



A method for assessing exposure of terrestrial wildlife to environmental radon (^{222}Rn) and thoron (^{220}Rn)



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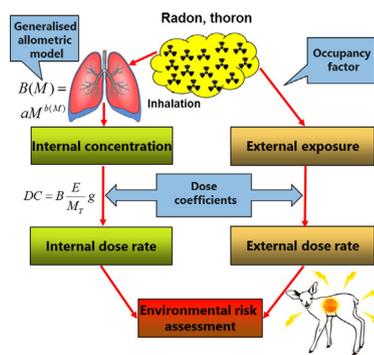
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HIGHLIGHTS

- New method for dose rates from radon, thoron and progeny to terrestrial wildlife
- Generalised allometric approach to model respiration and metabolic rates
- Calculation of doses for terrestrial ICRP reference animals and plants
- Envisaged application to study the impact of NORM radionuclides to the biosphere
- Enables future examination of radon exposure in context of ICRP DCRLs for wildlife.

GRAPHICAL ABSTRACT



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ABSTRACT

A method is presented to calculate radiation dose rates arising from radon, thoron and their progeny to non-human biota in the terrestrial environment. The method improves on existing methodologies for the assessment of radon to biota by using a generalised allometric approach to model respiration, calculating dose coefficients for the ICRP reference animals and plants, and extending the approach to cover thoron in addition to radon-derived isotopes. The method is applicable to a range of environmental situations involving these radionuclides in wildlife, with an envisaged application being to study the impact of human activities, which bring NORM radionuclides to the biosphere. Consequently, there is a need to determine whether there is an impact on non-human biota from exposure to anthropogenically enhanced radionuclides.

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1. Introduction

The radioactive isotopes $^{220,222}\text{Rn}$ appear in the environment as members of decay chains of naturally occurring ^{232}Th and ^{238}U ,

thus having the historical names “thoron” and “radon”, respectively.

Environmental radiological protection aims to ensure protection from anthropogenic sources of radiation exposure, including those naturally occurring radionuclides (NORM) that might be released into the environment due to human activity. Being of primordial origin, exposure to radon isotopes and their radioactive progeny has been generally

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regarded as a background exposure and deemed not relevant for radiation protection. However, naturally occurring radionuclides such as $^{220,222}\text{Rn}$ and their radioactive progeny can give significant exposure to terrestrial wildlife. For example, results show that absorbed dose rates to burrowing mammals as a consequence of exposure to ^{222}Rn are likely to be at least one order of magnitude higher than those suggested in previous evaluations of natural background exposure rates which had omitted this radionuclide and exposure pathway (Beresford et al., 2012). The resulting dose rates in some areas are considerably in excess of incremental no-effects benchmark dose rates that have been suggested for use in screening levels (Beresford et al., 2012).

Unlike humans, various species are known to live in soil in close proximity to radon sources. Their exposures to these naturally occurring radionuclides are often questioned. Moreover, elevated levels of radon (^{222}Rn) and thoron (^{220}Rn) can appear in the environment as a result of human activity, e.g. due to uranium mining, enrichment and processing, oil and gas production, geothermal energy and water production, among others. Elevated activity concentrations of TENORM (technologically enhanced naturally occurring materials), including radon and thoron predecessors (^{232}Th and ^{238}U), can be regarded as anthropogenic sources of radiation exposure. In such cases, human presence can be deliberately restricted or humans might not be present anyway (for example in the oceans or underground), thus no public exposure concerns could be raised for humans, but exposures to wildlife inhabiting such places can still be questioned and may need to be assessed in the context of natural preservation and protection. In other words, animals and plants inhabit places in immediate proximity to the sources of the radioactive noble gases and, for them, radon and thoron with their progenies may become (unlike for humans) potentially relevant radiologically.

Assessing doses of radiation exposure due to radon isotopes and their progeny commonly appears as a difficult task due to complicated processes of radon effluence, build-up of radioactive progeny, chemical forms and attachment to aerosols, intake, deposition and retention of radon-related radioactivity in the body of living organisms. Only a few studies in rodents consider the lung deposition of radon products using a model of the tracheobronchial tree (Harley, 1988; Hofmann et al., 2006).

Due to complexity, radon dosimetry appeared for decades as a scientific challenge. Even for humans, exposure to radon isotopes and their progeny is not covered by standard ICRP biokinetic and dosimetric models (ICRP, 2010) and, consequently, no human dose coefficients have been recommended by ICRP.

The diversity of non-human biota, expressed by their biological, morphological and metabolic differences, makes radon dosimetry for wildlife an even more complex task than that for humans. The problem is compounded by the shortage of studies dealing specifically with methodologies for the calculation of radon and thoron doses to wildlife (most investigations are orientated to human dosimetry or use laboratory animals as a surrogate for human exposures).

Laboratory rats particularly are used in inhalation studies as a surrogate for human exposures and dosimetry models for inhaled radon progeny in the rat lung have been developed, with the objective of predicting bronchial dose distributions (Harley, 1988; Hofmann et al., 2006; Strong and Baker, 1996; Winkler-Heil et al., 2015). These models are quite complex, involving a full model of the tracheobronchial tree and associated lung deposition, redistribution within the airways and clearance processes for radon and thoron daughters. Such approaches are by necessity biological species-specific and require a number of parameters that are not available except for the laboratory animals studied. As such, they go beyond the need for a practical assessment tool useable for radiological screening purposes, which has to be sufficiently generic to cover a variety of terrestrial animals and must have an in-built level of conservatism (approximately one order of magnitude) in order to be adequately robust.

Although the use of simplified and conservative methods for non-human biota appears as rational and appropriate, there are very few methods for radon already being in use (MacDonald and Laverock, 1998; Vives i Batlle et al., 2008). To our knowledge, no method has been published to calculate doses to non-human biota as a result of exposure to thoron, but a method has been developed for ^{41}Ar , $^{85,88}\text{Kr}$ and $^{131\text{m},133}\text{Xe}$ wildlife dose assessment (Vives i Batlle et al., 2015).

The radon approach by MacDonald and Laverock (1998) was designed for burrowing mammals, although the equations have been adapted to calculate radiation doses for birds (Kitowski et al., 2015). The method by Vives i Batlle et al. (2008), which has the advantage of having a wider range of application for different terrestrial animal and plant species, was initially developed in response to a need by the England and Wales Environment Agency to improve on an earlier interim approach, so as to conduct a trial assessment with set ^{222}Rn authorisation limits under the UK Radioactive Substances Act (RSA) 1993. The approach, which was later applied in a uranium mining context (Kryshev et al., 2013) was further developed as a dose assessment screening tool (Vives i Batlle et al., 2012), though it is as yet to be integrated into the ERICA tool for radiological impact to non-human biota. It was subsequently used in a study to derive exposures of burrowing mammals to ^{222}Rn (Beresford et al., 2012), becoming the initial basis of the more detailed and widely applicable methodology presented here.

This article deals with the issue of radon, thoron and progeny to non-human biota, providing a bespoke allometric method to calculate dose rates to terrestrial wildlife. We have recalculated the potential α -energy concentration (PAEC) for radon and thoron, following the dosimetric approach adopted by ICRP and using the contemporary radionuclide emission data also recommended by ICRP (ICRP, 2008b). Then, we deliver tables with conservative (assuming full retention) estimates of dose coefficients (DCs) for non-human entities due to radon, thoron and their progeny, covering both internal and external exposure situations. The presented DCs illustrate the importance of having an appropriate definition of a critical organ (part of the body) for internal exposure to radionuclides emitting non-penetrating radiation (α -particles) and show that an implausible choice of the critical organ or tissue may lead to growth of uncertainty by several orders of magnitude.

2. Materials and methods

2.1. Main dosimetric properties of radon and thoron progeny

The dosimetry of radon (^{222}Rn) and its daughter products is a widely considered topic, having been the object of numerous ICRP publications (ICRP, 1987; ICRP, 1993; ICRP, 2010; ICRP, 2014b). Thoron (^{220}Rn), due to its shorter half-life, is usually neglected in assessments of human indoor exposure, because of significant decay during transport from the point of origin to human dwellings. However, assessment of radiation exposure of animals and plants living in direct proximity to sources of radon gas may require accounting for contributions of radon as well as of shorter-lived thoron.

Figs. 1 and 2 give the energies of α -particles emitted by radon isotopes and their progeny accounted via the ratio of transient activities of daughter nuclides to that of the parent. From the figures, the total α -energy emitted per single decay of the parent nuclide can be seen to vary within a factor of two (^{220}Rn) or three (^{222}Rn) for non-equilibrated mixtures of decay chain members. The value of the equilibrium factor $F = 0.4$ shown in the figures is commonly assumed in human dosimetry (Keller et al., 1984; Wenbin et al., 1990; Wrixon et al., 1988) and thus can be used as a plausible default value for exposure of terrestrial wildlife in the outdoor environment when experimentally-based information is missing. Consequently, $F = 1$ can be regarded as a conservative value. However, as seen from Fig. 2, Thoron is a short-lived nuclide and, after reaching equilibrium with its daughter ^{216}Po in about 10 min, decays significantly, resulting in a highly non-equilibrium state with other progeny (^{212}Pb , ^{212}Bi , ^{212}Po , and

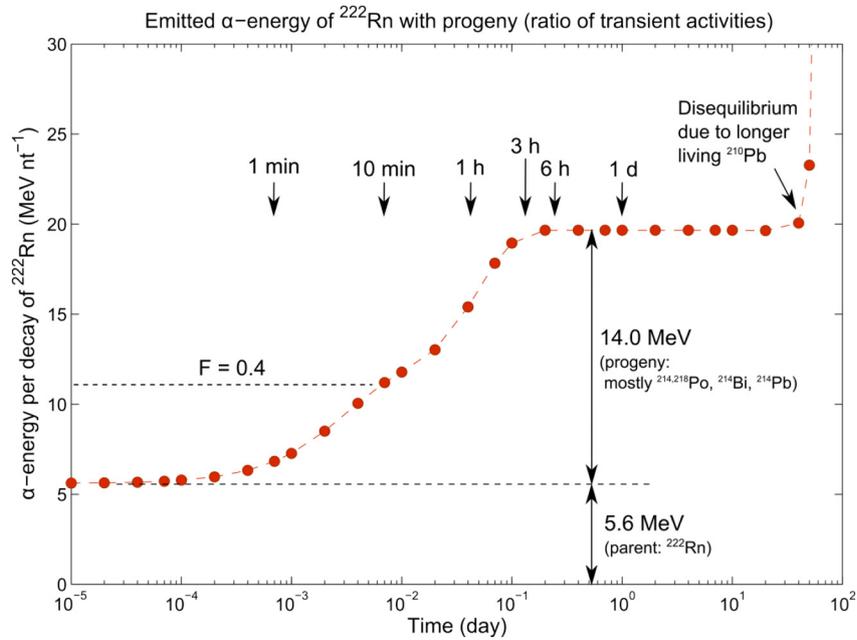


Fig. 1. Energy of α -particles emitted by radon (^{222}Rn) and its progeny per decay of the parent nuclide.

^{208}Tl). Thus, estimates of biota exposure in this case may appear more realistic with an equilibrium factor equal to one.

As seen in Figs. 1 and 2, the use of transient activity ratios in expressing the total energy emitted by radioactive parent and daughter nuclides may be inconvenient for radon and simply impractical for thoron. For these decay chains, a ratio of time-integrated activities appears as a practical alternative compatible with the concept of potential α -energy, which is common for radon dosimetry (ICRP, 1993; Porstendörfer, 1994). However, computing time-integrated activity ratios requires assessment-specific data: exposure time and location factors. In this paper, a generic approach is presented, which conservatively assumes conditions of full equilibrium between the

parent and the daughters. Although convenient and plausible in many practical situations, this generic assumption of equilibrium might however become invalid for certain assessment-specific time and location conditions.

2.2. Assessment of internal exposures

2.2.1. Simplified representation of radon respiration

For terrestrial animals and plants, the main pathway of internal exposure to radon and its progeny is respiration. The approach described here assumes conservatively full deposition and absorption of activity in respired air. Consequently, the DCs derived in the present study

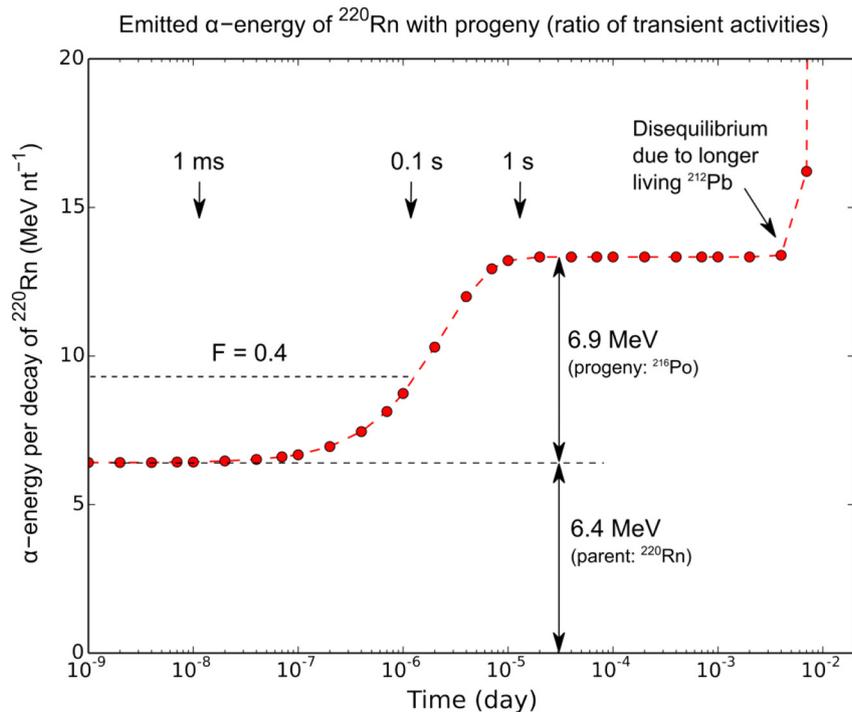


Fig. 2. Energy of α -particles emitted by thoron (^{220}Rn) and its progeny per decay of the parent nuclide.

indicate upper bounds of radiation exposure due to environmental radon isotopes and their progeny. DCs for internal radon exposure in the present study are formulated as aggregated quantities, namely, per activity concentration in the ambient air, thus aggregating dose per unit activity in the body and concentration ratio between activity in the body and in the ambient air. This makes them different from the internal DCC definition adopted in the ICRP dosimetry framework for non-human biota (ICRP, 2008a), which are formulated in terms of dose rate per unit activity concentration in the body or organ. In other words, our formulation is complementary to the ICRP approach, and can be regarded as conservative in the sense that it can eliminate from consideration situations of low radiological relevance by calculating doses that may be safely said to not have been exceeded. This can be regarded as a more practical and convenient alternative in certain exposure situations.

Respiration of radon and its progeny is modelled as constant flow into the relevant respiratory system, conservatively assuming that radon gas is in equilibrium with its daughters (equilibrium factor $F = 1$), thus helping to avoid an underestimation of the dose due to a variety of environmental conditions which may not be fully known at the time of the assessment. The degree of conservatism incurred by this equilibrium assumption does not typically exceed as factor of 2 or 3, because environmental measurements usually show the annual mean value of F in open air to be 0.4 (Keller et al., 1984), 0.51 ± 0.12 (Kojima, 1996) or 0.6 (Wenbin et al., 1990). Elsewhere (Porstendörfer, 1994), a range 0.4–0.8 at 1.5 m above ground was also reported. Other authors (Beresford et al., 2012) applied an equilibrium factor for outdoor air of $F = 0.8$.

Full absorption of progeny is assumed within the respiratory organs/systems, and no further redistribution of the deposited activity due to biokinetic processes, exhalation or excretion from the organism, is accounted for. Parent radon isotopes are chemically inert gases and they are assumed to escape without significantly contributing to the total internal dose. This is because, although it can be present in physical solution, chiefly in the body water and fat, radon has a small solubility in water and body fluids and, being chemically inert, it does not participate at normal pressures in biochemical reactions of the human body (Tobias et al., 1949).

Under the above assumptions, the following equation applies to the conversion coefficient, DC ($\mu\text{Gy h}^{-1} \text{Bq}^{-1} \text{m}^3$), which is defined as absorbed dose rate in target tissues due to radon progeny per unit activity concentration of parent isotope (^{220}Rn or ^{222}Rn) in ambient air:

$$DC = B \frac{E}{M_T} g \quad (1)$$

where B is the respiration (breathing) rate ($\text{m}^3 \text{h}^{-1}$), E is the total energy absorbed in the target tissues due to radiation emitted by the radon progeny until decay to (quasi)stable lead isotopes ($\mu\text{J Bq}^{-1}$), M_T is the mass of the target tissue/organ (kg), and g is the geometrical factor which takes into account (in)homogeneity of activity deposition in airways/respiratory organs (dimensionless).

Alpha-particles contribute about 95% to the total emitted energy of radon progeny (ICRP, 2008b) and this energy can be represented using concept of the potential α -energy (ICRP, 1987; ICRP, 1993), thus assuming respiration of equilibrium mixture of radon daughters and neglecting the contribution of electrons and photons to the absorbed dose. The reason for neglecting the electron and photon contributions is not only the fact that they carry 5% or less of the emitted energy, but also that they are more penetrating radiation types and their absorbed fractions in the tissue of interest can be significantly less than one, depending on the organism's anatomy or morphology. Updated values of the potential α -energy, based on data from the ICRP Publication 107 (ICRP, 2008b), are shown in Table 1, which is functionally similar to the previously published Table A1 in ICRP Publication 50 (ICRP, 1987) and Table 2 in Publication 65 (ICRP, 1993).

Table 1
Potential α -energy for radon (^{222}Rn) and thoron (^{220}Rn) progeny calculated using emission data from ICRP Publication 107 (ICRP, 2008b).

Radionuclide	Half-life	Potential α energy			
		Per atom		per unit of activity	
		(MeV)	(10^{-12} J)	(MeV Bq^{-1})	(10^{-10} J Bq^{-1})
Radon (^{222}Rn) progeny					
^{218}Po	3.10 min	13.95	2.23	3743	6.0
^{214}Pb	26.8 min	7.84	1.26	18,176	29.1
^{214}Bi	19.9 min	7.84	1.26	13,496	21.6
^{214}Po	164.2 μs	7.84	1.26	1.9×10^{-3}	3.0×10^{-6}
Total (at equilibrium), per Bq of radon				35,415	56.74
Thoron (^{220}Rn) progeny					
^{216}Po	0.145 s	15.86	2.54	3,318	5.3×10^{-3}
^{212}Pb	10.64 h	8.95	1.43	494,807	792.7
^{212}Bi	60.55 min	8.95	1.43	46,931	75.2
^{212}Po	3.0×10^{-7} s	8.95	1.43	2.5×10^{-6}	4.0×10^{-9}
Total (at equilibrium), per Bq of thoron				542,047	870.73

Due to short range of α -particles in tissue, they can be regarded as non-penetrating and fully depositing their energy in the tissue. In other words, absorbed fractions for α -particles are assumed equal to one ($AF_{\alpha} \approx 1$) and the total α -energy released in decay of radon progeny is assumed to be absorbed internally. The geometric factor g accounts for the heterogeneity of the airways of some organisms, which might result in energy deposition not in the living tissues but in internal air or in mucous or other inert biological fluids. Simple reasoning leads to the conclusion that the geometrical factor may vary from 0.5 (α -emitters on interface) to 1.0 (α -emitters deep in tissue).

The target tissue exposed by the radon progeny varies significantly depending on the physico-chemical properties of inhaled radon and its radioactive progeny, as well as on the biological variety of breathing organisms. For most types of organisms though, since the internal dose rate is predominantly due to α -radiation, the lungs will receive virtually the entire internal dose rate. A convenient assumption is that the sensitive tissues of the respiratory system have a cylindrical shape, since they consist of the epithelium surrounding the walls of the airways, as is the case for humans (Hofmann and Winkler-Heil, 2015; ICRP, 1994; ICRP, 2002). The most significant difference between human and rat lungs, in fact, is the branching structure of the bronchial tree, which is relatively symmetric in humans, but monopodial in rats (Winkler-Heil et al., 2015). Thus, assuming that the shape of the airways is a cylinder with

Table 2
Parameters for calculation and values of aggregated unweighted DCs for internal exposure of animals due to progeny of radon isotopes $^{220,222}\text{Rn}$.

Parameter or quantity	Amphibian (ICRP frog) ^a	Reptile (ERICA snake) ^a	Mammal small (ICRP rat)	Mammal big (ICRP deer)	Bird (ICRP duck) ^a
M (kg)	0.0314	0.744	0.314	245	1.26
a (m)	0.08	1.2	0.2	1.3	0.3
b (m)	0.03	0.035	0.06	0.6	0.1
c (m)	0.025	0.035	0.05	0.6	0.08
B ($\text{m}^3 \text{h}^{-1}$)	2.1×10^{-3}	0.023	0.012	2.5	0.034
DCs per air concentration of ^{222}Rn ($\mu\text{Gy h}^{-1} \text{Bq}^{-1} \text{m}^3$)					
DC_B	1.4	1.8	1.7	4.2	1.9
DC_{TB}	0.15	0.20	0.18	0.46	0.21
DC_L	0.032	0.014	0.017	4.1×10^{-3}	0.012
DC_{WB}	3.8×10^{-4}	1.7×10^{-4}	2.1×10^{-4}	5.8×10^{-5}	1.5×10^{-4}
DCs per air concentration of ^{220}Rn ($\mu\text{Gy h}^{-1} \text{Bq}^{-1} \text{m}^3$)					
DC_B	22	28	26	65	30
DC_{TB}	2.4	3.0	2.8	7.0	3.2
DC_L	0.49	0.21	0.26	0.062	0.18
DC_{WB}	5.9×10^{-3}	2.6×10^{-3}	3.2×10^{-3}	8.9×10^{-4}	2.4×10^{-3}

^a DC for non-mammals are shown for illustrative purposes only.

radius R_{aw} and accounting for the small thickness h_T of the sensitive tissue ($h_T \ll R_{aw}$), we can express the mass of target tissues M_T as:

$$M_T = \rho_T S_T R_{aw} \left(\frac{h_T}{R_{aw}} + \frac{h_T^2}{2R_{aw}^2} \right) \approx \rho_T S_T h_T \quad (2)$$

where ρ_T is the density of the target tissue taken equal to 10^3 kg m^{-3} , and S_T is the tracheobronchial surface area (m^2).

The active depth of sensitive tissue, i.e. the thickness of the bronchial epithelium (without cilia), is assumed conservatively (for lack of species-specific information) to be $55 \mu\text{m}$ as for the ICRP human respiratory tract model (ICRP, 1994).

2.2.2. Allometric scaling of respiration parameters for animals

The respiratory tract properties, including breathing rate, vary among organisms because of their biological and morphological diversity. Despite this variability, there exist structural similarities between related organisms; these similarities are expressed by so-called allometric relationships or 'laws' (Kleiber, 1947; Rubner, 1883). Allometric scaling can be used to assess organism-specific parameters. For example, in mammals, the breathing rate has been found to correlate with body mass M according to the following allometric equation:

$$B(M) = a M^b \quad (3)$$

where a and b are the base and exponent, the latter being close to $3/4$, according to Kleiber's allometric scaling law (Kleiber, 1932; Kleiber, 1947). For example, cardiac output and pulmonary exchange scale as $M^{3/4}$ in mammals (Schmidt-Nielsen, 1984), and similarly for the rate of respiratory ventilation (West and Brown, 2005). However, the above equation is an approximation, and experimental data suggest that the relationship between mass and metabolic rate has convex curvature on a logarithmic scale (Kolokotronis et al., 2010). This means that extrapolating the breathing rate as a function of body mass using Eq. (3) from either small or large masses will result in an underestimation at the opposite end of the mass range.

This problem can be rectified by using generalised allometric equations (ICRP, In press; Ulanovsky, 2016). For example, for the breathing rate of terrestrial mammals, the generalised allometric equation is as follows:

$$B(M) = a^* M^{b^*} = e^{\beta_0} M^{1+\beta_1+\beta_2 \ln M}, \quad (4)$$

where B is the ventilation rate ($\text{m}^3 \text{ h}^{-1}$) and M is the mass of the organism (kg).

From the compilation of Bide et al. (2000) on ventilation rate for terrestrial mammals, the following values have been found statistically significant (Ulanovsky, 2016): $\beta_0 = -3.562 \pm 0.050$, $\beta_1 = -0.226 \pm 0.019$ and $\beta_2 = (7.26 \pm 4.45) \times 10^{-3}$. Note that neglecting the log-quadratic term in exponent reduces Eq. (4) to:

$$B(M) = e^{\beta_0} M^{1+\beta_1},$$

which is simply the 'Kleiber law' with an exponent of 0.77 instead of 0.75. In this sense, Eq. (4) can be called a generalisation of the first-order allometric equation.

Using the respiratory tract parameters of the ICRP 'Reference Man' (ICRP, 2002) and applying allometric scaling, the DCs for internal exposure of animals (terrestrial mammals) can be expressed as simple allometric power functions for a range of target tissues such as, for example, the bronchial epithelium (B), tracheobronchial tree (TB), full lung (L) and whole body (WB):

$$DC_B = \frac{E}{\rho_T h_T S_B^{RM}} \left(\frac{M_{RM}}{M} \right)^{\frac{2}{3}} B(M) \quad (5)$$

$$DC_{TB} = \frac{E}{\rho_T h_T S_{TB}^{RM}} \left(\frac{M_{RM}}{M} \right)^{\frac{2}{3}} B(M) \quad (6)$$

$$DC_L = \frac{E}{a_L M^{b_L}} B(M) \quad (7)$$

$$DC_{WB} = \frac{E}{M} B(M) \quad (8)$$

where a_L and b_L are the base and exponent of the allometric formula for lung mass (kg) as a function of whole body mass (kg), namely 0.0128 and 1.02 (Vives i Batlle et al., 2012), $S_{TB}^{RM} = 0.269 \text{ (m}^2\text{)}$ and $S_B^{RM} = 0.0291 \text{ (m}^2\text{)}$ are the surface area of the tracheobronchial tree and the bronchial epithelium of the ICRP Reference Man, and $M_{RM} = 70 \text{ kg}$ is the mass of the ICRP Reference Man.

The above approach is derived for terrestrial mammals. Thus, there is no guarantee that respiration rates of other lung-breathing ICRP reference organisms such as birds, reptiles and amphibians still follow Eq. (4) for the breathing rate or the other allometric relationships for respiratory system implicitly present in Eqs. (6)–(8). As a practical solution, Vives i Batlle et al. (2012) have suggested that the allometric approach for mammals could be used conjecturally for organisms having structurally simpler breathing systems if no other option is available, and that this is likely to give conservative estimates for these organisms.

2.2.3. Internal exposure of plants

For plants, a simple conservative approximation is used, whereby the whole surface area of the plant is assumed to be exchanging gases with the atmosphere and the following approximations for DCs of plant tissue (DC_S) and whole plant (DC_P) have been suggested (Vives i Batlle et al., 2012):

$$DC_S = E \frac{a_{PL} a M^{b_{PL}-1}}{2\sqrt{6}h_T} \quad (9)$$

$$DC_P = E a_{PL} M^{b_{PL}-1}$$

where $a_{PL} = 1.95 \times 10^{-4} \text{ (m}^3 \text{ s}^{-1}\text{)}$ is the allometric base for respiration rate in plants calculated based on net CO_2 efflux data by Vives i Batlle et al. (2012) and previous data (Reich et al., 2005), and b_{PL} is the exponent of that allometric breathing rate, which for plants is calculated to be very close to unity at 1.02 (Vives i Batlle et al., 2012) so that Eq. (9) is virtually mass-independent. Moreover, a is the minor axis or average of non-equal minor axes of the ellipsoid representing the plant (m), h_T is the depth of sensitive tissue, which is based on the morphology of plant cells and the range of α -particles in plant tissue, whereupon the representative value of the depth of sensitive tissue can be taken conservatively to be $50 \mu\text{m}$.

It should be noted also that due to the important role of carbon dioxide in the metabolism of living species, the allometric approximation for the plant respiration rate in Eq. 9 may lead to additional conservatism of the aggregated DCs.

Due to its simplicity, the above approximation has been tested against a more complex dynamic model that considers interception of the unattached and attached fractions of the airborne radon daughters by plant stomata, diffusion of radon gas through stomata, permeation through the plant's epidermis and uptake of deposited activity to the plant interior (Vives i Batlle et al., 2011). This more sophisticated approach can calculate separately the dose contributions arising from radioactive materials deposited internally, externally and on the plant surface.

Results of this comparison are given in Table 8 of Vives i Batlle et al. (2011). The total (internal plus surface-deposition) dose rates for the present methodology are 18% lower than calculated by the plant dynamic model, which is reasonably consistent. External dose rates for the current approach are 1.9 times higher than the plant model, which

is not surprising, given that we adopted an equilibrium factor of 1, whereas the dynamic model generates an equilibrium factor for outdoor air of about 0.5.

2.3. Assessment of external exposures

2.3.1. Absorbed fractions and DC approach for animals and plants

External exposure of terrestrial animals and plants to radon isotopes and their progeny may occur in various locations: in soil, on the ground surface and in the air above. Due to the short range of α -particles even in air, external exposure to radon isotopes and their progeny is mainly created by photons and electrons emitted by ambient radioactive sources.

Under the assumptions of a uniform isotropic model, external exposure can be considered as complementary to internal and, consequently, it can be expressed via absorbed fractions for specific radiation types and for the given shapes of the body (Ulanovsky and Prohl, 2012).

The external dose assessment methodology adopted here allows expressing the DC for external exposure of terrestrial animals in soil and on the surface to sources distributed in soil, as well as for organisms above the ground interface exposed to sources in soil or in air. Being flexible and versatile, this approach is based on the dataset calculated by Monte Carlo technique for a set of pre-defined shapes corresponding to FASSET/ERICA organisms (Taranenko et al., 2004) and for tissue-equivalent spheres (Ulanovsky, 2014) for terrestrial organisms on and above ground surface exposed to radioactive sources in soil or in air. The DC for external exposure of terrestrial organisms can be interpolated for arbitrary masses and heights above ground, though obviating the effects of shape.

An alternative analytical parameterisation had been suggested based on a set of absorbed fractions for pre-defined set of shapes representing various aquatic and terrestrial animals (Vives i Batlle et al., 2004). Absorbed fractions for these shapes have been calculated using Monte Carlo integration of point kernels for photons and electrons (Berger, 1968; Berger, 1971). Consequently, these approximations for absorbed fractions have been applied to compute DCs for terrestrial animals exposed to radon and progeny isotopes in air. This approach accounted for the short-lived progeny of ^{222}Rn included ^{218}Po , ^{218}At , ^{214}Pb , ^{214}Bi and ^{214}Po . Longer-living, quasi-stable ^{210}Pb and its progeny ^{210}Bi and ^{210}Po have been ignored. Radiations emitted by the considered nuclides encountered 93 electron-, 75 gamma- and six α -lines, for which values for the decay energy or mean energy and the related quantum yields were taken from the ICRP Publication 38 (ICRP, 1983).

External exposure to α -particles is commonly ignored because of their short range and the shielding properties of tissue layers (e.g. fur, feather or dead skin) which cover the bodies of organisms. The contributions of low-energy ($E < 10$ keV) electrons and photons sources to the external DC have been found negligible in comparison with electrons and photons of higher energy.

3. Results and discussion

3.1. Dose coefficients for internal exposure

Calculated radon and thoron DCs for some ICRP Reference Animals and Plants (RAP) are given in Tables 2 and 3. The calculation of internal absorbed dose can be carried out simply by multiplying the listed DCs by the parent radon activity concentration in ambient air. A linear correction factor can be applied if an equilibrium factor different from unity is required.

Alpha-particles belong to class of densely ionising high-LET radiations. Consequently, an assessment of the radiobiological effects of exposure to radon might require weighting internal doses with an appropriate radiation weighting factor for α -particles (W_{α}). For human radiological protection, ICRP recommends using a value of 20 for the W_{α} (ICRP, 2007), whilst for non-human biota, where protection

Table 3

Parameters for calculation and values of aggregated unweighted DCs for internal exposure of plants due to progeny of radon isotopes $^{220,222}\text{Rn}$.

Parameter or quantity	Lichen & bryophytes (ICRP bryophyte)	Grasses and herbs (ICRP wild grass)	Trees (ICRP pine tree)
M (kg)	1.1×10^{-4}	2.6×10^{-3}	471
a (m)	0.04	0.05	10
b (m)	2.3×10^{-3}	0.01	0.3
c (m)	2.3×10^{-3}	0.01	0.3
B ($\text{m}^3 \text{h}^{-1}$)	6.5×10^{-5}	1.6×10^{-3}	360
DCs per air concentration of ^{222}Rn ($\mu\text{Gy h}^{-1} \text{Bq}^{-1} \text{m}^3$)			
DC_{SS}	0.031	0.14	5.5
DC_{WB}	3.3×10^{-3}	3.5×10^{-3}	4.5×10^{-3}
DCs per air concentration of ^{220}Rn ($\mu\text{Gy h}^{-1} \text{Bq}^{-1} \text{m}^3$)			
DC_S	0.48	2.2	85
DC_P	0.051	0.054	0.069

of a species is aimed at the population level and radiation weighting factors need to be formulated for biological endpoints that could “lead to changes in population size or structure” (ICRP, 2014a), there is no recommended value yet. Although a degree of consensus around a value of $W_{\alpha} = 10$ has emerged (Brown et al., 2008; Vives i Batlle et al., 2004), the DCs presented in this article are left un-weighted to avoid loss of generality.

The present approach is compatible with the earlier method of MacDonald and Laverock (1998), except that these authors considered the whole lung as reference tissue for dose calculation. For non-penetrating α -particles, a simple re-scaling procedure can be used to compare the dose for different reference tissues to the dose to a whole lung as calculated by MacDonald and Laverock (1998). Previous comparison (Vives i Batlle et al., 2012) showed the compatibility of the dose rates obtained by both methods.

As seen in Table 2, DCs for animals vary within four orders of magnitude between compartments of respiratory tract and the whole body. In real exposure situations, air contains the radioactive progeny in gaseous form and attached to aerosols and dust. Depending on the size of aerosol particles and their chemical form, deposition and further absorption of radioactive substances in airways may vary significantly, thus leading to various patterns of activity distribution between different parts of respiratory system and other organs. Due to this, it appears plausible to assume that more realistic dose estimates can be achieved by assuming fractional deposition in various compartments and, consequently, by computing the total internal dose as the weighted sum of partial doses in the compartments.

As previously stated, the DCs for amphibians, birds and reptiles are to be used conjecturally because the respiratory systems of these animals are not only dimensionally but also structurally different. DC values are given here for illustrative purposes and are not guaranteed for use in assessments until allometric modelling of the respiration rates for these organisms is established on a sounder basis.

The DCs shown above are given for various target tissues, while the whole body dose is the quantity most often used in assessments of environmental risk, including previous radon studies (Beresford et al., 2012; Vives i Batlle et al., 2008). This is due to scarcity of data on radiation effects in wild animals and plants with which the predicted dose rates to target tissues could be interpreted. The dose-rate benchmarks used by ICRP are based primarily on whole-body exposures (ICRP, 2008a).

3.2. Calculated external DC values

The DCs for the ICRP RAPs in the terrestrial environment exposed to external sources of radon and thoron isotopes and their progenies in the ambient air are given in Table 4. The data shown in the table come from two independent methods. The first method (Vives i Batlle et al., 2012) under assumptions of uniform isotropic model computes DCs for

Table 4

Comparison of the DC for animals and plants externally exposed to radon and thoron (^{220,222}Rn) and their progeny in ambient air.

Organism	DC (μGy h ⁻¹ Bq ⁻¹ m ³)			
	In infinite air ^a	In air ^b (h = 500 m)	In air ^b (h = 10 m)	On the ground ^b
Radon (²²²Rn) and progeny				
Amphibian (ICRP frog)	7.8 × 10 ⁻⁴	7.5 × 10 ⁻⁴	4.4 × 10 ⁻⁴	4.1 × 10 ⁻⁴
Reptile (FASSET snake)	7.6 × 10 ⁻⁴	7.5 × 10 ⁻⁴	4.4 × 10 ⁻⁴	4.1 × 10 ⁻⁴
Mammal (ICRP rat)	7.3 × 10 ⁻⁴	7.6 × 10 ⁻⁴	4.5 × 10 ⁻⁴	4.1 × 10 ⁻⁴
Mammal (ICRP deer)	3.8 × 10 ⁻⁴	5.1 × 10 ⁻⁴	3.0 × 10 ⁻⁴	2.8 × 10 ⁻⁴
Bird (ICRP duck)	6.9 × 10 ⁻⁴	7.5 × 10 ⁻⁴	4.4 × 10 ⁻⁴	4.1 × 10 ⁻⁴
Lichen and bryophytes (ICRP bryophytes)	9.9 × 10 ⁻⁴	6.0 × 10 ⁻⁴	3.5 × 10 ⁻⁴	3.3 × 10 ⁻⁴
Grasses and herbs (ICRP wild grass)	8.5 × 10 ⁻⁴	7.2 × 10 ⁻⁴	4.2 × 10 ⁻⁴	3.9 × 10 ⁻⁴
Tree (ICRP pine tree)	5.1 × 10 ⁻⁴	4.5 × 10 ⁻⁴	2.7 × 10 ⁻⁴	2.5 × 10 ⁻⁴
Thoron (²²⁰Rn) and progeny				
Amphibian (ICRP frog)	n.a.	6.7 × 10 ⁻⁴	4.0 × 10 ⁻⁴	3.8 × 10 ⁻⁴
Reptile (FASSET snake)	n.a.	6.9 × 10 ⁻⁴	4.1 × 10 ⁻⁴	3.9 × 10 ⁻⁴
Mammal (ICRP rat)	n.a.	6.9 × 10 ⁻⁴	4.2 × 10 ⁻⁴	3.9 × 10 ⁻⁴
Mammal (ICRP deer)	n.a.	4.9 × 10 ⁻⁴	3.0 × 10 ⁻⁴	2.8 × 10 ⁻⁴
Bird (ICRP duck)	n.a.	6.9 × 10 ⁻⁴	4.1 × 10 ⁻⁴	3.9 × 10 ⁻⁴
Lichen and bryophytes (ICRP bryophytes)	n.a.	4.5 × 10 ⁻⁴	2.7 × 10 ⁻⁴	2.5 × 10 ⁻⁴
Grasses and herbs (ICRP wild grass)	n.a.	6.0 × 10 ⁻⁴	3.6 × 10 ⁻⁴	3.5 × 10 ⁻⁴
Tree (ICRP pine tree)	n.a.	4.4 × 10 ⁻⁴	2.7 × 10 ⁻⁴	2.5 × 10 ⁻⁴

^a Uniform isotropic model method, using absorbed fractions based on Monte Carlo integration of photon and electron point kernels (Vives i Batlle et al., 2012).

^b Absorbed doses in tissue-equivalent spheres exposed to photon-only sources in air (Ulanovsky, 2014).

external exposure as complementary fractions to the full absorption limit. An analytical approximation, based on Monte-Carlo-integrated point kernels of various radiation in an infinite medium, is used for computation of absorbed fractions for photons and electrons. Further re-scaling of the computed DC, using density of air at normal conditions, allowed expressing the DC as per unit volume activity concentrations in air.

The second method (Ulanovsky, 2014) uses differential air kerma above infinite terrain due to radioactive sources in ambient air, calculated by a Monte Carlo method. Absorbed doses for living species have been derived from the differential air kerma using a dose-per-kerma conversion function, which is interpolated using data pre-computed by an analogue Monte Carlo method for tissue-equivalent spheres in isotropic monoenergetic photon fields. The results obtained with this method are provided for both radioactive radon isotopes (^{220,222}Rn) and their progeny.

The method based on the uniform isotropic model has been compared with the external DC for in-soil exposure to radon and progeny using a DC calculation facility (Ulanovsky et al., 2008) largely compatible to that available in the ERICA assessment tool (Brown et al., 2016; Brown et al., 2008). The comparison was satisfactory, with relative differences ranging from 3 to 10% in animals and 3 to 25% in plants. These differences are attributable to differences in the way the absorbed fractions are calculated by the two methods.

The comparison of the DC for animals and plants externally exposed to radon isotopes given in Table 4 demonstrates (a) good compatibility regardless of different methods and data used in their computations, (b) low inter-species variability of the external DC, and (c) variability of the DC due to change of exposure source from infinite to semi-space, predictably limited within a (geometrical) factor two. The low variability of the presented DC due to organism size and irradiation geometry implies that the effect of transient activities in the radon and thoron decay chains may become considerably stronger and more influential to dose estimates. As the DCs in Table 3 are computed assuming equilibrium conditions in the decay chains, they can be regarded as conservative estimates of the respective DCs resulting in non-equilibrated mixtures of radon isotopes and their progeny.

3.3. External dose calculation

Assessment of external exposures of terrestrial biota to environmental radon and its progeny should consider the mobility of the radioactive

gases and aerosols, which results in the existence of various configurations of radioactive sources and biological targets. The variability of habitats and life styles of biota also contribute to the variability of possible exposure scenarios. The universal method to cope with this diversity is to apply the superposition principle, which means that the dosimetric response to a complicated (realistic) exposure scenario can be characterised as a weighted sum of responses to simple basic exposure situations, for which the DCs are already known or can be easily derived. Weighting of a basic scenario is expressed via so-called ‘occupancy factor’, which is constructed to express: (a) the time-share spent by the organism in locations described by the basic ‘source-target’ configurations (e.g. in soil, on the ground surface, in air), and (b) the relative contribution of radiation sources affecting the organism at the specified location.

An example of applying the occupancy factors in an external dose assessment can be given for the situation where the organism is exposed to radiation arising from (a) radon present in the air-filled soil pores (e.g. in burrows) and (b) direct immersion in the atmosphere with radon and its progeny. Both components of the external dose can be represented by the following equations:

$$\begin{aligned}
 D_S &= DC_{ext} \frac{C_{Rn}^s}{CF} F(f_S + 0.5f_{SS} + r_f f_A) \\
 D_I &= DC_{ext} C_{Rn}^a F(f_A + 0.5f_{SS})
 \end{aligned}
 \tag{10}$$

where D_S and D_I (μGy h⁻¹) represent the dose rates from radon in the air-filled soil pores and direct immersion in the atmosphere, respectively; C_{Rn}^s (Bq kg⁻¹) is the concentration of ²²²Rn in soil, C_{Rn}^a (Bq m⁻³) is the concentration of ²²²Rn in atmospheric air, CF (m³ kg⁻¹) is the factor used to convert volume concentration of radon in the air of the soil pores to mass concentration of radon in soil, accounting for soil porosity, DC_{ext} (μGy h⁻¹ Bq⁻¹ m³) is the DC, f_S, f_{SS} and f_A (dimensionless) represent the occupancy factors for three exposure situations: below ground in soil, on the soil surface and immersion in air above the ground, and F is the equilibrium factor (if a value different from 1 is used). A dimensionless radiation-dependent reduction factor r_f is introduced to modify the dose for organisms in above-ground air as received from radiation sources in soil. It is zero for α-particles and low-energy electrons and approximately 0.25 for higher energy electrons and photons.

It is not possible to give a CF value for all soils of different characteristics under varying moisture conditions. By way of example, an indicative value for the CF of 10⁻⁴ m³ kg⁻¹ can be obtained by assuming that radon in pore air is at the same concentration as ground level air

concentrations. This can be calculated as follows: The effective porosity of soil typically varies within the ranges 0.01–0.18 for clay and 0.16–0.46 for medium sand (McWorter and Sunada, 1988). In wet soil, a portion of the available pore space will be occupied by water. An assumption is made for free air space of 0.15 by volume. Assuming also a bulk density for soil of 1500 kg m^{-3} , the free air space would be $0.15/1500$ or $10^{-4} \text{ m}^3 \text{ kg}^{-1}$. Thus, this value can be used as a conversion factor between activity concentration in air (Bq m^{-3}) and in wet soil (Bq kg^{-1}).

Occupancy factors can be set as, for example, default values in the ERICA assessment tool (Brown et al., 2016; Brown et al., 2008), which for terrestrial animals assumes 100% occupancy on the soil surface except for rat which is considered to have 100% occupancy inside the soil.

3.4. Uncertainties in dose calculation

The methodology presented here is based on calculated dose coefficients, which as such cannot be validated against direct measurement. However, it is possible to evaluate the uncertainties in the dose calculation process. On the one hand, the analytical approximation used to calculate absorbed fractions with body shapes from spherical to highly protracted or oblate ellipsoids has an uncertainty (expressed by an absolute coefficient of variation) not exceeding 15% for photons and 10% for electrons (Ulanovsky, 2014; Ulanovsky and Prohl, 2006; Ulanovsky et al., 2008). On the other hand, the second-order polynomial formula used to estimate the breathing rate as a function of organism mass (Eq. (4)) has a low uncertainty of the residuals characterised by a geometric standard deviation of 1.47, corresponding to a ratio of 97.5% to 2.5% percentiles being equal to approximately 4.6. The differences between absorbed whole body dose and air kerma for the energies and organism sizes involved are also negligible, such that the latter can serve as a reasonable surrogate for the average whole-body absorbed dose (ICRP, In press). Ultimately, the numerical factors influencing our Monte Carlo-calculated DCs are not the main uncertainty sources for exposure scenarios and attention should focus on other more significant aspects of Eq. (10), such as the determination of contamination of the environment in specific locations, the CR used to convert activity concentration between soil and air, the equilibrium factor and the occupancy factors used in the assessment.

4. Conclusions

A method has been presented to calculate radiation dose rates arising from radon and thoron progenies to a selection of terrestrial biota represented by the ICRP Reference Animals and Plants. This method is relatively simplified in terms of assuming spherical and ellipsoidal geometries, uniform distribution of radionuclides in the biota, absorbed doses averaged to the level of the whole organism, etc.

That radon or thoron and their progeny are natural sources of radiation is not a real argument to neglect them in an impact assessment for wildlife, especially given the releases of radioactivity from the industrial or technological applications resulting in enhanced concentrations of NORM in the biosphere. These may be 'natural' isotopes but man artificially introduces them in significant quantities in the surface environment and one should have methods to deal with their radiological impact on non-human biota.

The implications of the contribution that $^{220,222}\text{Rn}$ makes to wildlife dose rates and effects arising thereof, needs to be further explored with reference to the application of the ICRP derived consideration reference levels (DCRLs) for wildlife (ICRP, 2008a) and other suggested benchmark dose rates. The problem is compounded by the fact that data on radiation effects arising from exposure of radon or thoron to biota are not currently available. Hence, this study represents a start for enabling a future examination of the consequences of radon exposure and subsequent comparisons with exposure to background (radon) levels, signalling the way for future investigations.

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References

- Beresford, N., Barnett, C., Vives i Batlle, J., Potter, E.D., Ibrahim, Z.-F., Barlow, T.S., et al., 2012. Exposure of burrowing mammals to ^{222}Rn . *Sci. Total Environ.* 431, 252–261.
- Berger, M.J., 1968. Energy deposition in water by photons from point isotropic sources. *J. Nucl. Med.* 9, 15–25.
- Berger, M.J., 1971. Distribution of absorbed doses around point sources of electrons and beta particles in water and other media. *J. Nucl. Med.* 12, 5–23.
- Bide, R.W., Armour, S.J., Yee, E., 2000. Allometric respiration/body mass data for animals to be used for estimates of inhalation toxicity to young adult humans. *J. Appl. Toxicol.* 20, 273–290.
- Brown, J.E., Alfonso, B., Avila, R., Beresford, N.A., Coplestone, D., Pröhl, G., et al., 2008. The ERICA tool. *J. Environ. Radioact.* 99, 1371–1383.
- Brown, J., Alfonso, B., Avila, R., Beresford, N., Coplestone, D., Hosseini, A., 2016. A new version of the ERICA tool to facilitate impact assessments of radioactivity on wild plants and animals. *J. Environ. Radioact.* 153, 141–149.
- Harley, N.H., 1988. Radon daughter dosimetry in the rat tracheobronchial tree. *Radiat. Prot. Dosim.* 24, 457–461.
- Hofmann, W., Winkler-Heil, R., 2015. From cellular doses to average lung dose. *Radiat. Prot. Dosim.* 167, 49–54.
- Hofmann, W., Crawford-Brown, D.J., Fakir, H., Monchaux, G., 2006. Modelling lung cancer incidence in rats following exposure to radon progeny. *Radiat. Prot. Dosim.* 122, 345–348.
- ICRP, 1983. Radionuclide Transformations - Energy and Intensity of Transmissions. ICRP Publication 38 (Annals of the ICRP 11). Pergamon Press, Oxford.
- ICRP, 1987. Lung Cancer Risk from Exposures to Radon Daughters. ICRP Publication 50 (Annals of the ICRP 17/1). Pergamon Press, Oxford.
- ICRP, 1993. Protection Against Radon-222 at Home and at Work. International Commission on Radiation Protection Publication 65. Annals of the ICRP 23, 2.
- ICRP, 1994. Human Respiratory Tract Model for Radiological Protection. (ICRP Publication 66. Annals of the ICRP 24). 1–3. International Commission on Radiological Protection, Elsevier Science Ltd., Oxford.
- ICRP, 2002. Guide for the Practical Application of the ICRP Human Respiratory Tract Model (ICRP Publication SG3). Annals of the ICRP 32, 1–2. International Commission on Radiological Protection, Elsevier Science Ltd., Oxford.
- ICRP, 2007. The 2007 Recommendations of the International Commission on Radiological Protection. ICRP Publication 103, Annals of the ICRP 37, 2–4. Pergamon Press, Oxford.
- ICRP, 2008a. The Concept and Use of Reference Animals and Plants for the Purposes of Environmental Protection. In: Valentin, J. (Ed.), International Commission on Radiological Protection Publication 108, Annals of the ICRP 38, 4–6. Elsevier, Oxford (76 pp.).
- ICRP, 2008b. Nuclear Decay Data for Dosimetric Calculations. International Commission on Radiological Protection Publication 107. Annals of the ICRP 38, 3.
- ICRP, 2010. Lung Cancer Risk from Radon and Progeny and Statement on Radon. International Commission on Radiation Protection Publication 115, Annals of the ICRP 40, 1.
- ICRP, 2014a. Protection of the Environment under Different Exposure Situations. ICRP Publication 124, Annals of the ICRP 43, 1.
- ICRP, 2014b. Radiological Protection against Radon Exposure. International Commission on Radiological Protection Publication 126. Ann. ICRP 43, 3.
- ICRP. Dose Coefficients for Non-human Biota Environmentally Exposed to Radiation. International Commission on Radiological Protection Publication 136, (In press).
- Keller, G., Folkerts, K.H., Muth, H., 1984. Special aspects of the Rn-222 and daughter product concentrations in dwellings and in the open air. *Radiat. Prot. Dosim.* 7, 151–154.
- Kitowski, I., Komosa, A., Chodorowski, J., 2015. Absorbed radiation dose from radon to the Sand Martin *Riparia riparia* during breeding at the sand mines in eastern Poland. *Int. J. Environ. Res.* 9, 1097–1106.
- Kleiber, M., 1932. Body size and metabolism. *Hilgardia* 6, 315–351.
- Kleiber, M., 1947. Body size and metabolic rate. *Physiol. Rev.* 27, 511–541.
- Kojima, H., 1996. The equilibrium factor between radon and its daughters in the lower atmosphere. *Environ. Int.* 22, S187–S192.
- Kolokotronis, T., Savage, V.M., Deeds, E.J., Fontana, W., 2010. Curvature in metabolic scaling. *Nature* 464, 753–756.
- Kryshev, I.I., Sazykina, T.G., Kryshev, A.I., Boboshko, V.I., Frantsev, V.N., Ermakova, M.A., 2013. Effect of radioactive emissions due to uranium mining and processing operations at the Priargunsky mining and chemical works on the populace and biota. *At. Energy* 113, 214–218.
- MacDonald, C.R., Laverock, M.J., 1998. Radiation exposure and dose to small mammals in radon-rich soils. *Arch. Environ. Contam. Toxicol.* 35, 109–120.
- McWorter, D.B., Sunada, D.K., 1988. Groundwater Hydrology and Hydraulics. Water Resources Publications, Fort Collins, Colorado, USA.
- Porstendörfer, J., 1994. Properties and behaviour of radon and thoron and their decay products in the air. *J. Aerosol Sci.* 25, 219–263.
- Reich, P.B., Tjoelker, M.B., Machado, J.-L., Oleksyn, J., 2005. Universal scaling of respiratory metabolism, size and nitrogen in plants. *Nature* 439, 457–461.
- Rubner, M., 1883. Über den Einfluss der Körpergröße auf Stoff- und Kraftwechsel. *Z. Biol.* 19, 535–562.
- Schmidt-Nielsen, K., 1984. Scaling: Why is Animal Size so Important? 1984. Cambridge University Press

- Strong, J.C., Baker, S.T., 1996. Lung deposition in rodents during exposure to attached radon progeny. *Environ. Int.* 22.
- Taranenko, V., Pröhl, G., Gómez-Ros, J.M., 2004. Absorbed dose rate conversion coefficients for reference biota for external photon and internal exposures. *J. Radiol. Prot.* 24, A35–A62.
- Tobias, C.A., Jones, H.B., Lawrence, J.H., Hamilton, J.G., 1949. The uptake and elimination of krypton and other inert gases by the human body. *J. Clin. Investig.* 28, 1375–1385.
- Ulanovsky, A., 2014. Absorbed doses in tissue-equivalent spheres above radioactive sources in soil. *Radiat. Environ. Biophys.* 53, 729–737.
- Ulanovsky, A., 2016. Dosimetry for animals and plants: contending with biota diversity. *Ann. ICRP* 45, 225–238.
- Ulanovsky, A., Prohl, G., 2006. A practical method for assessment of dose conversion coefficients for aquatic biota. *Radiat. Environ. Biophys.* 45, 203–214.
- Ulanovsky, A., Prohl, G., 2012. Dosimetry for reference animals and plants: current state and prospects. *Ann. ICRP* 41, 218–232.
- Ulanovsky, A., Prohl, G., Gomez-Ros, J.M., 2008. Methods for calculating dose conversion coefficients for terrestrial and aquatic biota. *J. Environ. Radioact.* 99, 1440–1448.
- Vives i Batlle, J., Jones, S.R., Gomez-Ros, J.M., 2004. A method for calculation of dose per unit concentration values for aquatic biota. *J. Radiol. Prot.* 24, A13–A34.
- Vives i Batlle, J., Jones, S.R., Copplestone, D., 2008. Dosimetric model for biota exposure to inhaled radon daughters. Environment Agency Science Report SC060080 (34 pp.).
- Vives i Batlle, J., Smith, A.D., Vives-Lynch, S., Copplestone, D., Prohl, G., Strand, T., 2011. Model-derived dose rates per unit concentration of radon in air in a generic plant geometry. *Radiat. Environ. Biophys.* 50, 513–529.
- Vives i Batlle, J., Copplestone, D., Jones, S.R., Williams, C., 2012. Allometric methodology for the assessment of radon exposures to wildlife. *Sci. Total Environ.* 427–428, 50–59.
- Vives i Batlle, J., Jones, S.R., Copplestone, D., 2015. A methodology for ^{41}Ar , $^{85,88}\text{Kr}$ and $^{131\text{m},133}\text{Xe}$ wildlife dose assessment. *J. Environ. Radioact.* 144, 152–161.
- Wenbin, M., Yan, J., Chanzhu, Q., Weidao, Z., 1990. Investigation of indoor and outdoor radon and its daughters in the region of Lianing, People's Republic of China. *Radiat. Prot. Dosim.* 30.
- West, G.B., Brown, J.H., 2005. The origin of allometric scaling laws in biology from genomes to ecosystems: towards a quantitative unifying theory of biological structure and organization. *J. Exp. Biol.* 208, 1575–1592.
- Winkler-Heil, R., Hussain, M., Hofmann, W., 2015. Stochastic rat lung dosimetry for inhaled radon progeny: a surrogate for the human lung for lung cancer risk assessment. *Radiat. Environ. Biophys.* 54, 225–241.
- Wrixon, A.D., Green, B.M.R., Lomas, P.R., Miles, J.C.H., Cliff, K.D., Francis, E.A., et al., 1988. Natural radiation exposure in UK dwellings. National Radiological Protection Board Report NRPB R-190, Chilton, UK.