seventy percent of the **estimated** number of deaths are in homes where the radon level would be below 50 Bq m^{-3}. The difference between the risk from radon to smokers and non-smokers is recognised. However, in areas where the mean level of radon is 64 Bq m^{-3}, all dwelling are apparently targeted for remediation.

In the US [29], the EPA recommends that the owners of homes testing down to 74 Bq m^{-3} should consider mitigating their homes, as there is said to be no "safe" level of radon. The EPA has estimated that, out of a total of 146,400 lung cancer deaths in 1995, 21,100 (14 percent) were related to indoor radon exposure. The great majority of these radon-related deaths would have been attributed to radon levels less than 100 Bq m^{-3}. Most of them were calculated from an estimated interaction between radon and cigarette smoking. Using the collective dose from all homes, with high and low level of radon, the estimate for radon alone was only about 1000 (less than 1 percent) [30].

The use of such figures for policy decisions and to evaluate the cost-effectiveness of reducing radon levels, appears to contravene the ICRP recommendation (paragraph 66 of ref. [11]), which emphasises that:

> "…. [because of the] uncertainty on health effects at low doses, the Commission judges that it is not appropriate, for the purposes of public health planning, to calculate the

hypothetical number of cases of cancer …. that might be associated with very small radiation doses received by large numbers of people over very long periods of time ….".

To put this into context, the dose rate from inhalation of 50 Bq m^{-3} of radon is of the order of 1 mSv per year. Hence, calculating the "hypothetical number of cases of cancer" from radon levels less than 50 Bq m^{-3} is the equivalent of calculating the "hypothetical number of cases of cancer" from annual dose commitments of less than 1 mSv due to fall-out from the Chernobyl reactor accident, which would really not be credible.

As already noted in this paper, studies of the risks from domestic radon show that effects on the incidence of lung cancer are uncertain for radon levels that are less than 100 Bq m^{-3}. At these levels, there might be an inverse correlation (i.e. the incidence of lung cancer decreasing as the concentration of radon increases) or no effect at all. This being so, estimates of the numbers of deaths from radon related lung cancers based on LNT would not be valid, and no discernible benefit should be expected from the reduction of domestic radon levels below 100 Bq m^{-3}.

Reservations must therefore be expressed about some of the findings of ref. [15], *viz* that:

- "[The] current government policy in England of requiring basic preventive measures in all new homes where the mean radon concentration is at least 52 Bq m^{-3} is highly cost effective".
- "Policies involving remedial work on existing homes with high radon levels cannot prevent most radon related deaths, as these are caused by moderate exposure in many homes".
- "Lowering the action level [for remedial work on existing homes] from its current value of 200 Bq m^{-3} to 100 Bq m^{-3} would improve cost effectiveness".

Also, with radon concentrations ranging (worldwide) up to a least 100 Bq m^{-3} outdoors, and a typical level from all isotopes being 20 Bq m^{-3} [3], there must be a limit to the effectiveness of remediating homes to reduce exposures. Whatever the effect of radon may be at these levels – and whether they are "safe" or not – is irrelevant in this context. The average level outdoors in Britain is about 4 Bq m^{-3} [31] and 7-10 Bq m^{-3} in the US [32]. Presumably, in areas targeted for remediation, the level outdoors is likely to be above average and highest where the indoor level is highest.

A conclusion of ref. [15], that radon may need to become a factor in sales of houses, could be a cause for concern when it also found that "remediation rates among homeowners who are lifelong non-smokers are about twice those of homeowners who are current smokers". This suggests that a good understanding of the risk is not the dominant issue in decision making by many members of the public.

6. The Situation in Australia

Potentially harmful exposures to radon would have occurred in mines, particularly uranium mines, in Australia before the problem was recognised. With proper regulation, there should now be no discernible occupational risk from radon.

As far as the author is aware, there is no direct evidence of any risk from inhaling radon in Australian homes.

A study by Langroo *et al* in 1990 [14] and confirmed by Wright *et al* in 2009 [33] showed that the average radon levels in 3,400 randomly distributed and randomly selected dwellings in Australia was 11 Bq m^{-3}. Ninety nine percent of the measured concentrations were smaller than 50 Bq m^{-3}, 0.1% exceeded 100 Bq m^{-3} and only two dwellings exceeded the Australian Indoor Radon Action Level of 200 Bq m^{-3}.

A significant risk to non-smokers should not be expected from inhaling domestic radon at these levels. A small risk to smokers from radon could be estimated by extrapolation from overseas data obtained at levels greater than 200 Bq m^{-3}. This compounds with the much greater risk from smoking itself, e.g. a total lifetime risk on average to smokers of about 13% at 200 Bq m^{-3} compared with about 10% at the mean level of 11 Bq m^{-3}.

7. Conclusions and Recommendations

The risk coefficient of $8x10^{-10}$ per Bq h m^{-3}, recommended in the ICRP draft report "Radiological Protection against Radon Exposure" (October 2011), appears to lead to overestimation of the risks from radon when these estimates are compared with observed risks such as deaths from motor vehicle traffic accidents and lung cancers from all causes. The estimated risks from radon considerably overstate the risk to non-smokers in particular. Without qualification, therefore, the ICRP draft report has the potential to cause unwarranted concern, particularly in relation to workplaces where active ventilation substantially reduces the equilibrium factor between radon and its decay products.

For homes: from consideration of worldwide studies of the risks from exposure to domestic radon, the following recommendations are made for the purpose of this paper:

1. The design objective for new homes (which might be occupied by either smokers or non-smokers) should be the limitation of radon levels to less than 100 to 200 Bq m^{-3}, depending on cost, i.e. to less than 100 Bq m^{-3} if there is no cost penalty in doing so, but in any case to less than 200 Bq m^{-3}.
2. Owners of existing homes in areas where domestic radon levels are expected to be greater than 200 Bq m^{-3} should be advised to have the level measured. Information that is provided should not be in terms that are going to cause unnecessary alarm.
3. Non-smokers with existing homes in which the radon level is found to be greater than 600 Bq m^{-3} should be advised to consider remediation to reduce the level. This would be a very unusual situation in Australia, where the vast majority of non-smokers do not need to be protected from radon.
4. Smokers with existing homes in which the radon level is found to be greater than 200 Bq m^{-3} should be advised to consider remediation to reduce the level. They should also be advised that a more effective and cheaper way of reducing the risk **from radon** is by not smoking.
5. No-one should be made to feel they need to spend money modifying their homes when they do not.

Estimates provided in the BEIR VI report [4] and in reference [15] show that most of the cancers predicted from radon exposure are in homes with low radon levels, where remediation is not recommended. Hence, remediation would have very little impact on the total collective dose and estimated risk from radon [30]. Furthermore, this risk is estimated from application of the LNT model to doses at all levels, whereas there is substantial uncertainty regarding the risk from radon exposure – even for smokers – at low levels (less than about 100 Bq m^{-3}). There may, in fact, be no risk from radon in homes where levels are low.

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