

# Doses to organs and tissues from radon and its decay products

G M Kendall and T J Smith

National Radiological Protection Board, Chilton, Didcot, Oxon OX11 0RQ, UK

E-mail: Gerry.Kendall@nrpb.org

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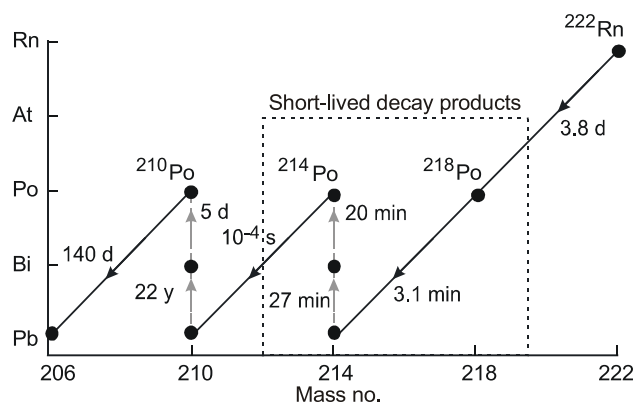
## Abstract

This paper discusses the doses from radon and from its short-lived decay products to a number of organs and tissues and to the foetus. The aim is to put all these doses into context rather than concentrating only on the largest contributions. There is also a brief discussion of the evidence from epidemiology on the risks of exposure to radon and its decay products. As is well known, under normal circumstances the greatest hazard is to the respiratory tract from inhalation of radon decay products. Radon decay products may also give substantial doses to skin. Under some circumstances it seems likely that ingested radon could give significant doses to the stomach. Other risks appear to be smaller; the results given here allow them to be compared.

## 1. Introduction

The natural radioactive gas, radon, decays through a series of short-lived decay products before reaching  $^{210}\text{Pb}$ , a nuclide with a 20 year half-life, and finally stable  $^{206}\text{Pb}$ . Lead-210 is an important nuclide in its own right, but is beyond the scope of this paper which deals with radon and its short-lived decay products. Details of the decay scheme for radon are shown in figure 1.

Radon is known to present a risk of lung cancer when it, or rather its decay products, are inhaled. This hazard was recognised early (see ICRP (1993a) for a discussion of historical developments as well as a general review) and has been extensively reviewed and commented upon, for example by the US National Research Council (NRC) (NRC 1999a). Two other potential hazards are also recognised. Calculations suggest that the ingestion of water which contains particularly high levels of radon can lead to a significant risk of stomach cancer (Hursh *et al* 1965 has an early discussion). Calculations also indicate that deposition of radon decay products may, under some circumstances, lead to significant doses to sensitive cells in the skin, again with cancer as a possible consequence (Harvey (1971) drew attention to the possibility of skin dose from alpha emitters). A brief summary of risks from radon and its decay products, albeit with emphasis on the lung, has been given elsewhere (NRPB 2000b).



**Figure 1.** Decay scheme for  $^{222}\text{Rn}$  showing the short-lived decay products to  $^{210}\text{Pb}$  and the subsequent transformations to  $^{206}\text{Pb}$ .

However, radon and its decay products can also give doses to other organs and tissues. These are normally much less important but do also arouse interest. This paper describes these doses, in particular those to body organs other than the organ of intake (i.e. the lung in the case of inhalation, stomach for ingestion). There is also interest in the contribution to overall doses from both radon itself and from its decay products, even when one or the other normally dominates. This might be because of unusual circumstances in which the normal equilibrium is significantly disturbed. There may also be particular interest in doses to a specific organ (e.g. red bone marrow) or to the foetus. This paper therefore considers doses to various organs and tissues (and to the foetus) from inhalation and from ingestion of radon gas and its short-lived decay products. Doses to the skin from external radiation by radon decay products are also considered. We do not attempt to list all published papers on these topics. In the section above we have given a historical reference on each main theme. In the sections which follow we cite some of the main papers and reviews with emphasis on those with results that we use.

In order to keep the tables of manageable size, the organs for which doses are listed have been selected primarily on the basis that they receive higher than average doses. However, doses to gonads are also given because of the high tissue weighting factor (ICRP 1991). Dose to muscle is generally indicative of the level of dose received by organs other than those considered specifically.

Estimates of doses from internal emitters are obtained by mathematical modelling (ICRP 1979, 1990). A brief outline of the modelling involved is provided in the appendices. These provide only a simplified account and the original references should be consulted for more details. In the calculations reported here the radiation and tissue weighting factors, tissue masses, radioactive half-lives etc are those of the International Commission on Radiological Protection (ICRP) (ICRP 1983, 1991, 1996a). Approximate half-lives and other parameters are given in the text.

We note that 'radon' is often used as a convenient shorthand for radon and its immediate short-lived decay products when they are inhaled. In this paper we consider the gas and its short-lived decay products separately.

## 2. Doses from inhaled radon and its decay products

It has been known for many years that exposure to an atmosphere containing radon can increase the risk of lung cancer (a historical review is in ICRP 1993a; see also Sevc *et al* 1976, ICRP

1987 and NRC 1988, NRC 1999a). It is now understood that the most important component of the dose comes not from the gas itself, but rather from its short-lived decay products. Radon itself is an inert gas with a half-life of about 4 days and almost all the gas that is inhaled will be breathed out again. However, the decay products are isotopes of solid elements and will quickly attract to themselves molecules of water and other atmospheric gases. These, in turn, attach to natural aerosol particles. If inhaled, the decay products, whether attached to aerosol particles or 'unattached', will largely be deposited on the surface of the respiratory tract and, because of their short half-lives (less than half an hour) will decay there. Because strong evidence on the risks of inhaled radon is available from epidemiological studies, it is normal to derive risk factors from this source. The alternative is to estimate doses using models based on the available information on distribution and retention of radionuclides in different organs and tissues. These doses are then used to estimate risks based on risk factors recommended by the ICRP. This indirect approach is usual in radiation protection where risks are generally too small to be observed. It is unnecessary in the case of radon decay products. However, in this paper, in order to allow comparisons between different routes of exposure, some calculated doses to the respiratory tract will be presented.

There is no international consensus on the calculation of doses from inhaled radon decay products and the ICRP has recommended that the model of the human respiratory tract should not normally be used for calculating doses from radon decay products for the assessment of risks (ICRP 1993a, para 38). The doses to the respiratory tract given here, therefore, cannot be regarded as definitive. However, they provide a convenient yardstick against which other doses can be assessed. We note that a specific variant of the human respiratory tract model has been developed for radon decay products (Marsh and Birchall 2000); this does not, as yet, follow material outside the respiratory tract and provide estimates of doses to other organs. The calculations presented here, carried out using the PLEIADES code (Bailey *et al* 2000), use type M and type F kinetics coupled with the appropriate ICRP systemic model for the element in question. PLEIADES is one of the codes used by the ICRP Task Group on Dose Calculations ('DOCAL') in calculating dose coefficients for recent publications (ICRP 1990, 1993b, 1994b, 1995a, 1995b, 1996a, 1996b, 1998, 2001). The appendices present more detail of the calculational methods. PLEIADES uses the new ICRP model of the human respiratory tract (ICRP 1994a, 1994b). A number of parameters must be chosen in the application of the ICRP model of the respiratory tract. Clearance kinetics are discussed above and particle size in the section which follows. Otherwise parameters appropriate to an adult worker are applied.

Table 1 gives dose coefficients for the inhalation of radon decay products attached to aerosols of 1 and 200 nm activity median aerodynamic diameter (AMAD). These correspond broadly to the unattached (sometimes called ultrafine) and to the attached fractions respectively. Deposition of the unattached fraction is dominated by diffusion (see appendix C); the activity median thermodynamic activity (AMTD) for this aerosol is about 0.6 nm. As noted above, results are presented for type F and type M aerosols. Type F material has a 10 min half-time for absorption to blood, while that for type M is much longer (see appendix C). For type M, virtually all the decay products decay in the respiratory tract. For type F, a significant proportion is cleared before it decays and doses to tissues outside the respiratory tract are somewhat higher, while effective dose is reduced. However, for type F most of the short-lived decay products which escape from the lung will decay in blood before reaching sensitive organs. This results in a low component of dose to the whole body.

Perhaps 10% of the activity of radon decay products is associated with the unattached fraction (Porstendoerfer and Reineking 1992, NRC 1999a). The pattern of organ doses differs somewhat for the 1 and 200 nm AMAD aerosols. A greater proportion of the smaller particles deposit in the extrathoracic part of the respiratory tract and therefore give relatively higher doses

**Table 1.** Dose coefficients ( $\text{Sv Bq}^{-1}$ ) for inhalation of short-lived radon decay products. (Note: ET is the extrathoracic part of the respiratory tract, comprising the nose, mouth, pharynx and larynx. CED is committed effective dose.)

		Aerosol of 1 nm AMAD (0.6 nm AMTD)				Aerosol of 200 nm AMAD			
	Tissue	$^{218}\text{Po}$	$^{214}\text{Pb}$	$^{214}\text{Bi}$	$^{214}\text{Po}$	$^{218}\text{Po}$	$^{214}\text{Pb}$	$^{214}\text{Bi}$	$^{214}\text{Po}$
Absorption type F	Lung	2.4E-08	3.8E-08	1.1E-07	4.6E-14	9.3E-09	1.1E-08	3.1E-08	1.2E-14
	ET	2.6E-08	1.6E-07	3.7E-07	2.1E-13	2.0E-09	1.3E-08	2.9E-08	1.7E-14
	Next	Kidney	Kidney	Kidney	Kidney	Kidney	Kidney	Kidney	Kidney
	Highest	1.1E-10	6.0E-09	7.9E-09	6.9E-19	8.3E-11	4.7E-09	5.6E-09	5.0E-19
	CED	3.5E-09	8.9E-09	2.2E-08	1.1E-14	1.1E-09	1.8E-09	3.8E-09	1.9E-15
Absorption type M	Lung	6.6E-08	3.6E-07	2.9E-07	4.6E-14	2.3E-08	1.1E-07	8.5E-08	1.2E-14
	ET	3.9E-08	2.8E-07	5.4E-07	2.1E-13	3.1E-09	2.2E-08	4.3E-08	1.7E-14
	Next	Stomach	Kidney	Kidney	Kidney	Kidney	Kidney	Kidney	Kidney
	Highest	5.4E-11	7.0E-10	8.1E-10	1.9E-19	9.1E-12	4.8E-10	5.6E-10	5.9E-19
	CED	7.9E-09	4.3E-08	4.9E-08	1.1E-14	2.8E-09	1.3E-08	1.0E-08	1.9E-15

here, though doses to all tissues are higher than for the larger aerosol. Committed effective doses are somewhat higher per unit activity for the unattached compared with the attached fraction (by a factor of three to five). Note that the immediate decay product of  $^{214}\text{Bi}$ ,  $^{214}\text{Po}$ , gives negligible doses because of its very short half-life and intakes of this nuclide in its own right can be ignored. Ingrowth of  $^{214}\text{Po}$  from  $^{214}\text{Bi}$  (i.e.  $^{214}\text{Po}$  produced by decay of  $^{214}\text{Bi}$  in the body) is, of course, considered in the calculated dose coefficients.

If radon is allowed to reach equilibrium with its decay products there will be equal activities of each. In practice, the later decay products tend to be lost by various processes, particularly deposition on surfaces, sometimes called 'plateout' (Porstendoerfer and Reineking 1992). The precise mixture of nuclides in any circumstance will depend on a number of factors such as the aerosol concentration and ventilation rate. We will assume that the activities of

$$^{222}\text{Rn}:^{218}\text{Po}:^{214}\text{Pb}:^{214}\text{Bi}$$

are in the ratio of

$$1:0:0.9:0.45:0.225.$$

This corresponds to an equilibrium factor,  $F$ , of 0.41, reasonably typical of domestic, and many occupational, conditions (Wrixon *et al* 1988, NRC 1999a). Table 2 gives dose coefficients for inhalation of a mixture of 10% unattached (1 nm AMAD) and 90% attached (200 nm) aerosols of the individual nuclides and for the mixture of decay products specified above. Results for selected organs are given, together with the committed effective doses. Dose to kidney is shown because it is the organ receiving the highest dose outside the respiratory tract. For type M material, dose to the lung accounts for more than 99% of the effective dose; for type F, over 95% of the effective dose is due to the respiratory tract, including the extrathoracic part.

The annual intake of indoor air for an adult may be taken to be  $7300 \text{ m}^3$  (NRPB 2000a). This figure, appropriate for a sedentary worker or member of the general public, is based on data relating to Reference Man (ICRP 1975) updated in the light of reviews undertaken during the development of the new ICRP model of the human respiratory tract (ICRP 1994a, Robinson 1996). For illustrative purposes, table 2 also shows annual doses from radon decay products if the concentration of radon gas is  $200 \text{ Bq m}^{-3}$  (corresponding to an annual exposure of about 0.9 WLM in traditional units).  $200 \text{ Bq m}^{-3}$  has been chosen as the action level by many countries, including the UK (Colgan 1996). The annual committed effective dose is then

**Table 2.** Summary of dose coefficients from inhaled radon decay products together with annual doses from decay products and radon at 200 Bq m<sup>-3</sup>. Note: the activity of decay products in comparison with radon is taken to be <sup>218</sup>Po:<sup>214</sup>Pb:<sup>214</sup>Bi in the ratio 0.9:0.45:0.225;  $F = 0.41$ . 90% of decay products are taken to be attached to aerosols, 10% unattached. Annual volume breathed taken to be 7300 m<sup>3</sup>. RBM is red bone marrow. CED is committed effective dose. Kidney is shown as the organ receiving the next highest dose after the respiratory tract. Dose to foetus from Rn gas taken to be that to muscle over 9 months. Dose to skin is taken from Eatough and Henshaw, quoted in NAS, 1999. It does not depend on type.

	Type F Dose coeffs Sv Bq <sup>-1</sup> intake					Type M Dose coeffs Sv Bq <sup>-1</sup> intake					<sup>222</sup> Rn
	<sup>218</sup> Po	<sup>214</sup> Pb	<sup>214</sup> Bi	Mixture	Annual dose at 200 Bq m <sup>-3</sup> mSv	<sup>218</sup> Po	<sup>214</sup> Pb	<sup>214</sup> Bi	Mixture	Annual dose at 200 Bq m <sup>-3</sup> mSv	Annual dose at 200 Bq m <sup>-3</sup> mSv
Lung	1.1E-08	1.4E-08	3.9E-08	2.5E-08	35.8	2.7E-08	1.3E-07	1.1E-07	1.1E-07	159	1.2
Ext. thor	4.4E-09	2.7E-08	6.3E-08	3.0E-08	44.5	6.7E-09	4.8E-08	9.3E-08	4.9E-08	70.9	
Stomach	3.3E-11	1.7E-10	9.4E-11	1.3E-10	0.19	1.2E-11	7.7E-11	4.9E-11	5.6E-11	0.08	0.06
Small intest.	3.0E-11	1.7E-10	7.5E-11	1.2E-10	0.17	6.9E-12	4.9E-11	1.8E-11	3.2E-11	0.05	0.06
Colon	2.8E-11	1.5E-10	8.2E-11	1.1E-10	0.16	3.9E-12	2.4E-11	9.9E-12	1.7E-11	0.02	0.05
RBM	3.9E-11	3.2E-10	6.8E-11	1.9E-10	0.28	4.2E-12	3.7E-11	7.4E-12	2.2E-11	0.03	0.65
Bone sur	3.4E-11	2.1E-09	6.8E-11	1.0E-09	1.48	3.6E-12	2.5E-10	7.3E-12	1.2E-10	0.17	0.03
Liver	5.8E-11	5.0E-10	6.8E-11	2.9E-10	0.43	6.3E-12	5.6E-11	7.6E-12	3.2E-11	0.05	0.09
Breast	2.8E-11	1.4E-10	6.8E-11	1.0E-10	0.15	3.0E-12	1.6E-11	7.8E-12	1.2E-11	0.02	0.42
Kidney	8.7E-11	4.8E-09	5.8E-09	3.6E-09	5.20	9.5E-12	5.0E-10	5.9E-10	3.7E-10	0.54	0.05
Gonads	2.8E-11	1.4E-10	6.8E-11	1.0E-10	0.15	2.9E-12	1.5E-11	7.1E-12	1.1E-11	0.02	0.05
Brain	2.7E-11	1.5E-10	6.8E-11	1.1E-10	0.15	2.9E-12	1.5E-11	7.1E-12	1.1E-12	0.02	0.06
Bladder	2.8E-11	2.1E-10	1.0E-10	1.4E-10	0.21	2.9E-12	2.2E-11	1.0E-11	1.5E-11	0.02	0.05
Muscle	2.8E-11	1.4E-10	6.8E-11	1.0E-10	0.15	3.0E-12	1.6E-11	7.4E-12	1.1E-11	0.02	0.05
CED	1.4E-09	2.5E-09	5.6E-09	3.6E-09	5.30	3.3E-09	1.6E-08	1.4E-08	1.3E-08	19.7	0.28
Foetus	1.3E-11	5.3E-11	2.6E-11	4.1E-11	0.06	1.6E-12	6.2E-12	2.9E-12	4.9E-12	0.01	0.04
Skin					25					25	

about 5 mSv if type F behaviour is assumed or about 20 mSv for type M. Epidemiological data would agree with the former figure while more detailed dose calculations would be close to the type M result (ICRP 1993a, Birchall and James 1994, Marsh and Birchall 2000). Alternative models, predicting somewhat different doses, have also been presented (e.g. Porstendoerfer 2001). This small discrepancy between epidemiology and dosimetry is well known (ICRP 1993a). It will not be discussed here, other than to emphasise that type F biokinetics should not be accepted as correct purely because they produce dose estimates which agree with epidemiology. However, it does suggest that the calculations for type F and type M may be taken to illustrate an indicative spectrum of doses from radon decay products. These results therefore provide a useful basis for comparing the doses calculated for various organs and also the doses from radon gas compared with decay products.

The doses from radon decay products to organs outside the respiratory tract depend on the rate at which material is cleared to blood. For type M material, clearance to blood is so slow that the great majority of short-lived decay products deposited in the lung decay there. Clearance to blood is much faster for type F materials and these give lower doses to lung and larger doses to organs outside the respiratory tract. This can be seen from the data in table 2. It is interesting to look at the doses to the kidney, the most exposed organ outside the respiratory tract. For type M material the dose to the kidney is always at least an order of magnitude lower than the committed effective dose and two orders of magnitude lower than the lung dose. For type F material, the kidney dose is relatively larger, but still always substantially lower than that to lung. It may be noted that the doses to the extrathoracic part of the respiratory tract ('ET') are also higher than those to the kidney and may be higher than those to the lung as a whole.

These doses from radon decay products may be compared with those which would result from breathing air containing radon gas, but not the decay products. Decay products formed within the body are of course considered in the calculation. Table 2 also shows estimates of dose from 1 year's exposure to  $^{222}\text{Rn}$  at  $200 \text{ Bq m}^{-3}$ . These dose estimates are based on a paper by Khursheed (2000) in which he uses a dynamic model to calculate doses to a wide range of tissues from inhalation and ingestion of radon gas. The calculated committed effective dose is about 0.3 mSv, an order of magnitude lower than that from decay products if they display type F behaviour and two orders of magnitude lower than type M. This illustrates the rule given in the introduction, that the inhaled decay products are generally much more important than radon gas.

It is interesting to consider the relative magnitude of doses to different organs and tissues from inhalation of radon gas. Doses are generally much lower than those from decay products. As with the decay products, the lung is the organ receiving one of the highest doses, in this case largely because of the contribution from decays in the air within the lung. Over half the committed effective dose is due to lung dose. However, radon is more soluble in tissues with a higher fat content (Nussbaum and Hursh 1957). Fat (which is not thought to be radiosensitive) receives the highest dose of all tissues outside the lung. Red bone marrow has a reasonably high fat content and this also receives a relatively high dose, as does the female breast. The doses to these two tissues are perhaps a factor two higher than those from the radon decay products if type F behaviour applies and an order of magnitude higher than type M. They remain, however, much less than the doses to lung.

A foetus can take up material from the mother's bloodstream via the placenta. Elements vary in their ability to cross this placental barrier. It has been recommended by the ICRP (ICRP 2001) that the concentration of lead in the foetus is taken to be equal to the concentration in the mother regardless of when the intake occurred; for polonium and bismuth the concentration ratio (foetal:maternal) is taken to be 0.1, indicating modest discrimination against these elements by the placenta. The ICRP makes no recommendation for radon gas. However,

for many radionuclides the dose to the foetus is similar to that to maternal muscle. As the fat content of the foetus is low, it can be assumed that maternal muscle provides a reasonable surrogate.

### 3. Doses to skin

Radon decay products can deposit on skin in much the same way that they plate out on other surfaces. When they decay, much of the alpha particle energy will be harmlessly absorbed in the dead outer layer of the skin. However, where the skin is thin, for example on the face, the alpha particles may be able to reach the sensitive basal cells where it is believed that skin cancers originate.

Estimates of dose have been given by a number of authors (e.g. Harvey 1971, Sevcova *et al* 1978, Harley *et al* 1983, Miles 1986, Eatough and Henshaw 1992, Harley and Robbins 1992, Eatough and Henshaw 1995, Eatough 1997). NRPB has reviewed skin dosimetry generally, but without particular focus on alpha emitters (NRPB 1997). The dose will depend on the concentration of radon decay products in the air, their mean lifetime and on the deposition velocity. The last is a crucial parameter which depends on particle size and shape and on air movement. Radon decay products are initially formed in a charged state and their deposition velocity can be strongly influenced by environmental electric (but not magnetic) fields. Once deposited on skin, the radon decay products emit alpha particles with a range of several tens of microns (about 47 and 70  $\mu\text{m}$  for alpha particles from  $^{218}\text{Po}$  and  $^{214}\text{Po}$ ). This is comparable with the depth of the basal layer of the epidermis. However, the target cells for induction of skin cancer are not known with certainty and the detailed anatomy of skin varies from individual to individual and across the body surface. These factors introduce uncertainty into the calculation of doses, though the difficulties are arguably less than those encountered in lung dosimetry. These calculations refer to unprotected skin. Under normal circumstances, most of the skin on the body is protected by clothing or by hair. It had been suggested (ICRP 1992) that the radiosensitivity of skin was very largely limited to those parts of it which were also exposed to ultraviolet radiation. These are the parts which will tend to receive the dose from radon decay products. However, the latest analysis of basal cell carcinoma in survivors of the atomic bombs do not support this idea (Ron *et al* 1998).

Despite these uncertainties, it seems very probable that the dose to the basal layers of the skin from radon decay products is not negligible and that, if these are the cells at risk, there is some risk of skin cancer. Estimates of dose to the skin from a year's exposure at the action level of 200  $\text{Bq m}^{-3}$  are also given in table 2. These are from the BEIR VI report (NRC 1999a) and relate to the skin of the face and neck, where the basal cells are taken to be at a depth of 50  $\mu\text{m}$ . BEIR VI quote substantial ranges based on work by Harley and Robbins (1992) and by Eatough and Henshaw (1992). The central value (25  $\text{mSv yr}^{-1}$  at 200  $\text{Bq m}^{-3}$ ) is based on later work by the latter authors (Eatough and Henshaw 1995). Eatough *et al* (1999) have presented experimental results using personal 'wristwatch' dosimeters which support the magnitude of these doses, and would, if anything, suggest that they might be somewhat higher.

It has been suggested that doses to skin from radon decay products may be enhanced by rain-out (Henshaw *et al* 2001). The removal of radon decay products from the atmosphere by rain has been discussed by a number of authors. There appears to be a consensus that the high rates of diffusion of unattached decay products will result in them quickly becoming incorporated into water droplets. Attached decay products are much less quickly removed and atmospheric mixing may be important (Jacobi 1961, Blaauboer and Smetsers 1997). Whatever the mechanism by which it accumulates, high concentrations of radon decay products have been reported in some rain samples (Paatero 2000). The measurements of Paatero and of

Henshaw *et al* suggest that much of the removal of decay products occurs in the early part of a period of rain. Henshaw *et al* argue, on the basis of surface deposition on plastic alpha track detectors, that if such rain falls on skin it will give large doses. The practical implications of these observations, assuming that the observations on detector plastic carry over to skin, are not easy to assess. Most people are, of course, averse to rain and will try to avoid it, or at least to minimise the amount of wetted skin.

Eatough and Henshaw (1995) have estimated that perhaps 2% of non-melanoma skin cancers in the UK may be caused by radon decay products. This estimate depends critically on the depth of the sensitive cells. It should be remembered that most such skin cancers have a high survival rate, though this is not to imply that they are of no significance.

#### 4. Doses from ingestion

Radon is soluble in water and this route of exposure may also be important if high concentrations are found in drinking water (Hursh *et al* 1965, Suomela and Kahlos 1972, Dundulis *et al* 1984, Gosink *et al* 1990, Crawford-Brown 1991, Sharma *et al* 1997, NRC 1999b). If such water is ingested, most models describe the radon as remaining in the stomach for several tens of minutes before being passed to the small intestine where it is transferred to blood and is rapidly lost from the body (Khursheed 2000). Calculations show that the dose to the lining of the stomach can be significant and this implies some risk of stomach cancer. However, as is discussed below, an association between radon and stomach cancers has not been demonstrated in epidemiological studies. This is in contrast to the wealth of data on lung cancers induced by inhaled radon decay products. This is not unexpected since the doses to stomach are generally much smaller than those to lung (see below). UNSCEAR (2000) gives very rough estimates of global mean annual effective doses: 1.1 mSv from inhalation of decay products; 0.002 mSv from ingestion of water.

Table 3 presents doses from ingestion of radon decay products and from radon itself. These data are from the ICRP CD compendium of dose coefficients (ICRP 1998) and from Khursheed (2000) respectively. Ingested material is first held in the stomach. Because all the nuclides concerned have short physical and/or biological half-lives most of them decay here and dose to stomach dominates that to other organs, both for radon itself and for the decay products. Over 95% of the effective dose from ingested radon gas is due to stomach dose; for the decay products, the figure is over 80%.

The annual water intake for an adult may be taken as 600 l (NRPB 2000a). For illustrative purposes, table 3 also shows annual doses from intakes of 600 l of water containing 1000 Bq l<sup>-1</sup>. This is recommended by the European Union as an Action Level for radon in private water supplies (European Commission 2001) and has been adopted in the UK (NRPB 2001). In many cases, a substantial fraction of this water is likely to have been boiled before it is ingested. Boiling expels most, if not effectively all, the radon, so doses incurred in practice will usually be lower than those shown. Doses from intakes of other volumes of water or from other concentrations of radon may be obtained by scaling the doses shown here.

In contrast to the situation for inhalation, doses from ingestion of radon gas dominate those from ingestion of the decay products even if it is conservatively assumed that all the decay products are in equilibrium. This is because radon, with its much greater half-life, irradiates tissues for longer than do the decay products. Once it leaves the stomach, ingested radon gas enters the blood stream, passes through the liver and is lost from the lungs. The stomach receives much the highest dose, with minor contributions from liver and lung. Fatty tissues (e.g. red bone marrow and breast) also receive higher doses than other body organs because of

**Table 3.** Summary of dose coefficients from ingested radon decay products and from radon gas together with annual doses from ingesting water containing 1000 Bq l<sup>-1</sup>. (Note: all radon decay products are taken to be in equilibrium. Assumed annual water intake 600 l. RBM is red bone marrow. CED is committed effective dose. Dose coefficients for the foetus assume intake of 1 Bq over pregnancy. Dose coefficient for the foetus from Rn gas are taken to be that to muscle.)

	Radon decay products				<sup>222</sup> Rn		
	Dose coeffs Sv Bq <sup>-1</sup> intake				Annual dose at 1000 Bq l <sup>-1</sup> mSv	Dose coeffs Sv Bq <sup>-1</sup> intake	Annual dose at 1000 Bq l <sup>-1</sup> mSv
	<sup>218</sup> Po	<sup>214</sup> Pb	<sup>214</sup> Bi	Mixture			
Lung	9.8E-12	1.2E-11	2.1E-12	2.4E-11	0.01	2.1E-09	1.26
Stomach	1.9E-10	8.6E-10	8.6E-10	1.9E-09	1.15	8.4E-08	50.4
Small intest.	6.9E-11	5.5E-10	2.3E-10	8.5E-10	0.51	4.3E-09	2.6
Colon	2.5E-11	1.5E-10	3.6E-11	2.1E-10	0.13	8.8E-11	0.1
RBM	1.4E-11	3.0E-11	2.6E-12	4.7E-11	0.03	1.1E-09	0.66
Bone surf.	1.2E-11	1.9E-10	1.7E-12	2.0E-10	0.12	5.2E-11	0.03
Liver	2.0E-11	4.1E-11	3.0E-12	6.4E-11	0.04	9.5E-10	0.57
Breast	9.7E-12	1.1E-11	1.7E-12	2.2E-11	0.01	7.4E-10	0.44
Kidney	3.0E-11	3.3E-10	6.2E-11	4.2E-10	0.25	8.1E-11	0.05
Gonads	1.1E-11	2.3E-11	6.0E-12	4.0E-11	0.02	8.8E-11	0.05
Brain	9.6E-12	1.0E-11	7.1E-13	2.0E-11	0.01	1.0E-10	0.06
Bladder	1.0E-11	1.8E-11	2.5E-12	3.1E-11	0.02	8.8E-11	0.05
Muscle	9.9E-12	1.3E-11	2.5E-12	2.5E-11	0.02	8.8E-11	0.05
CED	3.5E-11	1.4E-10	1.1E-10	2.9E-10	0.17	1.0E-08	6.00
Foetus	5.1E-12	1.3E-11	4.6E-12	2.3E-11	0.01	1.0E-10	0.05

radon's preferential solubility in fat. Other body organs receive doses which are typically two orders of magnitude lower than the committed effective dose.

As discussed above, the foetus does not preferentially take up radon from the bloodstream and would receive a dose similar to that to muscle.

We note that other calculations of doses from ingested radon have also been presented, notably by the NRC (1999b) in the United States. These are broadly consistent with those of Khursheed (2000), though the NRC dose to stomach, while still the highest to any organ, is lower than that calculated by Khursheed by a factor of about three ( $2.4 \times 10^{-8}$  versus  $8.4 \times 10^{-8}$  Sv Bq<sup>-1</sup>). The NRC also summarise other dose coefficients in the literature in their table 4.4, the range is from  $1.1 \times 10^{-7}$  to  $1.6 \times 10^{-9}$  Sv Bq<sup>-1</sup>). The difference in dose coefficient between the calculations of Khursheed and NRC arises because the NRC calculation models diffusion of radon into the stomach wall, while those of Khursheed simply hold the radon in the stomach until it is absorbed from the small intestine. Those comparing the two calculations should also note that those of Khursheed have conservatively assumed a higher daily intake of unboiled water, again by a factor of about three.

Some of the radon dissolved in tap water will escape to indoor air. The proportion lost in this way will depend on circumstances. However, UNSCEAR has suggested that, as a general rule, radon in tap water gives rise to radon in room air at a concentration  $10^{-4}$  lower than that in the water (UNSCEAR 1993). The NRC (NRC 1999b) in a recent review also recommended this value. This means that radon in drinking water at 1000 Bq l<sup>-1</sup> would give rise to radon in room air at about 0.1 Bq l<sup>-1</sup>, i.e. 100 Bq m<sup>-3</sup>. Comparison of the doses in table 2 (scaled by a half) and those in table 3 indicates that radon, initially dissolved in water, which escapes to atmosphere may give rise to an inhalation hazard which exceeds that from ingestion. In a

somewhat different context, we note that radon escaping from ground water can give substantial occupational doses by inhalation to those employed in some water works (Schmitz and Nickels 2001). It is also true that the geological factors which lead to high concentrations of radon in water may independently give rise to high levels of radon in indoor air.

## 5. Discussion

This paper has presented a wide-ranging summary of doses from radon and its decay products by inhalation, ingestion and deposition on skin. Of course, significant uncertainties are attached to estimates of doses from radionuclides (Leggett *et al* 1998, Leggett 2001, Harrison *et al* 2001). These may be especially significant in the present context, where doses have been estimated for short-lived radon decay products using models which were developed primarily with longer-lived radionuclides in mind. Little attention should therefore be given to small differences in calculated doses to organs. However, substantial differences, which can usually be understood on the basis of the expected behaviour of the element in question, give an indication of the pattern of risks to different organs and tissues.

We also note that other authors have presented calculations of doses from radon and its decay products. Some of these have been discussed above. The calculations of Henshaw and co-workers on doses to red bone marrow also deserve mention. In particular, Richardson *et al* (1991) have presented calculations which are equivalent to an annual dose to red bone marrow of about  $0.89 \text{ mSv yr}^{-1}$  from inhaling radon at  $200 \text{ Bq m}^{-3}$ . Allen *et al* (1995) have presented data consistent with this estimate and also information about age dependence of fat cells in bone marrow. The figure presented here,  $0.65 \text{ mSv yr}^{-1}$ , is not significantly different from these estimates. The former figure was used in an assessment of doses from all sources of radiation in the Seascale area (Simmonds *et al* 1995).

In the sections above, we have noted that epidemiological studies have detected the association between elevated exposures to radon decay products and lung cancer but not, in our view, other effects of exposure to radon or its decay products. It may be objected that this is purely because the appropriate studies have not yet been conducted. In principle, a well designed epidemiological investigation of a highly exposed cohort of sufficient size might demonstrate, for example, a risk of stomach cancer in those drinking high radon water over long periods. But assembling such a cohort would be very difficult in most parts of the world, and probably quite impractical in the UK.

We note that Boice *et al* (1996) have argued that factors such as confounding and statistical power are likely to prevent single epidemiological studies from demonstrating a relative risk if it is in the region of 1.3–1.4 or less. This is not to deny the consequences of the linear no-threshold hypothesis, that low doses of radiation lead to small deleterious effects. But it warns that such effects may not be demonstrable. It is easier to demonstrate an effect the larger the number of radon induced cancers of the type in question and the smaller the background level of such cancers. Even allowing for the latter point, there is, perhaps, an argument that those risks which are large enough for epidemiology to demonstrate are of greater public health importance.

The estimates of annual doses given above relate to intakes at the respective UK Action Levels for radon in the air in dwellings and for radon in private water supplies. Doses from different concentrations of radon may be obtained by linear scaling. Typical UK doses would be lower than those given above by about a factor of 10 for radon in air and perhaps a factor of 1000 for radon in water.

Much the largest doses from inhaled radon and decay products are to the respiratory tract. There is plentiful epidemiological evidence to confirm the link between radon exposure and lung cancer (NRC 1999a, Darby *et al* 2001). Much of this evidence comes from occupationally

exposed miners (Lubin *et al* 1994), but direct evidence from case-control studies of domestic exposures is mounting (Lubin and Boice 1997, Darby *et al* 1998). The National Radiological Protection Board (NRPB) has advised that, in the UK, action should be taken to reduce radon concentrations in homes if they exceed the Action Level of 200 Bq m<sup>-3</sup> (NRPB 1987, 1990). This advice has been accepted by government (Parliament 1987). It has been estimated that perhaps a quarter of a million people live in homes where the radon in air concentration exceeds the UK action level (Wrixon *et al* 1988).

Doses to other body organs from inhaled radon decay products are usually at least an order of magnitude lower (generally much lower) than those to lung (table 2). No strong evidence for a link between radon exposure and other types of cancer has been found in a large combined study of miner cohorts (Darby *et al* 1995).

Nevertheless, interest in other types of malignant disease has been expressed, in particular leukaemia as a consequence of irradiation of red bone marrow. Red bone marrow does not receive a particularly high dose from inhaled radon decay products. Dose to red bone marrow is relatively larger if exposures to pure radon are considered, but even here it is not dominant and under normal circumstances, where radon decay products predominate, its contribution is small. Some geographical correlation studies have reported a link between radon levels and leukaemia, but more reliable case-control studies have not (Lubin *et al* 1998, Laurier *et al* 2001).

However, radon decay products may deliver relatively high doses to skin (delivered, of course, by plateout rather than by inhalation). Particular uncertainties apply to these dose calculations. Not only is the position of the sensitive cells not known with certainty but the doses are certainly influenced by environmental electric fields and by air movements. However, calculations suggest that doses can be substantial. Non-melanoma skin cancers have been observed in populations exposed to low-LET radiation (UNSCEAR 2000) and in one cohort of miners exposed to radon (Sevcova *et al* 1978). Etherington *et al* (1996) reported a correlation between radon levels and non-melanoma skin cancer in an ecological study in Devon and Cornwall. We are not aware of other cohort or case/control studies of radon and non-melanoma skin cancer. The evidence is thus scanty. Reporting of these malignancies is often patchy and they are difficult to study by conventional epidemiology using cancer registries. Such non-melanoma skin cancers have low lethality so their public health importance is diminished, though not eliminated.

Elevated levels of stomach cancer have been seen in survivors of the atomic bombs (UNSCEAR 2000) and in the combined cohort of miners exposed to radon (Darby *et al* 1995). However, in the latter example, there was no trend in mortality with dose and doses to stomach from radon in air are relatively low. It is also possible that, in mines, other radioactive materials cleared from the lung may be swallowed and irradiate the stomach. There is a paucity of direct information on the induction of stomach cancer by ingestion of high-radon water, though Kjellberg and Wiseman (1995) reported a correlation between stomach cancer and radon levels in an ecological study set in Pennsylvania. In the absence of more evidence, particularly from cohort or case-control studies, this hazard is certainly not established by epidemiology. However, calculations indicate that the risk is not negligible. The NRPB has endorsed a recommendation from the European Union that radon levels in private water supplies should not exceed 1000 Bq l<sup>-1</sup>.

Two thirds of the UK population obtain their water from reservoirs or rivers where radon levels are likely to be about 1 Bq l<sup>-1</sup> (Hesketh 1980). Radon levels in public water supplies are generally low (Henshaw *et al* 1993). About 50 000 (0.3%) of homes in England and Wales are estimated to have private water supplies (Jiggins 2001). Only a small proportion of these are likely to exceed the action level for radon in drinking water. However, some such supplies have been found in southwest England (BGS/DETR 2000).

## 6. Conclusions

Under normal circumstances, the largest dose from radon and its decay products will be that to the lung, delivered by the decay products rather than radon gas. This suggests that, when considering the radiological impact of radon and its decay products, the conventional focus on the risk of lung cancer from inhaled radon decay products is appropriate. Nevertheless, calculations suggest that the dose to the basal layers of the skin may also be high, with a consequent possible risk of skin cancer. Unless countermeasures are taken, a smaller number of people may also run a significant risk of stomach cancer from radon ingested in drinking water. Doses to other organs and tissues are smaller, though not necessarily negligible. The results presented here allow these risks to be quantified and compared.

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## Appendix A. Calculating doses to organs and tissues from radionuclides

It is generally not possible to measure the doses from radionuclides to organs and tissues. Instead these doses must be calculated. This is done in two stages. In the first, the number of radioactive decays in each region of the body is calculated using biokinetic models which describe where in the body the material goes and for how long it is retained. These models are described in more detail in appendix B.

In the second stage of the calculation, each region of the body in which radioactive decays take place is considered to be a source of radiation. The energy absorbed in all organs or tissues of interest, considered as 'targets', from decays in this source region is calculated. Summing the contributions from all source regions gives the total dose to the target tissues. The contribution to the dose in a target tissue per radioactive decay in the source is called the specific effective energy (SEE). In general, calculation of SEEs for penetrating radiation such as gamma rays requires Monte Carlo techniques applied to the transport of radiation through the human body, represented by a standard anthropomorphic phantom. Fortunately, the results of such calculations are available, for example in publications of or for the ICRP (ICRP 1994b, para 36).

The calculation of SEEs for alpha particles is simple if the radionuclide concerned is distributed throughout a tissue which is large compared to the range of the alpha particles (typically several tens of microns). It can then be assumed that all the energy is absorbed in the source tissue and none in any other. However, where the target tissue is small and/or the radionuclide is on its surface the situation is more complex. The calculation of doses then depends on the exact position of the sensitive target cells in relation to the radionuclide.

This more complex situation applies to most of the calculations described in this paper. The ranges of the 6.0 and 7.68 MeV alpha particles from  $^{218}\text{Po}$  and  $^{214}\text{Po}$  are about 47 and 70  $\mu\text{m}$ . Radon and decay products will rarely be homogeneously distributed in the tissue containing the sensitive cells, indeed the precise position of the sensitive cells is often variable or uncertain.

## Appendix B. Biokinetic models

In order to calculate the dose from radionuclides to organs and tissues, it is necessary to model where in the body the material goes and for how long it is retained. These characteristics are described as the biokinetics of the material in question. For convenience, it is usual to consider the intake, distribution and removal of material in stages. Generic biokinetic models describe the routes of intake, the respiratory and/or gastrointestinal tract, and another model describes the systemic behaviour of the specific material once it reaches the bloodstream. The simplest such systemic biokinetic models assign specific fractions to the organs in question, together with a simple biological retention half-time. Thus a model might specify a 6 h retention half-time in blood after which 50% went to liver with a retention time of 100 days, followed by excretion, the other 50% being retained in bone with a 200 day retention time, again followed by excretion.

More realistic, but more complex, models involve recycling in which material in blood is transferred to body organs (say, liver and bone). Material in liver is eventually transferred back to blood. A proportion is excreted, but some re-enters liver and some bone. The periods of retention in body organs in such models cannot, in general, be simply described.

Biokinetic models are formulated in terms of the behaviour of stable material. When dose calculations are carried out it is, of course, necessary to allow for the loss of material by radioactive decay.

The systemic biokinetic models used in this paper are outlined below. Note, however, that relatively slow transfers between body organs will have a small effect on patterns of dose for the short-lived decay products of radon.

### *B.1. Polonium*

The systemic model is given in ICRP Publication 67 (ICRP 1993b); it does not involve recycling. It is assumed that of polonium entering the circulation, fractions of 0.3, 0.1, 0.05, 0.1 and 0.45 are taken up by liver, kidneys, spleen, red bone marrow and all other tissues. In all cases the biological half-time is 50 days.

### *B.2. Bismuth*

The systemic model is given in ICRP Publication 30 (ICRP 1979); it does not involve recycling. It is assumed that of bismuth reaching the circulation, 30% is rapidly excreted, 40% is taken up by the kidneys and 30% is uniformly distributed throughout all other organs and tissues of the body. In all tissues, fractions of 0.6 and 0.4 are assumed to be retained with biological half-lives of 0.6 and 5 days respectively.

### *B.3. Lead*

The systemic model is given in ICRP Publication 67 (ICRP 1993b). It is based on that for the alkaline earths and does involve recycling. Uptake and retention in bone is the most important feature but the model also considers retention in soft tissues and routes of excretion. It takes account of initial uptake onto bone surfaces, transfer from surface to bone volume, bone remodelling, and recycling from bone and soft tissues to blood. Trabecular and cortical bone are considered separately and bone volume is represented as exchangeable and non-exchangeable compartments.

In order to allow for differences in retention between lead and the alkaline earths, the model is adapted to include red blood cells, to consider excretion via sweat and hair, and

to include two compartments for both the liver and kidneys. Radioactive progeny of lead produced in bone volume are assumed to follow the behaviour of the parent radionuclide until removed from the bone volume. Isotopes of bismuth and polonium produced in other body tissues by the decay of lead are assumed to have their own biokinetic behaviour.

### Appendix C. Dosimetry of the respiratory tract

Like other inhaled solid radioactive materials, radon decay products are usually attached to aerosol particles. When aerosol particles are inhaled some or all will be deposited in the different parts of the respiratory tract (i.e. the lung and the airways outside the chest up to and including the nose). Once they have been deposited they will either be cleared as discrete particles by various biological processes, such as mucus flow, or be dissolved and enter the blood stream. However, short-lived radionuclides may decay before these removal mechanisms can operate.

#### *C.1. Aerosol deposition mechanisms*

Relatively large particles are predominantly deposited by aerodynamic mechanisms (sedimentation under gravity and impaction onto surfaces). The characteristics of any particular mixture of aerosol particles are described by the activity median aerodynamic diameter (AMAD). Half of the total radioactivity contained in the aerosol is attached to particles larger than the AMAD and half to smaller ones. Relatively small aerosols are predominantly deposited by diffusion, which is a thermodynamic process. The activity median thermodynamic diameter (AMTD) is used to summarise the thermodynamic characteristics of an aerosol, just as the AMAD describes the aerodynamic behaviour. A fuller description of deposition processes can be found in ICRP Publication 66 (ICRP 1994a).

The very small particles (1 nm AMAD or 0.6 nm AMTD) considered in this paper are very mobile and the great majority (almost 90%) are deposited by diffusion in airways of the head and neck before reaching the lung. The larger particles (200 nm AMAD) are less mobile, they can penetrate deeper into the lung but most are breathed out again. Only 30% are retained in the airways, two-thirds of which are in the deep, alveolar, region.

#### *C.2. Dissolution rates and lung absorption types*

Lung absorption types are defined to divide material into three broad classes of solubility—those that are cleared from the lung by absorption to blood quickly (type F, for ‘fast’), moderately (type M) or slowly (type S). Expressed as approximate half-times for one or two components of clearance, the absorption rates for types F and M correspond to:

Type F (fast) :	10 min (100%);
Type M (moderate) :	100 min (10%); 140 days (90%).

As noted in the main text, a variant of ICRP’s model of the respiratory tract has been implemented as a computer program known as RADEP (Marsh and Birchall 2000). This is designed to explore the calculation of doses from radon decay products. RADEP does not use the default absorption types. It is flexible regarding absorption parameters, but most of the calculations which have been presented assigned the decay products a 10 h half-time for clearance to blood. This is based largely on observations of clearance of  $^{212}\text{Pb}$  (Marsh and Birchall 1999). Both the 10 h clearance half-time of RADEP and the half-times of type M are long compared to the radioactive half-times of radon decay products. Other things being equal, therefore the lung doses predicted by RADEP will be similar to those of type M.

### C.3. Respiratory tract dosimetry

The ICRP Publication 66 model of the human respiratory tract allows the calculation of doses to a number of tissues associated with the thoracic or extrathoracic airways. These are combined using 'assigned fractions' to give equivalent doses to the thoracic and extrathoracic airways,  $H_{TH}$  and  $H_{ET}$  respectively. The former attracts the lung tissue weighting factor of 0.12 in the summation for effective dose and will be called lung dose in this paper. The latter is included in the list of remainder tissues (see, for example, ICRP 1996a, section 2.5).

The tissues contributing to  $H_{TH}$  with their assigned fractions are

BB	0.333	bronchial region
bb	0.333	bronchiolar region
AI	0.333	alveolar-interstitial region
LN <sub>TH</sub>	0.001	lymphatics.

Those contributing to  $H_{ET}$  are

ET <sub>1</sub>	0.001	anterior nose
ET <sub>2</sub>	0.998	posterior nose, mouth, pharynx, larynx
LN <sub>ET</sub>	0.001	lymphatics.

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### Résumé

Cet article étudie les doses venant du radon et de ses produits de désintégration de vies courtes, dans un certain nombre d'organes et de tissus, et au fœtus. Le but est de placer toutes ces doses dans le contexte, plutôt que de ne se consacrer qu'aux distributions les plus importantes. On discute aussi, brièvement, comment l'épistémologie démontre les risques dûs à l'exposition au radon et à ses descendants. Comme on le sait bien, dans des conditions normales, le danger le plus grand se situe au niveau du tractus respiratoire, après l'inhalation des produits de désintégration du radon. Les produits de désintégration du radon peuvent aussi donner des doses appréciables à la peau. Dans certaines circonstances, il semble probable que l'ingestion de radon puisse donner des doses appréciables à l'estomac. D'autres risques apparaissent comme plus faibles; les résultats donnés ici permettent de les comparer.

### Zusammenfassung

Diese Studie untersucht die Dosen durch Radon und seinen kurzlebigen Zerfallsprodukten für eine Reihe von Organen, Gewebe und den Fötus. Ziel ist es, alle diese Dosen im Kontext zu sehen, anstatt sich auf nur die grössten Beiträge zu konzentrieren. Diese Arbeit enthält auch eine kurze Diskussion epidemiologischer Beweise über die Risiken der Bestrahlung durch Radon und seiner Zerfallsprodukte. Wie man weiss, besteht unter normalen Umständen die grösste Gefahr für die Atemwege durch Inhalation von Radon-Zerfallsprodukten. Radon-Zerfallsprodukte können auch erhebliche Dosen an die Haut abgeben. Unter gewissen Umständen erscheint es wahrscheinlich, dass mit der Nahrung aufgenommenes Radon erhebliche Dosen für den Magen ergeben könnte. Andere Risiken scheinen geringer zu sein; die hier vorgestellten Ergebnisse erlauben einen Vergleich.

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