

Oak Ridge National Laboratory

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Environmental Sciences Division

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ABSTRACT

This report provides cancer risk coefficients and age-specific effective dose coefficients for inhalation of ^{222}Rn , ^{220}Rn , and dosimetrically important short-lived radioactive progeny of these two radon isotopes. The coefficients tabulated here are for inhalation of individual radionuclides without accompanying progeny but include the contribution to dose of ingrowth of radioactive progeny in the body following intake of a parent radionuclide. This approach allows the user to derive effective dose estimates or cancer risk estimates for any known or hypothetical combination of ^{222}Rn or ^{220}Rn and its short-lived progeny in air.

The tabulated coefficients are based on biokinetic and dosimetric models developed for application in upcoming reports of the International Commission on Radiological Protection (ICRP) on occupational and environmental intake of radionuclides. The age-specific biokinetic models for the elements addressed here (lead, polonium, and bismuth) are summarized in the following section. The dosimetric models applied here are generic age- and gender-specific models that convert the predicted time-dependent distribution of a radionuclide in the body to tissue-specific dose coefficients, which are used to derive an effective dose coefficient.

SUMMARY OF BIOKINETIC MODELS USED TO DERIVE RISK AND DOSE COEFFICIENTS

MODEL FOR INHALED RADON

The age-specific biokinetic model applied here to inhaled radon is an extension to younger age groups of a model proposed by Leggett et al. (2013) for adult males and females. The model structure is shown in Figure 1. Parameter values are based on physical laws governing transfer of a non-reactive and soluble gas between materials. Essentially, the biokinetics of radon is assumed to be determined by the blood-to-air partition coefficient and the blood perfusion rates, tissue-to-blood partition coefficients, and volumes of the tissues represented by the compartments of the model shown in Figure 1. The partition coefficient for two compartments is defined as the ratio of the concentrations of the gas in the compartments at equilibrium.

Tissue-to-blood partition coefficients for radon are based on results of *in vivo* and *in vitro* studies of the solubility of radon in blood and tissues of rats and *in vitro* data on the solubility of radon in human blood and fat. The following tissue-to-blood partition coefficients for radon were selected from reported experimental data: 11.0 for fat (which accumulates radon to a much greater extent than other tissues, due to a relatively slow release from fat back to blood; 0.7 for kidneys and liver; and 0.4 for bone and for soft tissues containing relatively little fat. For soft tissues that contain a relatively high percentage of fat by mass (e.g., breast tissue and red marrow in adolescents and adults), mass-weighted averages of partition coefficients for fat (11.0) and lean tissue (0.4) were applied. An experimentally determined blood-to-air partition coefficient of 0.43 is assumed.

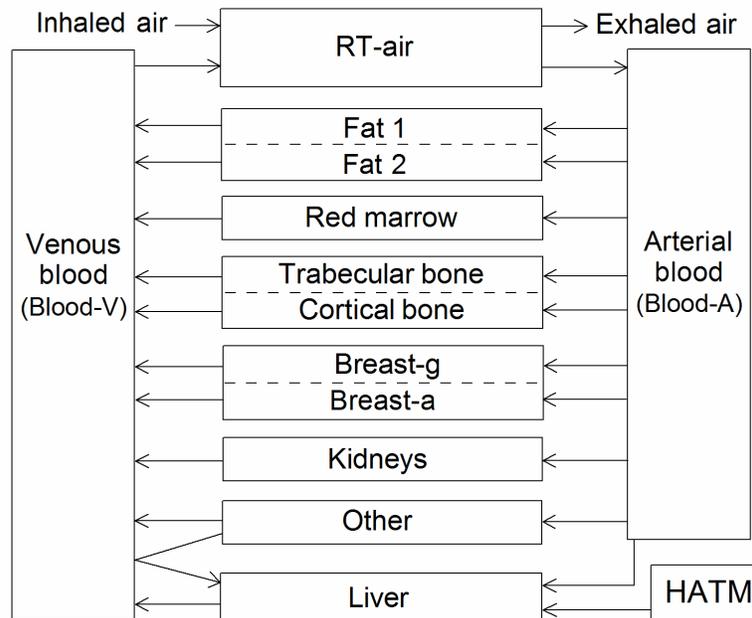


Figure 1. Structure of the biokinetic model for radon. RT air = respiratory tract air; Breast-g = glandular tissue of breast; Breast-a = adipose tissue of breast; HATM = Human Alimentary Tract Model (ICRP, 2006).

In the radon model used here, radon entering the respiratory air (RT-air) is assumed to equilibrate rapidly between RT-air and pulmonary blood, with relative concentrations in the two pools determined by the blood-to-air partition coefficient (0.43). Gas retained in the pulmonary blood is assumed to be distributed in arterial blood to tissues in proportion to the percentage of cardiac output received by each tissue. The transfer coefficient from a tissue to venous blood is determined by the blood perfusion rate, the volume of the compartment, and the tissue-to-blood partition coefficient. The gas is carried in the venous blood to the pulmonary blood. The cycle continues until the body burden is depleted due to exchange between pulmonary blood and RT-air and loss from the body in expired air. Age-specific tissue volumes were based on reference tissue masses given in ICRP Publication 89 (2002) and tissue densities from ICRP Publications 89 and 23 (1975): fat or adipose tissue, 0.92 g cm^{-3} ; red marrow, 1.0 g cm^{-3} ; all other soft tissues, 1.04 g cm^{-3} ; bone, 1.9 g cm^{-3} . Age-specific values of cardiac output were taken from ICRP Publication 89. The age-specific distribution of cardiac output was extrapolated from values for adults given in ICRP Publication 89, by assigning two-fold higher blood flow to bone in pre-adults, adjusted blood flow to breast based on the typical mass of breast at a given age, and an adjusted blood flow to “Other” (all tissues not identified explicitly in the model structure) so that total blood flow added to 100% of cardiac output.

In the model, breast is divided into two compartments: breast-g, representing glandular tissue of the breast; and breast-a, representing the adipose tissue that makes up the rest of the breast. The tissue-to-blood partition coefficient for breast-a is derived as a mass-weighted average of the tissue-to-blood partition coefficients for fat and other: $[0.8 \times 11(\text{fat})] + [0.2 \times 0.4(\text{other})] = 8.9$.

The derivation of transfer coefficients is illustrated for systemic radon in a reference adult male. Radon is assumed to clear from arterial blood (Blood-A) at the rate $6.5 \text{ L min}^{-1} \times 1440 \text{ min d}^{-1} / 1.431 \text{ L} = 6541 \text{ d}^{-1}$, where 1.431 L is the volume of Blood-A (assumed to represent 27% of a total blood volume of 5.3 L in an adult male). Radon transfers from venous blood (Blood-V) to RT-air at the rate $6.5 \text{ L min}^{-1} \times 1440 \text{ min d}^{-1} / 3.869 \text{ L} = 2419 \text{ d}^{-1}$, where 3.869 L is the reference volume of venous blood in an adult male (73% of total blood volume). The transfer coefficient from Blood-A to Kidneys, for example, is $0.19 \times 6541 \text{ d}^{-1} = 1243 \text{ d}^{-1}$, where 0.19 is the reference fraction of cardiac output received by the kidneys. The transfer coefficient from Kidneys to Blood-V is $1440 \text{ min d}^{-1} \times 0.19 \times 6.5 \text{ L min}^{-1} / (0.298 \text{ L} \times 0.7) = 8525 \text{ d}^{-1}$, where 0.298 L is the volume of the kidneys and 0.7 is the kidneys-to-blood partition coefficient.

Biokinetic models for short-lived radon progeny addressed here (isotopes of Pb, Po, and Bi)

The predicted behaviors of inhaled Pb, Po, and Bi following deposition in the respiratory tract are based on a combination of three types of kinetic models: a largely generic respiratory model for inhaled particulate material; a generic alimentary tract model describing the flow of swallowed material through the tract, together with an element-specific gastrointestinal absorption fraction; and element-specific biokinetic models describing the distribution, retention, and excretion of Pb, Po, and Bi after absorption to blood.

Respiratory model

The respiratory tract model used here is the ICRP’s Human Respiratory Tract Model (HRTM). The HRTM was introduced in ICRP Publication 66 (1994) and updated in ICRP Publication 130 (2015). The updated HRTM was used to derive the risk and dose coefficients tabulated in this report.

The structure of the updated HRTM is shown in Figure 2. The model divides the respiratory system into extrathoracic (ET) and thoracic regions. The airways of the ET region are further divided into two categories: the anterior nasal passages (ET₁), in which deposits are removed by extrinsic means such as nose blowing, and the posterior nasal passages (ET₂) including the nasopharynx, oropharynx, and the larynx, from which deposits are swallowed. The airways of the thorax include the bronchi (compartments BB₁ and BB_{seq}), bronchioles (bb₁ and bb_{seq}), and alveolar-interstitial region. Material deposited in the thoracic airways may be cleared into blood by absorption, to the alimentary tract by mechanical processes

(that is, transported upward and swallowed), and to the regional lymph nodes (LN_{ET} for the ET region and LN_{TH} for the thoracic region) via lymphatic channels.

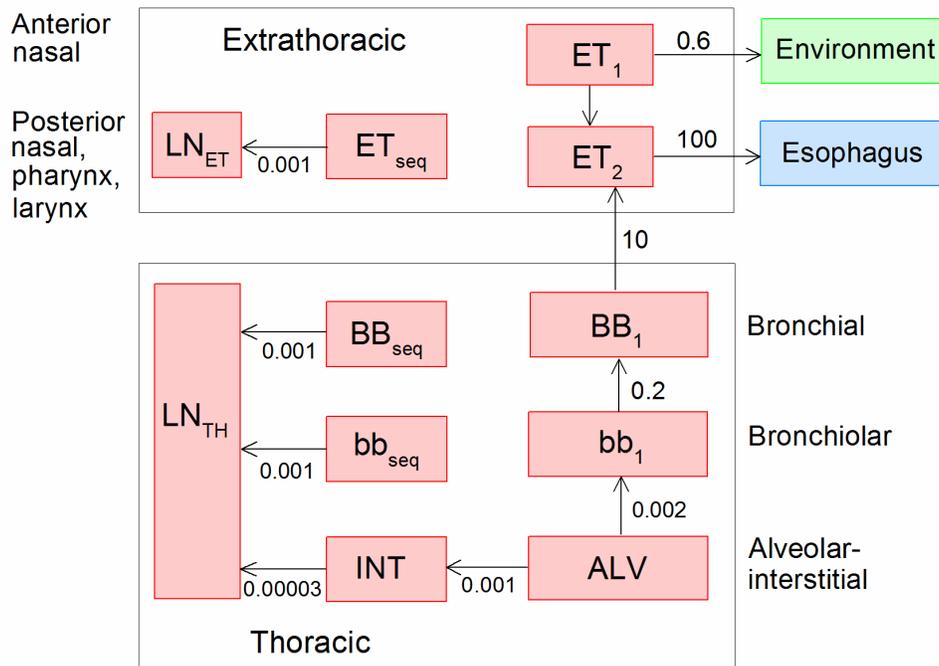


Figure 2. Structure of the updated Human Respiratory Tract Model (HRTM) (ICRP, 2015). Numbers next to arrows are age-independent particle transport rates (d^{-1}) along indicated paths. Abbreviations: ET = extrathoracic; BB = bronchi; bb = bronchioles; LN = lymph nodes; ALV = alveolar; INT = interstitial; TH = thoracic; seq = sequestered.

Particle transport rates are shown beside the arrows in Figure 2. These turnover rates are reference fractional turnover rates (d^{-1}). For example, particle transport from BB_1 to ET_2 is assigned the rate constant $10 d^{-1}$.

In addition to mechanical clearance, deposited activity is removed from the respiratory tract by absorption to blood. The rate of absorption to blood depends on the rate of dissolution of the particles in lung fluids, which in turn depends on the physical and chemical properties of the deposited particles. Dissolved activity is assumed to be immediately absorbed to blood except for: (1) a small fraction assumed to be retained for an extended period in tissues of the respiratory tract and eventually transferred to respiratory lymph nodes, and (2) an element-specific “bound” fraction (zero for some elements) of the dissolved activity is assumed to be retained in respiratory tissues and gradually transferred directly to blood.

A common absorption rate is applied to all respiratory regions in which absorption occurs (all respiratory compartments other than ET_1). The absorption rate is described by a dissolution model within the HRTM. The relatively simple form of the dissolution model shown in Figure 3 appears to be applicable to the preponderance of inhaled aerosols, including radon progeny. In this dissolution model it is assumed that a fraction f_r of deposited material dissolves at the relatively fast rate s_r and the remaining fraction $1-f_r$ dissolves more slowly at the rate s_s . The relatively soluble and less soluble fractions are assigned to separate compartments upon deposition.

Table 1 lists the ICRP’s provisional reference values for the absorption parameter values f_r , s_r , and s_s for polonium, lead, and bismuth inhaled as radon progeny carried in indoor air. Absorption parameter values for inhaled activity are independent of age.

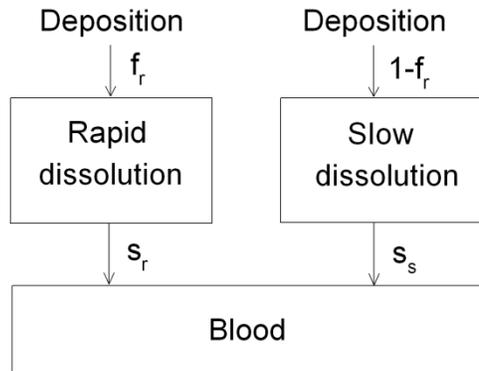


Figure 3. Model of time-dependent absorption within the HRTM. Fractions f_r and $1-f_r$ of deposited material have different dissolution rates (s_r and s_s , respectively).

Table 1. Absorption parameter values for inhaled radon progeny, applicable to all ages at intake.

Inhaled radon progeny	Dissolution parameter values					Absorption from the alimentary tract, f_A
	f_r	s_r (d ⁻¹)	s_s (d ⁻¹)	f_b	s_b (d ⁻¹)	
Polonium	1	3	–	0	–	0.1
Lead	0.1	100	1.7	0.5	1.7	0.2
Bismuth	1	1	–	0	–	0.05

Regional deposition in the HRTM assumed for inhaled radon progeny is shown in Table 2. These values are based on empirical data on typical physical of aerosols carrying radon progeny in homes or other buildings, together with a deposition model for inhaled material that predicts sites of deposition of aerosols in the respiratory tract based on their physical characteristics. The deposition fractions are assumed here to be independent of age at intake.

Table 2. Regional deposition (%) of radon progeny assumed in derivation of effective dose coefficients and risk coefficients for inhalation of individual radon progeny^a

	ET1	ET2	BB	bb	AI	Total
Rn-222						
Indoor	12.7	6.85	1.25	3.06	11.8	35.66
Mine	3.65	1.96	0.49	2.24	9.85	18.19
Rn-220						
Indoor	8.15	4.39	0.69	2.28	10.8	26.31
Mine	3.40	1.84	0.45	2.20	9.89	17.78

ALIMENTARY TRACT MODEL

The ICRP's Human Alimentary Tract Model (HATM), introduced in ICRP Publication 100 (2006), is used to describe transfer of radionuclides through the contents of the alimentary tract. The structure of the HATM is shown in Figure 4. The compartments and paths of movement represent the following processes:

- entry of a radionuclide into the oral cavity by ingestion or into the esophagus after mechanical clearance from the respiratory tract;
- sequential transfer through the lumen of the oral cavity, esophagus, stomach, small intestine, and segments of the colon, followed by emptying in feces;
- radionuclide deposition and retention on or between the teeth and return to the oral cavity;
- deposition and retention in the oral mucosa or walls of the stomach or intestines;
- transfer from the oral mucosa or walls of the stomach or intestines back into the luminal contents or into blood (absorption);
- transfer from secretory organs or blood into the contents of segments of the tract.

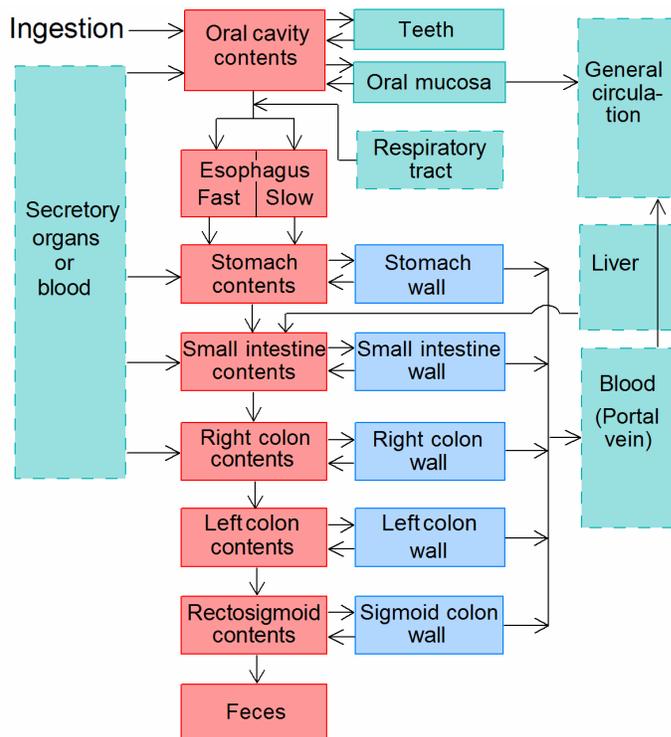


Figure Error! No text of specified style in document.. Structure of the Human Alimentary Tract Model (ICRP, 2006) and connections to the respiratory tract and systemic activity.

Entry into the alimentary tract by ingestion or transfer from the respiratory tract and sequential transfer through the lumen of the tract are regarded as generic processes in that the rates are assumed to be independent of the radionuclide or its form. Movement of material through the lumen of the alimentary tract is assumed to follow first-order kinetics. The residence times of material in the lumen of segments of the alimentary tract were initially estimated in terms of the mean transit time because this is the form in which data on alimentary tract motility generally are reported. The transit time of an atom in a region of

the tract is the length of time that it resides in that region, and the transit time of a substance in a region (also called the mean transit time) is the mean of the distribution of transit times of its atoms. The first-order transfer rate or “emptying rate” used to represent a transit time T (h^{-1}) in a segment of the alimentary tract is $1/T$, and the corresponding biological half-time (h) in the segment is $(\ln 2) \times T$. Transit times of lumenal contents are regarded as primary parameter values of the HATM, and the first-order transfer rates derived from those transit times are regarded as secondary values. Separate reference transit times are provided in ICRP Publication 100 for transfer of ingested solids, liquids, and total diet through the mouth and esophagus and for transit of non-caloric liquids, caloric liquids, solids, and total diet through the stomach.

BIOKINETIC MODELS FOR RADIONUCLIDES THAT REACH THE SYSTEMIC CIRCULATION

The biokinetic models for systemic Pb, Po, and Bi used in this analysis will be described in the final report on this task. Briefly, the model for Pb is the age-specific model for members of the public used in ICRP Publication 67 (1993). The models for Po and Bi were developed for use in upcoming ICRP documents on doses to members of the public.

RISK COEFFICIENT DERIVATION

Risk coefficients for Radon and its short lived progeny were derived using the method described in the EPA FGR13 report. The cancer risk (r_a) from a unit intake of the radionuclide at age (x_i) was calculated from the continuously varying absorbed dose rate (\dot{D}), the tissue specific risk per unit dose ($r(x)$) received at age x from the EPA blue book and the U.S. population’s age-dependent survival probability ($S(x_i)$) as shown in equation 1. If the cancer risk is known for the radionuclide intake at a particular age, the next step then involves calculating the cancer risk related to continuous lifetime exposure. The average lifetime cancer risk coefficient is calculated using the cancer risk for a unit intake at a specific age (r_a), the intake rate as a function of time ($u(x)$), and the U.S. population age dependent survival probability ($S(x_i)$) as shown in equation 2.

$$r_a(x_i) = \frac{\int_{x_i}^{\infty} \dot{D}(x)r(x)S(x) dx}{S(x_i)} \quad \text{Eq. 1}$$

$$\bar{r}_a = \frac{\int_0^{\infty} u(x)r_a(x)S(x) dx}{\int_0^{\infty} u(x)S(x) dx} \quad \text{Eq. 2}$$

RESULTS

Effective dose coefficients for inhaled ^{222}Rn , ^{220}Rn , or their dosimetrically dominant short-lived progeny are listed in Table 1. Mortality and Morbidity risk coefficients for these radionuclides are listed in Table 2.

Table 1. Age-specific effective dose coefficients (Sv Bq ⁻¹) for inhalation of radon-220, radon-222, and their dosimetrically dominant short-lived progeny. ^a							
Radio-nuclide	Age at intake						Per-Capita
	Infant	1 y	5 y	10 y	15 y	Adult	
Rn-220	2.49E-09	8.56E-10	4.61E-10	2.24E-10	1.59E-10	1.46E-10	2.21E-10
Rn-222	4.80E-08	1.26E-08	5.78E-09	1.25E-09	5.50E-10	4.27E-10	1.77E-09
Rn-220 progeny							
Bi-212	2.6E-08	2.1E-08	1.8E-08	1.7E-08	1.7E-08	1.6E-08	1.65E-08
Pb-212	7.5E-07	2.4E-07	1.6E-07	1.1E-07	9.0E-08	8.6E-08	1.05E-07
Rn-222 progeny							
Bi-214	1.4E-08	1.1E-08	1.1E-08	1.0E-08	9.9E-09	9.7E-09	9.90E-09
Pb-214	3.1E-08	1.8E-08	1.4E-08	1.3E-08	1.2E-08	1.2E-08	1.26E-08
Po-218	3.2E-09	2.5E-09	2.2E-09	2.1E-09	2.1E-09	2.0E-09	2.06E-09
^a Each dose coefficient is based on intake of the indicated radionuclide (the “parent”) without accompanying progeny but includes the dose from progeny produced in the body after intake of the parent.							

Table 2 - Morbidity and Mortality risk coefficients (Bq⁻¹) for inhalation of radon-220, radon-222, and their dosimetrically dominant short-lived progeny.

Radio-nuclide	Morbidity 1/Bq			Mortality 1/Bq		
	Female	Male	Population	Female	Male	Population
Bi-212	4.40E-09	1.70E-09	3.05E-09	3.60E-09	1.60E-09	2.60E-09
Bi-214	2.40E-09	9.40E-10	1.67E-09	2.00E-09	8.70E-10	1.44E-09
Pb-212	2.50E-08	8.90E-09	1.70E-08	1.90E-08	8.00E-09	1.35E-08
Pb-214	3.00E-09	1.20E-09	2.10E-09	2.50E-09	1.10E-09	1.80E-09
Po-218	5.40E-10	2.10E-10	3.75E-10	4.50E-10	1.90E-10	3.20E-10
Rn-220	4.70E-11	1.50E-11	3.10E-11	3.20E-11	1.20E-11	2.20E-11
Rn-222	9.80E-11	2.50E-11	6.15E-11	4.90E-11	1.50E-11	3.20E-11

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