Chapter 6. Models for Estimating the Radiation Hazards of Uranium Mines

K. N. Wise
Australian Radiation Laboratory

Abstract. Hazards to the health of workers in uranium mines derive from the decay products of radon and from uranium and its descendants. Radon daughters in mine atmospheres are either attached to aerosols or exist as free atoms and their physical state determines in which part of the lung the daughters deposit. The factors which influence the proportions of radon daughters attached to aerosols, their deposition in the lung and the dose received by the cells in lung tissue are discussed. The estimation of dose to tissue from inhalation or ingestion or uranium and daughters is based on a different set of models which have been applied in recent ICRP reports. The models used to describe the deposition of particulates, their movement in the gut and their uptake by organs, which form the basis for future limits on the concentration of uranium and daughters in air or on their intake with food, are outlined.

INTRODUCTION

In the early years of the 16th century, mines were opened at Joachimsthal and Schneeberg in Central Europe. By 1546 the miners were reported to have an unusually high frequency of a fatal lung disease. The malignant nature of this disease was noted in 1879 and shown to be lung cancer in the first quarter of this century. The ore in these mines has been worked at various times for cobalt, bismuth, nickel, radium, arsenic and nickel; some of these metals are known to be carcinogens. Today, it is known that excess cancer mortality exists not only for workers in uranium mines but also in fluorspar mines, in underground iron mines, and in underground hard-rock mines. Careful statistical studies have linked lung cancer risks to the level of radon-222 daughters in the mines; statistically significant respiratory cancer deaths are found in the exposure range 120-360WLM and at higher exposures after full account has been taken of smoking habits and residency (Lundin et al., 1971). Where the radon daughter levels are low, as in potash mining, there is no difference in the risks for surface workers and underground miners. The cancer cells seen are predominantly of the small-cell undifferentiated type; the basal cells have been implicated as a point of origin of the cancer cells (Kotin 1966).

The dust produced by the mining operations of drilling, blasting and ore loading is no less a problem, as these airborne dusts contain the long-lived alpha-emitting radionuclides of the uranium-238 and uranium-235 families. Such airborne radioactive contamination is also produced in uranium mills where the ore or the final product is physically processed. Along with the usual radiological hazards there is the additional one of heavy metal poisoning. For example, ingestion or inhalation of uranium can lead to kidney damage or death and as little as 60 mg of uranium in the blood at the one time could produce a human fatality (Eve 1964).

Inhalation is the principle mode of entry of either radon daughters or uranium ore dust into the body. For radon daughters the lung is the organ most at risk while for uranium and its descendants organs other than the lung

may be at risk. Part 1 discusses the estimation of the radiation dose deriving from radon daughters while Part 2 is confined to current views on how uranium and its descendants move around the body and the hazard they therefore impose.

PART 1 : RADON AND DAUGHTERS.

Physical properties

Radon decays by alpha emission; its immediate daughter products, which are also alpha emitting, have half-lives of less than 30 minutes and are, therefore, of more concern in the assessment of the hazard to the respiratory tract than the much longer lived radon-222. Lead-210 with its half-life of 22 years may be regarded as non-radioactive when considering the evaluation of inhalation hazard. Some physical properties of radon and its daughters are listed in Table 1.

As radon decays by alpha emission, the daughter polonium-218 (RaA) is formed as a recoiling ion with an initial kinetic energy of 101 keV and is positively charged at the end of its recoil path, which in air is about 100 μm. These ions have a high diffusion coefficient of 0.05 cm^2s^{-1} to 0.07 cm^2s^{-1} and attach themselves to any available surface fairly readily. If the available surface is an airborne aerosol, the attachment rate depends on the size distribution and the concentration of the aerosol (Raabe 1969). Polonium-218 decays, in turn, by alpha emission. If the decaying polonium-218 is attached to an aerosol particle, the recoiling atom of lead-214 will either penetrate into or remove itself from the particle. For large particles (> 0.5 μm), half the recoiling atoms of lead-214 could be expected to escape but all of the lead-214 could be expected to escape if the particle is sufficiently small (< 0.1 μm). Mercer (1976) has recently estimated the fraction lost by recoil to be 0.83. Loss of the bismuth-214 formed by beta-emission from lead-214 is negligibly small.

When the radon daughter products polonium-218, lead-214, bismuth-214 and polonium-214 are free in the air or associated with very small clusters of molecules (eg water vapour) they are called unattached radon daughters. If the radon daughters are incorporated into aerosol particles, which are at least an order of magnitude larger than unattached daughter clusters, they are said to be attached. Obviously, as the unattached daughters are considerably smaller than those attached to aerosols, (and therefore have a higher diffusion coefficient) they will deposit in the respiratory tract more efficiently.

Table 1

Properties of Radon and Radon Daughters.

Isotopes	Half-life	Major radiations	For α-emitting isotopes	
			Energy, MeV	Tissue range, μm
Rn-222	3.82 day	α	5.49	41
Po-218	3.05 min	α	6.00	47
Pb-214	26.8 min	β-		
Bi-214	19.7 min	β-		
Po-214	1.64 x 10-4 sec	α	7.69	71
Pb-210	20.4 yr	β-		
Bi-210	5.01 day	β-		
Po-210	138 day	α	5.31	39
Pb-206	Stable			

Aerosol characteristics

The size of aerosols in the atmosphere range from clusters of a few molecules to particles of about 40μm diameter. Particles of about 10 nm diameter and smaller have very short life-times because they rapidly attach to other aerosol particles and can exist in considerable concentrations only if they are produced constantly. At the other end of the scale, particles larger than 40 μm diameter are airborne only for a limited time and their occurrence is restricted to the vicinity of their source (eg from blasting, near heavy machinery or from vehicles moving on loose soil). The complete size distribution of aerosols cannot be obtained by one measurement procedure only - particles smaller than 0.1 μm can be obtained from measurement of their diffusion coefficient or their electrical mobility while particles larger than 0.5 μm can be measured using optical particle counters (IAEA 1978).

Three types of particle size distributions can be distinguished : namely background, oceanic and continental (Bricard 1977). The background aerosol corresponds to the purest air in the lower and middle troposphere with concentrations of up to 700 particles per cubic centimetre. The oceanic aerosol differs from the backround aerosol in that particles are predominatly of oceanic origin (eg salt spray) and are larger. The continental aerosol arise over polluted areas of continents with concentrations ranging from 10^4 to 10^5 per cubic centimetre.

The aerosol size distribution is generally similar to the log-normal distribution and two parameters used to describe the log-normal distribution, the median and the geometric standard deviation, are commonly used to summarise measurements on the aerosol size distributions - hence, use of terms such as count median diameter, activity median diameter or mass median diameter. Junge (1963) has given model distributions for the sizes of the atmospheric aerosols - the continental and oceanic aerosols have count median diameters of about 0.06 μm and 0.08 μm respectively with a geometric standard deviation of about 2.3. As the rate of attachment of radon daughters is proportional to the square of the particle diameter (Raabe 1969; Kruger and Nothling 1979) the corresponding activity median diameter should be about 0.26 μm and the geometric standard deviation about 2.3. George et al (1975) have reported activity median diameters ranging from 0.085 μm to 0.32 μm (mean 0.17 $\pm$ 0.6) while the geometric standard deviation ranged from 1.3 to 4 (mean 2.7 $\pm$ 0.6) for 27 underground mines.

Factors affecting unattached fraction

Two models appear in the literature which provide simple relationships between the unattached fraction and the departure of the daughters from equilibrium. The first model has been discussed by Raabe (1969, 1978) and is useful for the outdoor environment. The second model has been discussed by Jacobi (1964) and by Porstendorfer et al (1978) and is applicable to underground mine tunnels and indoors.

Raabe's model makes several assumptions which appear reasonable. Three of these are: (1) losses to fixed surfaces such as walls are not considered as spaces are generally larger than a two metre cylinder, (2) the concentrations of the unattached daughters are such that there is little agglomeration between them, and (3) the radon gas concentration is constant during the time period of interest. The concentration of daughters at any time after the air contained only pure radon can be found by solving the decay equations through use of Bateman's equations. The concentration of the unattached daughters can also be described through a set of differential equations which

include parameters for the rate of attachment of the unattached daughters to the aerosols and for recoil loss of the daughters from aerosols. Results from calculations based on Raabe's expressions are given in Fig. 1 to Fig. 3. Clearly, (1) for a given aerosol concentration the unattached fraction increases as the daughter concentrations depart further from equilibrium and (2) for a given departure from equilibrium the unattached fraction increases with decreasing aerosol concentration. According to Raabe's model the unattached fraction increases with decreasing size of the aerosol.

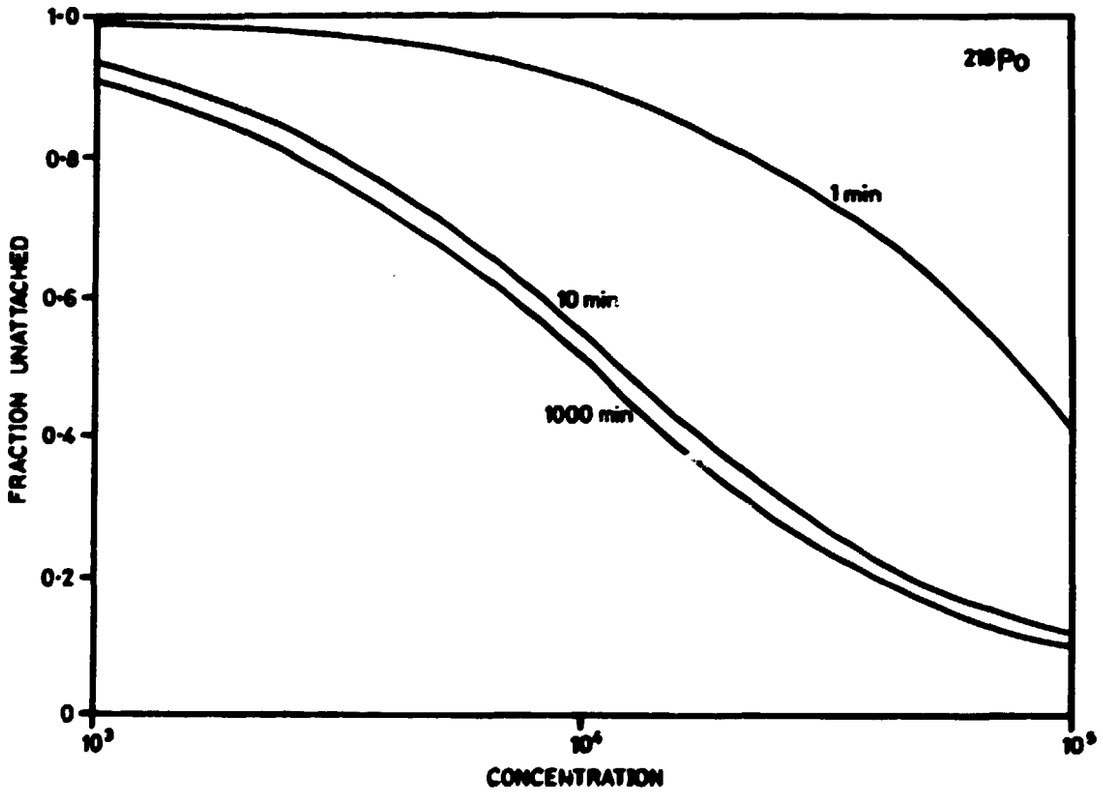

Figure 1. Fraction of ^{218}Po unattached to aerosols for air of ages 1 min, 10 min and 1000 min. The average surface diameter assumed for the aerosol is 0.06 μm.

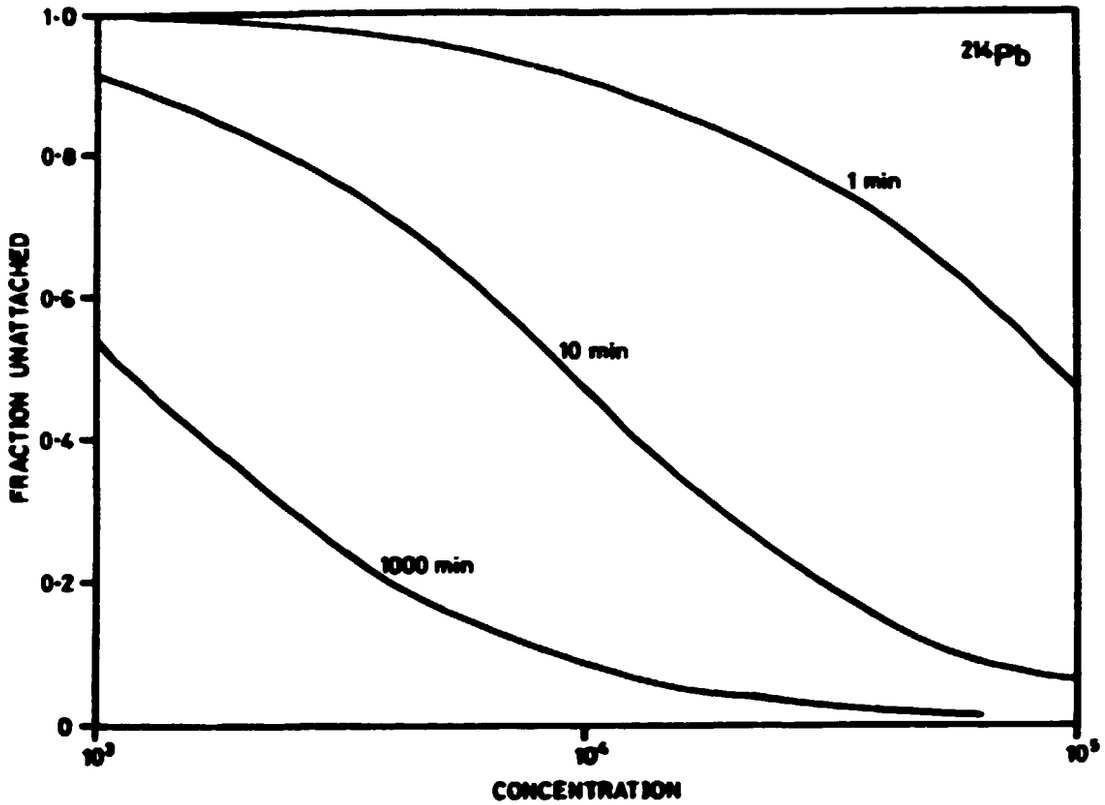

Figure 2. Fraction of ^{214}Pb unattached to aerosols for air of ages 1 min, 10 min and 1000 min. The average surface diameter assumed for the aerosol is 0.06 μm.

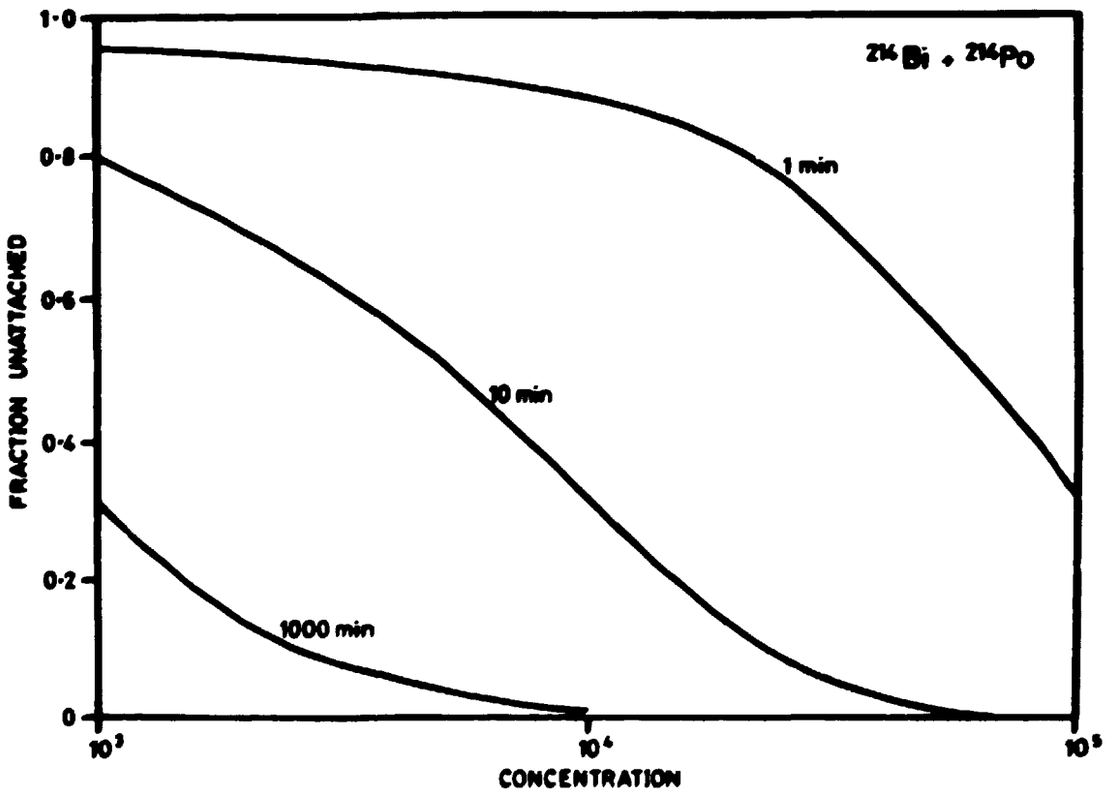

Figure 3. Fraction of 214Bi + 214Po unattached to aerosols for air of ages 1 min, 10 min and 1000 min. The average surface diameter assumed for the aerosol is 0.06 μm.

The second model (Jacobi 1964, Porstendorfer et al 1978) includes the effects of additional losses of radon daughters through attachment to the surfaces of the room or tunnel and through ventilation. The steady state solutions of the differential equations for the unattached and attached daughter concentrations, C_i^f and C_i^a, respectively, are:

$$C_i^f = \frac{\lambda_i C_{i-1}^f + p_{i-1}\lambda_i C_{i-1}^a}{\lambda_v + \lambda_i + \lambda_s + \lambda_f}$$

$$C_i^a = \frac{(1-p_{i-1})\lambda_i C_{i-1} + \lambda_s C_i^f}{\lambda_v + \lambda_i + \lambda_a}$$

where: λ_i is the decay constant for each daughter, i =1,2,3
p_i the recoil loss probability
λ_v the rate of loss by room ventilation
λ_s the rate of loss of unattached daughters by attachment to aerosols
λ_f, λ_a the rate of loss of unattached and attached daughters respectively by attachment to room surfaces.

For underground mines λ_v may be no more than a few air changes per hour (Leach, personal communication) while in domestic environments more than 10 air changes per hour are possible. Thus λ_v could range from 0 to 0.003 s^{-1} while data quoted by Stranden (1979) show λ_f and λ_a to be of the order of 7.2×10^{-3} and 1.8×10^{-5} s^{-1}. Fig. 4 gives estimates of the equilibrium fraction (or WL per 100 pCi/l of radon-222) and the unattached fraction when there are up to 3 air changes per hour.

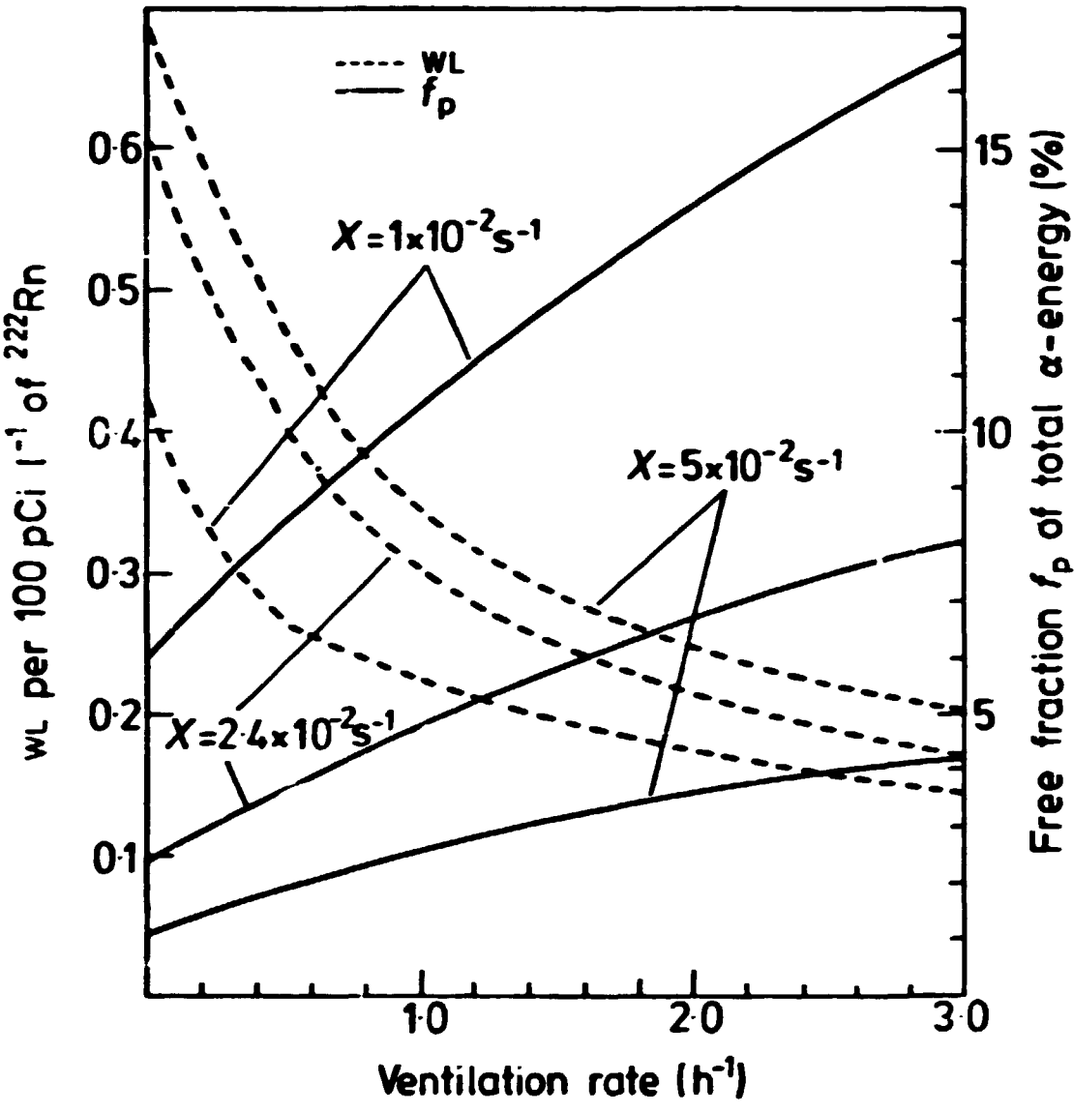

Figure 4. Variation of the equilibrium fraction (left hand scale, broken line) and unattached fraction (right hand scale, solid line) as a function of ventilation rate.

Again, the unattached fraction increases as the departure from equilibrium increases. Further, the unattached fraction increases with decreasing size or decreasing concentration (i.e. with decreasing attachment rate).

Measurements of the unattached fraction in mine environments have been done primarily in underground mines. Fig. 5 summarises in a cumulative distribution form results obtained by Craft et al (1966) and by Raghavayya and Jones (1974) - later amended by Mercer in 1975.

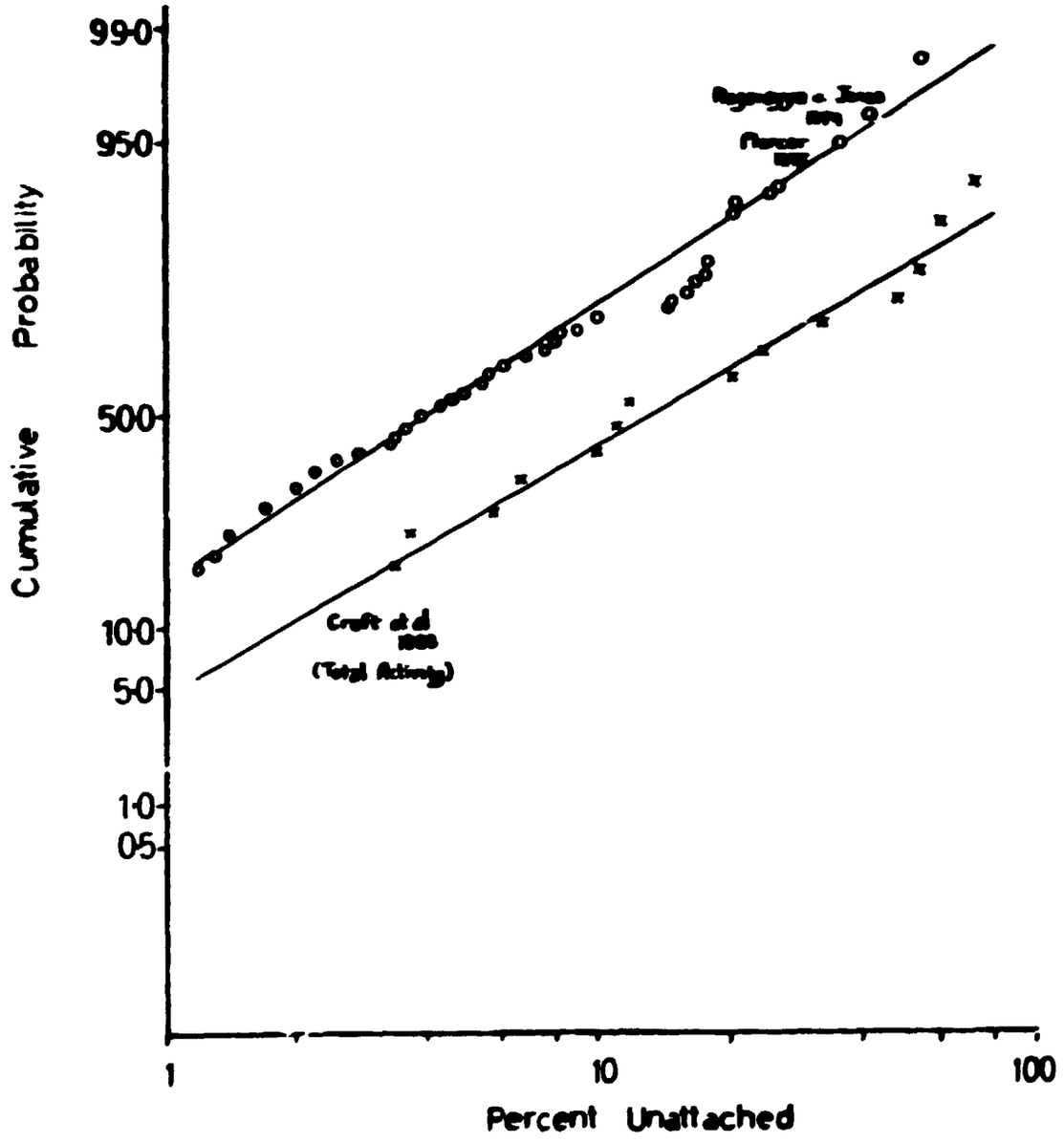

Figure 5. Cumulative probability distributions for fraction of activity unattached.

The figure shows that the unattached fraction appears to be distributed log-normally. Table 2 gives estimates of the median unattached fraction and the geometric standard deviation; these parameters characterise the log-normal distribution. Clearly, the most recent measurements of the unattached fraction in underground mines of the USA exhibit a median unattached fraction for ^{218}Po of about 0.035 and a geometric standard deviation of about 3.

Table 2

Summary of Measurements on Unattached Fraction

Location of Mine	Type of Mine	Measurement summarised here	Median	Geometric s.d.	No of Measurements	Reference
Southern Utah and Colorado	Underground	Total Uncombined	0.20	2.6	16	Croft et al, 1966
New Mexico	Underground	^{218}Po unattached - Mine A	0.08	3.9	10	George and Hinchliffe, 1972
		- Mine B	0.006	4.6	7	
		- Mine C	0.029	2.2	9	
		- Mine D	0.006	1.3	6	
		- Mine E	0.017	3.1	7	
		Mines A - E	0.04	3.3	39	
		Mines B - E	0.03	2.9	29	
New Mexico Colorado	Underground	^{218}Po unattached	0.039	3.6	60	Raghavayya and Jones, 1974 Harcar, 1975

(a) Mine A is not operated by Diesel engines, mines B to E are.

Few measurements are available on the unattached fraction in outdoor air. UNSCEAR (1977) quotes measurements in Sutton, England which range between 0.07 and 0.40. Another two series of measurements in New York City averaged 0.04 (range 0.01-0.06), and 0.09 (range 0.05-0.12). Preliminary measurements have been carried out on the fraction of ^{218}Po unattached to aerosols at Ranger Uranium Mines. Measurements were also made on the radon daughter concentrations and the working levels using an Environmental Working Level Meter made by Harshaw, while some measurements of the aerosol concentrations were made with an uncalibrated Royco 225 optical particle counter. These measurements were made on the ore body in a location some 400 metres from where the pit is being dug. The measurements on the daughters were done at night during times favourable to an inversion. These measurements showed:

- the average ratio of the daughter concentrations were ^{218}Po: ^{214}Po: ^{214}Bi = 1.00: 0.11: 0.01. It is estimated that this ratio corresponds to 7 minute old air
- the working levels were in the range 0.01-0.10 WL

- the unattached fraction ranged from 0.37 to 0.71; a much higher figure than encountered in underground mines.

The aerosol concentrations were roughly an order of magnitude lower than in urban Melbourne. The high unattached fractions are consistent with the observations that (1) the daughters are far removed from equilibrium and (2) the aerosol concentration is relatively low.

Models for lung dosimetry

Calculations based on theoretical models of lung geometry, deposition of aerosols on the airways, movement of mucus towards the throat and location of the sensitive cells have been widely used to estimate the hazard from radon daughters (Altshuler et al 1964; Haque and Collinson 1967; Harley and Pasternak 1972; Jacobi 1980; James 1980; Wise 1980). These parameters suffer from considerable uncertainty and it is not surprising that the estimates of the dose conversion factor vary widely. Their principal virtue, in my view, is that they allow us to make some judgement on the relative risk posed by different environments such as underground mines, open-cut mines and indoors. The models for lung dosimetry are briefly described here. A comparison is also made between the theoretical estimates of the dose to lung in underground mine atmospheres with the dose as estimated from epidemiological data.

The theoretical models of lung geometry that have been put forward are:

- Landahl geometry (1950) in which the lung is divided into six sections, each section having a fixed number of airways of given diameter and length

- Weibel geometry (1963) in which the lung is divided into 23 sections (generations); the number of airways doubles with each succeeding generation and in each generation the airways have a fixed diameter and length

- Horsfield-Cumming geometry (1968) which recognises that there is irregular branching of the airways throughout the lung; the diameter, lengths and numbers of airways have also been given. To date this model has not been used in the estimation of hazards from inhaled materials.

Table 3 and Table 4 give the dimensions and numbers of airways for the Landahl and Weibel geometries; for completeness the anatomical names corresponding to the airways and the speed of the mucus escalator are also given.

The amount deposited in the different regions of the respiratory tract is influenced by several factors which include type of airflow in the lung (turbulent or laminar), particle size, volume of air inhaled per breath and the number of breaths per minute. Generally, particles larger than tens of micrometres diameter are deposited in the nose and do not gain access to the respiratory tract. Particles larger than about a micrometre diameter deposit in the upper respiratory tract primarily by inertial impaction while in the lower respiratory tract, where air motion is slow, these particles deposit by gravitational sedimentation. Particles smaller than a tenth of a micrometre deposit throughout the lungs by Brownian diffusion. Fig. 6 graphically illustrates the relative importance of these mechanisms for the deposition of particles.

Table 3

Lung model [a] of Landahl

Region	Number	Radius (cm)	Length (cm)	Surface area (cm^2)	Mucus transit time (min)
Trachea	1	0.8	12	60	8
Main bronchus	2	0.5	6	40	6
Lobar bronchus	12	0.2	3	45	11
Segmental bronchus	100	0.1	1.5	100	37
Subsegmental bronchus	800	0.075	0.5	200	82
Terminal bronchus	$6x10^4$	0.03	0.3	3400	1980
Respiratory bronchus	$2x10^5$	0.025	0.15	4700	
Alveolar ducts	$2x10^6$	0.02	0.05	30000	
Alveolar sacs	$5x10^7$	0.02		250000	

Notes (a) The respiratory tract is divided into nine regions, six of which belong to the tracheo-bronchial compartment and three to the alveolar area.

(b) The physical dimensions of the airways are those given by Landahl (1950) while mucus transit times are based on the work of Altshuler et al (1964).

Table 4

Lung model [a] of Weibel

Region	Gener-ation	Number	Radius (cm)	Length (cm)	Surface area (cm^2)	Mucus transit time (min)
Trachea	0	1	0.9000	12	67.8	8
Main bronchus	1	2	0.6100	4.76	36.5	6
Lobar bronchus	2	4	0.4150	1.90	19.8	8
	3	8	0.2800	0.76	10.7	3
Segmental bronchus	4	16	0.2250	1.27	28.7	14
	5	32	0.1750	1.07	37.6	12
	6	64	0.1400	0.90	50.7	10
Sub-segmental bronchus	7	128	0.1150	0.76	70.3	32
	8	256	0.0930	0.64	95.7	27
	9	512	0.0770	0.54	134	23
Terminal bronchus	10	1.02×10^3	0.065	0.46	192	445
	11	2.05×10^3	0.0545	0.39	274	378
	12	4.09×10^3	0.0475	0.33	403	320
	13	8.19×10^3	0.0410	0.27	570	261
	14	1.63×10^4	0.0370	0.23	876	223
	15	3.27×10^4	0.0330	0.20	1360	194
	16	6.55×10^4	0.0300	0.165	2040	160
Respiratory bronchus	17	1.31×10^5	0.0270	0.141		
	18	2.62×10^5	0.0250	0.117		
	19	5.24×10^5	0.0235	0.099		
Alveolar duct	20	1.05×10^6	0.0225	0.083		
	21	2.09×10^6	0.0215	0.070		
	22	4.19×10^6	0.0205	0.059		
Alveolar sacs	23	8.39×10^6	0.0205	0.050		
Alveoli			0.02			

Notes (a) The respiratory tract is divided into 24 generations. All tubes in a generation are of equal length and diameter.

(b) The physical dimensions of the airways are those given by Weibel (1963) while mucus transit times are those of Harley and Pasternak (1972) and estimated by them from the work of Altshuler et al (1964).

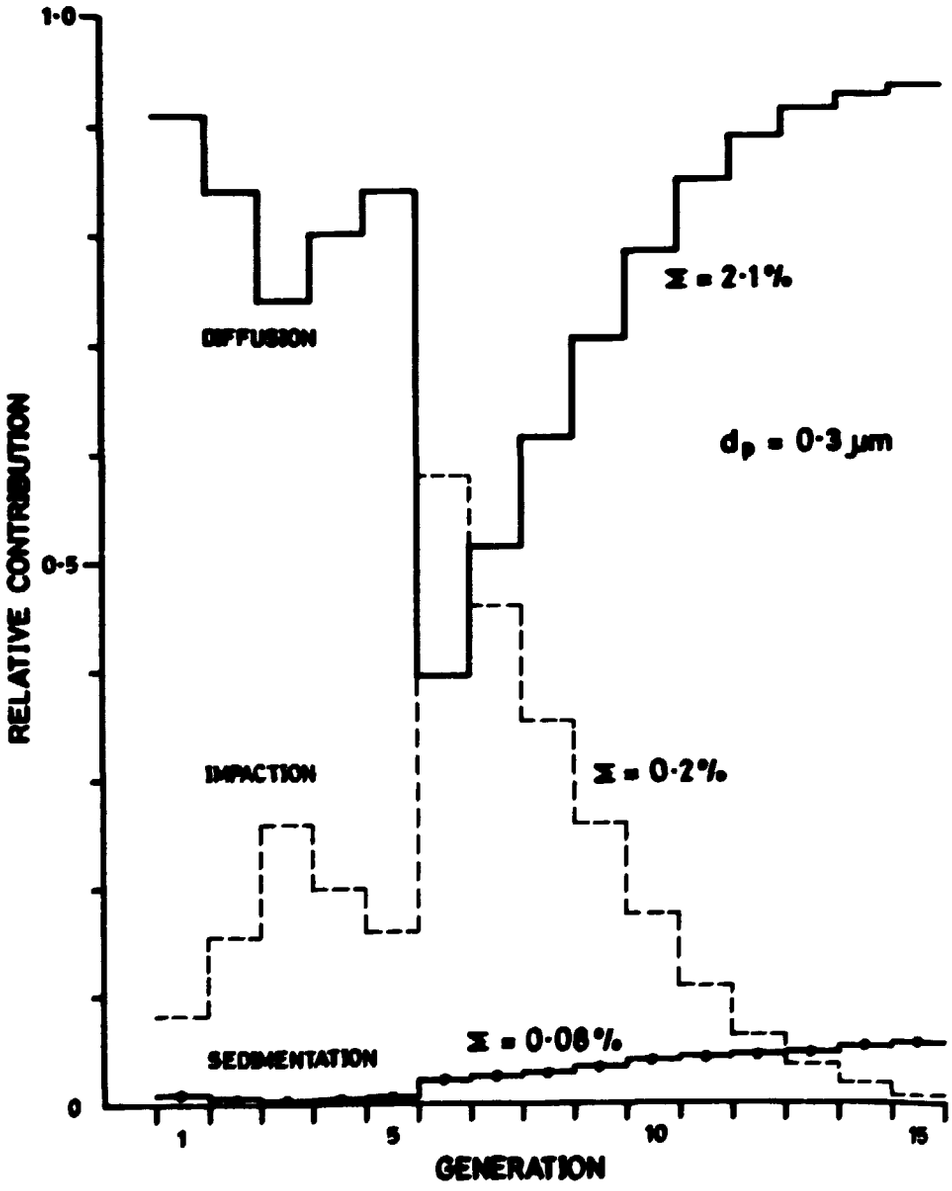

Figure 6. Relative contributions to the deposit of 0.3 μm diameter in each generation of the tracheobronchial region by the impaction, sedimentation and diffusion mechanisms. The percentage of particulates entering the trachea depositing in the tracheobronchial region is also shown.

Unattached radon daughters, however, with their diffusion coefficient of 0.054 cm^2 s^{-1}, which is four orders of magnitude higher than for particles, deposit very efficiently in the lungs - more than 50% is lost in the nasopharynx - the rest deposits in the upper respiratory tract, and none reach the lower respiratory tract. Fig. 7 gives estimates for the fraction of unattached and attached daughters deposited in different parts of the lung for several rates of inhalation. It is clear that unattached daughters are deposited more efficiently in the upper airways by some 2 orders of magnitude. It would appear that the inhalation rate does not affect the deposition greatly.

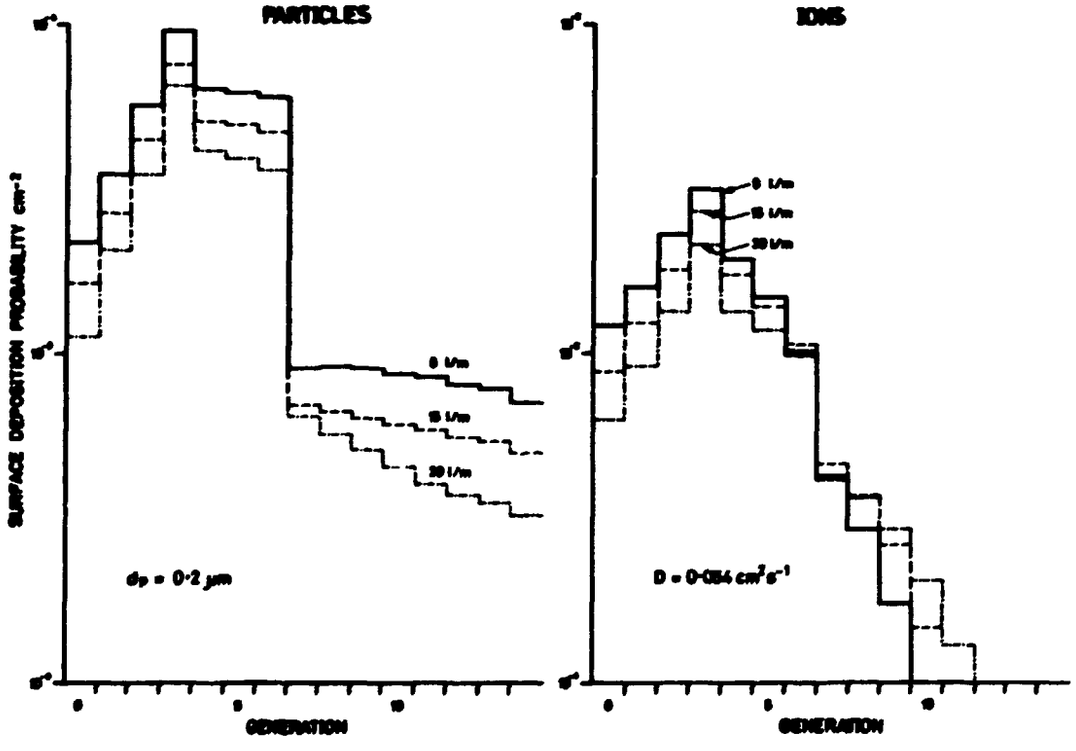

Figure 7. Deposition probabilities for each generation of the tracheobronchial region for 0.2 μm diameter particles and ions with a diffusion coefficient of 0.054 cm2 5-1.

One of the defence mechanisms of the lung against the invasion of material is for any deposited particulates on the lung airways to be moved upwards by a mucus stream towards the throat where it is swallowed. The speed of this mucus escalator varies from 1 cm min^{-1} at the trachea to less than 0.01 cm min^{-1} at the intrasegmental brochi. However, rates are rather variable even in an individual. In the alveolar region other removal mechanisms are active. Overall the clearance times can range from hours to days depending on where the material is deposited.

Obviously, radioactive material depositing on the surface of the lung airways give rise to a radiation dose to the tissue below. Some workers have suggested that the basal cells are the point of origin of lung cancer. The average depth of these basal cells varies from 80 μm at the main bronchi to 20 μm in the transitional bronchioles (Gastineau et al 1972); the standard deviation of the depth of the cells is of the order of 10 μm. Before the availability of this information on the depth of the cells, various authors used a number of depths. For example, Altshuler et al (1964) used 36 μm whereas Harley and Pasternak (1972) have used 22 μm for the depth of the cells. As the ranges of alpha particles from ^{218}Po and ^{214}Po are 47 μm and 71 μm, respectively, small changes in the depth of the basal cells used in the models change the estimates of the dose significantly. Harley and Pasternak have indicated that changing the depth of the basal cells by 5 μm changes the estimated dose by some 25%. The depth of the basal cells is thus a crucial parameter in the model. Therefore, it is not surprising that estimates of the dose to sensitive cells have varied over a large range due to differences in the depth of the basal cells chosen. Table 5 presents estimates for the dose to sensitive cells as made by different workers. These estimates should be contrasted with 14 mGy/WLM as deduced by Walsh (1979) from epidemiological data. Walsh also estimated that the Gray to Sieverts conversion factor is between 3 and 4 (ICRP currently recommend that this factor, called a quality factor, be 20). UNSCEAR (1977) recently reviewed the literature and suggested a conversion factor of 10 mGy WLM^{-1}. It would appear, however, that the use of a conversion factor of around 10 mGy WLM^{-1} rests on the premise that the unattached fraction is small and in modern mines rarely rises above 10%. This is supported by data from underground mines.

The measurements in the Northern Territory described above indicates that the unattached fraction may not always be low in some environments. Given the efficiency with which unattached daughters deposit on the lung airways this implies that in some environments the dose to lung could be higher than underground mining experience would suggest. Calculations based on the Weibel model show that the maximum dose from exposure to radon daughters occurs in the segmental region (where cancers do occur) and the dose ranges from 8 mGy WLM^{-1} to 30 mGy WLM^{-1} when the fraction of unattached ^{218}Po ranges from 0.02 to 0.10, as in underground mines and in the New York observations. For the conditions experienced at Ranger Uranium Mines 1 WLM of radon daughters would give rise to a maximum dose of 60 mGy to 120 mGy. Clearly more data is needed on the unattached fraction before conversion factors suitable for use in open-cut mines can be established.

Table 5

Summary of dose calculations published since 1964 for radon and radon daughters.

Investigation		Tissue	Calculated Dose mGy/WLM
Altshuler et al	1964	Segmented bronchi	30
Jacobi	1964	Secondary-quarternary bronchioles	29
Haque and Collinson	1967	Segmented bronchi	20
Walsh	1970	Bronchial epithelium	<10
Harley and Pasternak	1972	Segmented bronchi	2-3
Walsh	1979	Bronchial epithelium	14[a]
James	1980	Bronchioles	7-20
Wise	1980	Segmented bronchi	8-40

Note (a) Estimated from epidemiological data

PART 2 : URANIUM AND DAUGHTERS

We normally ingest some uranium with our food although environmental levels are negligibly small. UNSCEAR (1977) estimate that we ingest between 10 and 20 mBq/day of ^{238}U, leading to a body burden of about 0.2 Bq/kg and a dose of 3 μGy per year.

In a uranium mine, however, inhalation is the principle mode of entry of uranium ore dust into the body. To estimate the limits of intake for uranium and daughters, the approach adopted by the ICRP is to combine a very simple model for estimating the deposit of particulates in the lung (ICRP 1966) and a model of the gastro-intestinal tract (Eve 1966) with the latest available data on the rates of turnover of elements from blood and organs (Adams 1978, ICRP 1979). This provides a complete system from which the levels of radionuclides can be computed. A knowledge of the type and quantity of radiation emitted by these radionuclides then allows a computation to be made. Hence limits of intake can be established to ensure that the limits on the dose received are not exceeded.

The limits on exposure to radionuclides in air and water imbedded in the current code of practice can be traced back to an early standard (ICRP 1959). The Commission is replacing this standard with one which follows the precepts laid down in ICRP26 (1977). The first volumes of the new standard appeared

late in 1979 as ICRP30 parts 1 and 2 together with supplements; it is concerned only with the derivation of secondary standards that limit intake of radionuclides by workers. The limits are known as Annual Limits of Intake (ALI) either for ingestion or for inhalation. No standard is developed for water because water is only one source of ingested material; the total activity ingested in any one year should be controlled by use of ALI for ingestion. However, values are given for the Derived Air Concentration (DAC) which is obtained by dividing ALI (inhalation) by volume inhaled by Reference Man (ICRP, 1975) in a working year. It will be some years before the new methods and the resulting limits find their way into the regulations. However, the models which form the basis of these recent calculations are described briefly here to provide a frame of reference for an understanding of the changes that will take place in the future. Obviously, too, a discussion of the models highlights those factors which are important in determining the hazards from ingested or inhaled radionuclides.

ICRP Lung Model

This model was adopted by ICRP Committee 2 for radiation protection purposes and not for radiological studies. The model gives a simple description of the deposition and retention of radioactive particulates in the lung.

For this purpose the lung is divided into 3 major compartments:

- the nasopharynx compartment (N-P) which extends from the outside opening of the nose to the level of the larynx

- the tracheobronchial compartment (T-B) which consists of the trachea and the bronchial tree down to the terminal bronchioles. This region contains the entire ciliated tissue of the respiratory tract which is covered by a thin layer of mucus. The mucus is moved upward by the waving cilia

- the pulmonary compartment (P) which contains the airways from which exchange into the blood takes place.

In calculating the deposit of material in the lung, the committee focussed on 3 different ventilation rates representing the volume of air inhaled at 3 different levels of work; the ventilation frequency was fixed at 15 breaths per minute. From detailed calculations for inhaled particles of various aerodynamic diameters and of various geometric standard deviations, Committee 2 showed:

- a mean curve, ventilation rate 20 l/min, can be considered to represent the rates chosen; hence, altnough volume breathed per minute affects the total amount of dust inhaled, the fraction deposited in each compartment is not altered significantly

- for regions N-P and P, the fraction of different particle sizes deposited is linear when plotted on log-normal probability paper (see Fig. 8) while in region T-B the fraction deposited is approximately constant at 0.08 for all median diameters larger than 0.05 μm

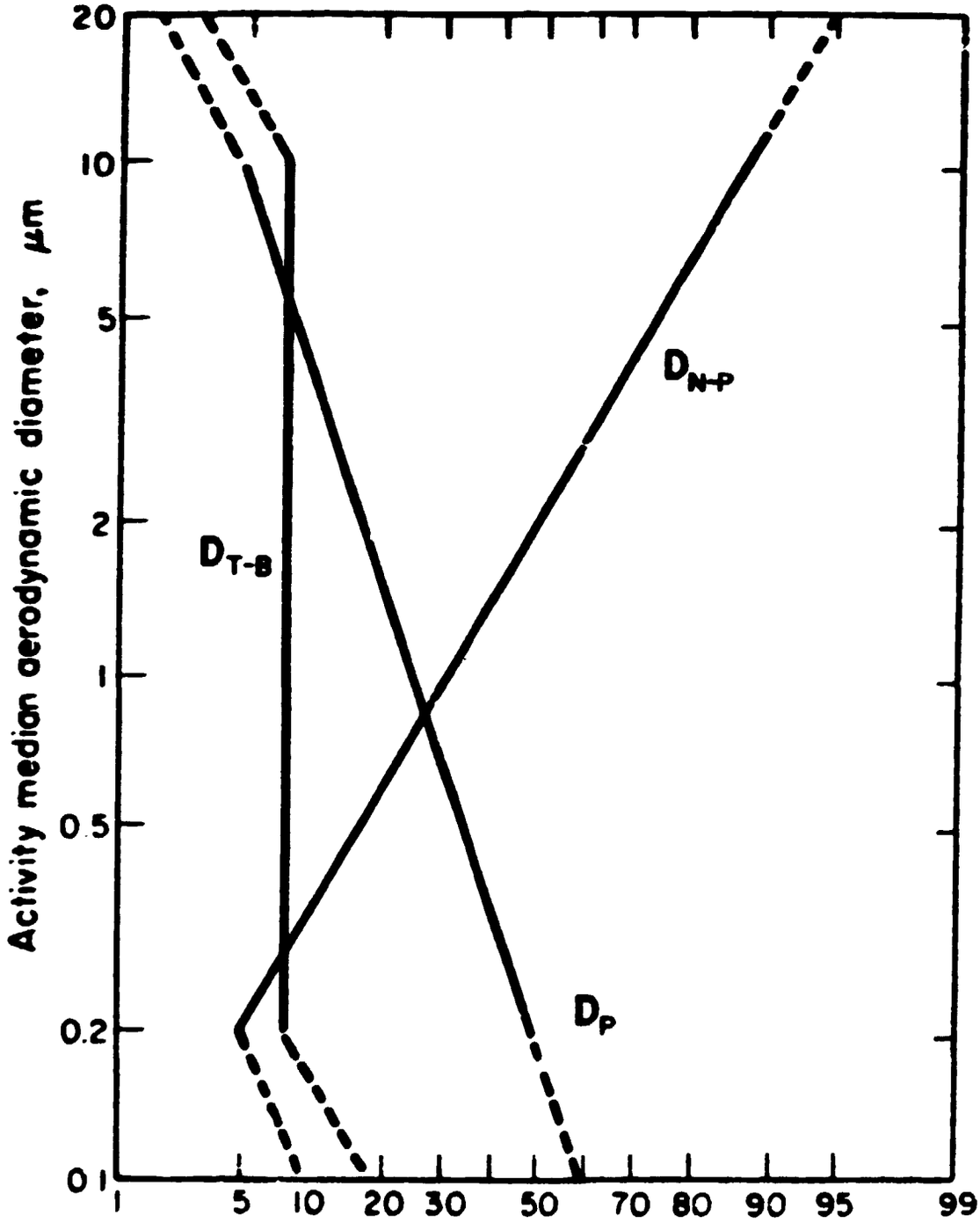

Figure 8. ICRP (1966) estimates of the percentage of inhaled aerosol deposited in each of 3 regions of the lung.

Uptake of radionuclides

Clearance of deposited material can be by transport directly to the blood or its upward movement towards the throat where it is swallowed and thus enters the GI tract.

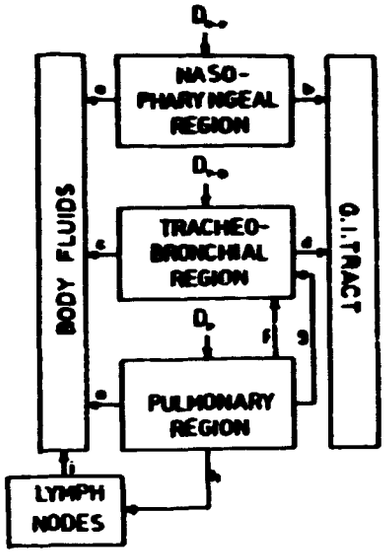

Figure 9. Compartments used in the ICRP model for calculating dose to tissue from inhaled radionuclides.

The pathways are shown diagrammatically in Fig. 9 and the fraction of material affected and the rate at which clearance takes place by each pathway are given in Table 6.

Table 6
Parameters for ICRP lung clearance model

Region	Pathway	Class D T	Class D F	Class W T	Class W F	Class Y T	Class Y F
N - P	a	0.01	0.5	0.01	0.1	0.01	0.01
(D_{N-P} = 0.30)	b	0.01	0.5	0.40	0.9	0.40	0.99
T - B	c	0.01	0.95	0.01	0.5	0.01	0.01
(D_{T-B} = 0.08)	d	0.2	0.05	0.2	0.5	0.2	0.99
	e	0.5	0.8	50	0.15	500	0.05
P	f	-	-	1.0	0.4	1.0	0.4
(D_p = 0.25)	g	-	-	50	0.4	500	0.4
	h	0.5	0.2	50	0.05	500	0.15
L	i	0.5	1.0	50	1.0	1000	0.9
	j	-	-	-	-		0.1

Notes: (1) T is the clearance half-time in reciprocal days.

(2) F is the fraction of material deposited of each region of the compartments N-P, T-B, and P.

(3) D_{N-P}, D_{T-B} and D_p are the fractions of material deposited in each region; they are for an aerosol with AMAD = 1 μm.

Deposited radionuclides are divided into 3 classes (1) Y for avid retention (biological half-lives over 100 days), (2) W for moderate retention (biological half-lives between 10 and 100 days) and (3) D for minimal retention (biological half-lives less than 10 days). Data on the clearance classifications for uranium and daughters are given in Table 7.

Table 7

Clearance classifications for uranium and daughters and the fraction transferred from small intestine to the body fluids.

Radionuclides	Lung Clearance Class	Compound	Fraction transferred to blood from SI f_1
Pb-210 to Pb-212	D	All compounds	0.1
Bi-210	D	Bismuth nitrate	0.05
	W	All other compounds	0.05
Po-210	W	Oxides hydroxides and nitrates	0.1
	D	All other compounds	0.1
Ra-223 to Ra-228	W	All compounds	0.2
Ac-225 to Ac-227	W	All compounds	0.0003
Th-227 to Th-234	Y	Oxides, hydroxides	0.0002
	W	All other compounds	0.0002
Pa-231 to Pa-233	W	All compounds	0.01
U-232 to U-238	D	Most hexavalent compounds	0.05
	Y	Most tetravalent compounds	0.05
	Y	UO_2, U_3O_8	0.002

Sources: Adams (1978), ICRP (1979).

Once transferred to the GI tract, the radionuclides move in turn from the stomach to small intestine, to upper large intestine, to lower large intestine before final excretion. A fraction, f_1, of the radionuclides is transferred into the blood at the small intestine. Table 8 gives estimates for the mean residence times in each region of the GI tract. Table 7 gives some estimates for f_1.

Table 8

Parameters for gastrointestinal tract model

Section of GI tract	Mass of contents (g)	Mean residence time (days)	λ $days^{-1}$
Stomach (ST)	250	1/24	24
Small intestine (SI)	400	4/24	6
Upper large intestine (ULI)	220	13/24	1.8
Lower large intestine (LLI)	135	24/24	1

Radionuclides reaching the blood can be taken up by specific organs. Radium for example, has long been known as a bone-seeking radionuclide. The radionuclide taken up by the organ can be lost from the organ at a rate characteristic of the element. Table 9 gives the fraction of some uranium daughter radionuclides transferred to various organs.

Table 9

Fraction of uranium and daughter nuclides transferred to source tissue.

Element	Bone	Liver	Kidneys	Spleen	Testes	Ovaries	Rest of Body
Lead	0.55	0.25	0.02				0.18
Bismuth			0.4				0.3
Polonium		0.1	0.1	0.1			0.7
Radium	0.46[a]						0.54
Actinium	0.2	0.6		0.05			0.15
Thorium	0.7	0.04					0.16
Protactinium	0.45	0.45			0.00035	0.00011	
Uranium	0.22		0.12				0.12

Notes: (a) 0.23 to cortical bone; 0.23 to trabecular bone

Sources: Adams (1978), ICRP (1979).

Table 10 gives the fraction of the radionuclide retained by time t in terms of the sum of several exponentials.

Table 10

Retention of stable elemental form in source organ as a function of time [a]

Element	Organ	A_1	T_1 days	A_2	T_2 days	A_3	T_3 days	A_4	T_4 days	A_5	T_5 days
Lead	Bone	0.6	12	0.15	180	0.25	12000				
	Rest	0.8	12	0.18	180	0.02	12000				
Bismuth		0.6	0.6	0.4	5						
Polonium		1.0	50								
Radium	Tr. Bone	0.53	0.023	0.44	3.6	0.0175	800	0.0175	3500		
	Cort. Bone	0.52	0.023	0.43	3.6	0.026	1300	0.026	9600		
	Rest	0.16	0.05	0.54	1.0	0.11	35	0.046	200	0.009	1400
Actinium		1.0	3500								
Thorium	Bone	1.0	8000								
	Rest	1.0	700								
Protactinium	Bone	1.0	100Y								
	Liver	1.0	40Y								
	Gonads	1.0	∞								
Uranium	Bone	0.90	20	0.10	5000						
	Rest	0.996	6	0.004	1500						

Note: (a) The retention function R(t) is $R(t) = \sum_i A_i \exp(-0.693\, t/T_i)$

Sources: Adams (1978), ICRP (1979).

Calculation of limits of intake

ICRP currently distinguishes two broad categories of radiation induced effects, namely:

- malignant and heriditary disease for which the probability of an effect occurring, rather than its severity, is regarded as a function of dose without threshold (stochastic effects) and
- effects such as opacity of the lens and cosmetically unacceptable changes for which a threshold or pseudo-threshold of dose must be exceeded before the effect is induced (non-stochastic effects).

To meet these basic limits for exposure of workers, the intake of the radionuclide in a year must satisfy

$$\sum_T W_T H_{50,T} < 0.05 \text{ Sv}$$

$$H_{50,T} < 0.50 \text{ Sv}$$

where $H_{50,T}$ is the total dose equivalent averaged throughout a specific tissue in the 50 years after intake of the radionuclide

and w_T is a weighting factor which is the ratio of the stochastic risk arising from tissue T to the total risk when the whole body is uniformly irradiated. The weighting factors are given in Table 11.

Table 11
Weighting factors recommended by ICRP for stochastic risks.

Organ or tissue	W_T
Gonads	0.25
Breast	0.15
Red bone marrow	0.12
Lung	0.12
Thyroid	0.03
Bone surfaces	0.03
Remainder [a]	0.30

Note: (a) W_T is 0.06 for each of the five of the remaining organs or tissues receiving the greatest dose equivalents.

In the terminology used by ICRP, tissue is the target organ and the dose equivalent for each type of radiation is computed from the number of transformations of the radionuclide which take place in a source organ S and the energy absorbed per g in T, modified for a quality factor Q appropriate to the type of radiation. Any daughter radionuclides produced in the body after intake of the radionuclide are also taken into account.

The annual limit of intake, a secondary limit designed to meet the basic limits for occupational exposure recommended by ICRP, is the greatest value of I which satisfies

$$I \sum_T w_T \ (H_{50,T} \text{ per unit intake}) < 0.05 \ \text{Sv}$$

and

$$I \ (H_{50,T} \text{ per unit intake}) < 0.50 \ \text{Sv}$$

If the first criterion is satisfied then ALI is said to be determined by a stochastic limit; if the second criterion is satisfied the ALI is determined by a non-stochastic limit. In the tables of ALI presented by ICRP, if the ALI is determined by the non-stochastic limit in a particular organ or tissue, that organ or tissue is listed beneath the value of ALI. The greatest value of ALI that satisfies the Commission's recommendation for limiting stochastic effects is listed in parenthesis. However, when the ALI is determined by the stochastic limit no organ is listed below the value of ALI. An example of a tabulation of ALI is given in Table 12.

A couple of notes of caution should be sounded on the use of these tables. Firstly ALIs for the inhalation route assume that the particles inhaled are 1 μm diameter. Secondly the ALIs are based on the radiological risk to the occupationally exposed; toxicity of the radioisotopes inhaled or ingested is not considered.

Table 12

Annual limits of intake, ALI(Bq), and derived air concentrations, DAC(Bq/m^3), for uranium isotopes calculated on the basis of exposure for 40 hours per week.

Radionuclide		Oral		Inhalation		
				Class D	Class W	Class Y
		$f_1 = 5 \times 10^{-2}$	$f_1 = 2 \times 10^{-3}$	$f_1 = 5 \times 10^{-2}$	$f_1 = 5 \times 10^{-2}$	$f_1 = 2 \times 10^{-3}$
^{230}U	ALI	1×10^5 (2×10^5) Bone surf.	2×10^6	2×10^4 (2×10^4) Bone surf.	1×10^6	1×10^6
	DAC	—	—	6×10^0	5×10^0	4×10^0
^{231}U	ALI	2×10^8 (2×10^8) LLI Wall	2×10^8 (2×10^8) LLI Wall	3×10^8	2×10^8	2×10^8
	DAC	—	—	1×10^3	9×10^4	7×10^4
^{232}U	ALI	8×10^4 (1×10^5) Bone surf.	2×10^6 (3×10^6) Bone surf.	8×10^3 (2×10^4) Bone surf.	1×10^4	3×10^2
	DAC	—	—	3×10^0	6×10^0	1×10^{-1}
^{233}U	ALI	4×10^5 (7×10^5) Bone surf.	7×10^6	4×10^4 (7×10^4) Bone surf.	3×10^4	1×10^3
	DAC	—	—	2×10^1	1×10^1	6×10^{-1}
^{234}U	ALI	4×10^5 (7×10^5) Bone surf.	7×10^6 —	5×10^4 (7×10^4) Bone surf.	3×10^4	1×10^3
	DAC	—	—	2×10^1	1×10^1	6×10^{-1}
^{235}U	ALI	5×10^5 (7×10^5) Bone surf.	7×10^6	5×10^4 (7×10^4) Bone surf.	3×10^4	2×10^3
	DAC	—	—	2×10^1	1×10^1	6×10^{-1}
^{236}U	ALI	5×10^5 (7×10^5) Bone surf.	8×10^6	5×10^4 (7×10^4) Bone surf.	3×10^4	1×10^3
	DAC	—	—	2×10^1	1×10^1	6×10^{-1}
^{237}U	ALI	6×10^7 (7×10^7) LLI Wall	6×10^7 (7×10^7) LLI Wall	1×10^8	6×10^7	6×10^7
	DAC	—	—	4×10^4	3×10^4	2×10^4
^{238}U	ALI	5×10^5 (8×10^5) Bone surf.	8×10^6	5×10^4 (8×10^4) Bone surf.	3×10^4	2×10^3
	DAC	—	—	2×10^1	1×10^1	7×10^{-1}
^{239}U	ALI	2×10^9	2×10^9	7×10^9	6×10^9	6×10^9
	DAC	—	—	3×10^6	3×10^6	2×10^6
^{240}U	ALI	5×10^7	5×10^7	1×10^8	1×10^8	9×10^7
	DAC	—	—	6×10^4	4×10^4	4×10^4

APPENDIX A

Method of mixtures applied to uranium and daughters.

ICRP2 (1959, p24) has this to say about the calculation of maximum permissible concentrations of mixtures of radionuclides.

8. Maximum permissible concentration of known mixtures of radionuclides. Suppose a person is exposed to concentrations $\rho_{aA}, \rho_{aB}, \ldots \rho_{wA}, \rho_{wB} \ldots \mu c/cm^3$ of isotopes $A, B, \ldots$ in air and in water, respectively, and also to external sources of γ and neutron radiations. Assume further that the external sources give doses R_γ^x, R_n^x to a given organ x for γ and neutron radiation, respectively. If L^x rem is the average weekly dose permitted to organ x by the basic rules, then the total dose to organ x is

$$\left[\frac{\rho_{aA}}{(\mathrm{MPC})_{aA}^x}+\frac{\rho_{aB}}{(\mathrm{MPC})_{aB}^x}+\ldots+\frac{\rho_{wA}}{(\mathrm{MPC})_{wA}^x}+\frac{\rho_{wB}}{(\mathrm{MPC})_{wB}^x}+\ldots\right]L^x+R_\gamma^x+R_n^x \quad (22)$$

This does not exceed L^x provided

$$\frac{\rho_{aA}}{(\mathrm{MPC})_{aA}^x}+\frac{\rho_{aB}}{(\mathrm{MPC})_{aB}^x}+\ldots+\frac{\rho_{wA}}{(\mathrm{MPC})_{wA}^x}+\frac{\rho_{wB}}{(\mathrm{MPC})_{wB}^x}+\ldots+\frac{R_\gamma^x}{L^x}+\frac{R_n^x}{L^x}\leqslant 1 \quad (23)$$

and thus provides a criterion for assessing whether or not the exposure is in excess of that permitted by the basic rules. If organ x is not listed as an organ of reference in Table 1, and if an independent estimate of the corresponding MPC values is not available, the MPC based on total body may be used with the correction factor $L^x/0.1$, i.e. $L^x(\mathrm{MPC})_a^{T.B.}/0.1$ may be substituted for $(\mathrm{MPC})_a^x$ in such cases. In general it will be necessary to calculate the dose for all the organs for which the dose may reasonably be considered to be in excess of the prescribed limits. Often this may include the total body even though no one of the radionuclides irradiates a major portion of the body. Assuming that a major portion of the body is being irradiated at somewhat comparable rates, the calculation is essentially as before except that the MPC values based on total body are to be used. Thus the criterion is

$$\frac{\rho_{aA}}{(\mathrm{MPC})_{aA}^{T.B.}}+\frac{\rho_{aB}}{(\mathrm{MPC})_{aB}^{T.B.}}+\ldots+\frac{\rho_{wA}}{(\mathrm{MPC})_{wA}^{T.B.}}+$$

$$\frac{\rho_{wB}}{(\mathrm{MPC})_{wB}^{T.B.}}+\ldots+\frac{R_\gamma^{T.B.}}{0.1}+\frac{R_n^{T.B.}}{0.1}\leqslant 1 \quad (24)$$

Note that the calculation requires the contributions to the dose from inhalation or ingestion of radionuclides or from irradiation by gamma rays or neutrons must be considered. Further, the calculation is done for each organ in turn; the calculation yielding the lowest concentration of the mixture is the maximum permissible for that mixture.

Table A summarizes the calculation of maximum permissible concentrations in air for members of the uranium -238 and uranium -235 series is secular equilibrium. The calculation has been simplified by ignoring any contribution to organ doses from ingestion of radionuclides and from gamma and neutron radiation.

TABLE A. Application of the method of mixtures[1] to occupational maximum permissible concentrations in air[2] of members of ^{238}U and ^{235}U series in secular equilibrium[3]

Nuclide	Nuclides soluble			Nuclides insoluble		
	Critical organ	M_{kb} Ci/m^3		Critical organ	M_{ke} Ci/m^3	
^{238}U series						
^{238}U	kidney	6	-10	lung	1	-10
^{234}Th	bone	6	-08	lung	3	-08
^{234}U	bone	6	-10	lung	1	-10
^{230}Th	bone	2	-12	lung	1	-11
^{226}Ra	bone	3	-11	(lung	5	-11)
^{210}Pb	kidney	2	-10	lung	2	-10
^{210}Po	spleen/kidney	7	-09	lung	2	-10
^{210}Bi	kidney	1	-06	lung	6	-09
^{235}U series						
^{235}U	kidney	6	-10	lung	1	-10
^{231}Th	GI	1	-05	GI	6	-06
^{231}Pa	bone	1	-12	lung	1	-10
^{227}Ac	bone	2	-12	lung	3	-11
^{227}Th	bone	3	-10	lung	2	-10
^{223}Ra	bone	2	-09	lung	2	-10
U_{equil}[3] [4]	for bone 1.64 pCi/m^3			for lung 6.53 pCi/m^3		

(1) The procedure for method of mixtures is described in N.Z. Mines (Radiation Protection) Regulations; application to the calculation of U_{equil} assumes that external radiation dose may be ignored.

(2) The occupational maximum permissible concentrations in air are taken from ICRP Publication 2 (1959) and ICRP Publication 6 (1964).

(3) U_{equil} pCi/m^3 is the concentration of ^{238}U in air, together with ^{235}U and all of their daughters in secular equilibrium (excluding $^{219,222}Rn$ and their short-lived daughters) continuous exposure to which would lead to occupational maximum permissible dose D to the critical organ. Ratio of activities, $^{235}U/^{238}U$, is .0450 (identical .00711% by mass). For nuclide k, M_k Ci/m^3 ($\mu Ci/cm^3$) is its occupational maximum permissible concentration in air, becoming M_{kb} where bone is the critical organ and M_{ke} for lung. Setting k=i for ^{238}U series and k=j for ^{235}U series,

$$D = \sum_i \frac{U_{equil}}{M_i} D.10^{-12} + \sum_j \frac{.0450\ U_{equil}}{M_j} D.10^{-12}$$

from which

$$U_{equil} = \frac{10^{12}}{\sum_i \frac{1}{M_i} + \sum_j \frac{.0450}{M_j}}\ pCi/m^3$$

(4) U_{equil} = 3.40 pCi/m^3 for the kidney as the critical organ with all nuclides soluble.

ICRP 24 calculates the limits on the total alpha activity from the activities of the insoluble radionuclides. Five of the radionuclides in the uranium-238 series are alpha emitters. The total alpha activity per unit volume is therefore 5 x 6.53 pCi/m^3 or 1.2 Bq/m^3. Also the annual operational limit for alpha activity in ore dust is

$$L = 5 \times 6.53 \frac{pCi}{m^3} \times 170 \frac{hours}{month} \times 12 \text{ months} \times \frac{1}{1000} \frac{m^3}{l}$$

$$= 67 \text{ pCi.h/l}$$

ICRP 24 gives this limit to one significant figure as 70 pCi h/l.

REFERENCES

Adams N., Hunt B.W. and Reissland J. A. (1978) Annual limits of intake of radionuclides for workers. National Radiological Protection Board Report NRPB-R82.

Altshuler B., Nelson N. and Kuschner M. (1964) Estimation of lung tissue dose from the inhalation of radon daughters, Health Phys. 10, 1137.

Bricard J. (1977) Aerosol production in the atmosphere, in J.O'M. Bockris (ed.) Environmental Chemistry, Plenum Press, New York. p 313-30.

Craft B.F., Oser J.L. and Norris F.W. (1966) A method for determining relative amounts of combined and incombined daughter activity in underground uranium mines. Amer. Ind. Hyg. Assoc. J. 27, 154.

Eve I.S. (1964) Some suggested maximum permissible single intakes of uranium. Health Phys. 10, 773.

Eve I.S. (1966) A review of the physiology of the gastrointestinal tract in relation to radiation doses from radioactive materials. Health Phys. 12, 131.

George A.C. and Hinchliffe L. (1972) Measurements of uncombined radon daughters in uranium mines. Health Physics 23, 791-803.

Haque A.K.M.M. and Collinson A.J.L. (1967) Radiation doses to the respiratory system due to radon and its daughter products, Health Phys. 13, 431.

Harley N.H. and Pasternak B.S. (1972) Alpha absorption measurements applied to lung dose calculations from radon daughters. Health Phys. 23, 771.

Horsfield K. and Cumming G. (1968) Morphology of the bronchial tree in man. J. Appl. Physiol. 24, 373.

IAEA (1978) Particle size analysis in estimating the significance of airborne contamination. IAEA Technical Report Series No. 179. International Atomic Energy Agency, Vienna.

ICRP (1959) Report of Committee II on permissible dose for internal radiation. ICRP Publication 2. Pergamon Press, New York.

ICRP Task Group on Lung Dynamics (1966) Deposition and retention models for internal dosimetry of the human respiratory tract. Health Phys. 12, 173.

ICRP (1975) Report of the Task Group on Reference Man. ICRP Publication 23. Pergamon Press, New York.

ICRP (1977) Recommendations of the International Commission on Radiological Protection. ICRP Publication 26. Annals of the ICRP 1 (3).

ICRP (1979) Limits for intakes of radionuclides by workers. ICRP Publication 30, Part 1 (Annals of the ICRP 2 (3-4)) and supplement (Annals of the ICRP 3 (1-4)).

Jacobi W. (1964) The dose to the human respiratory tract by inhalation of short-lived Rn-222 and Rn-220 decay products. Health Phys. 10, 1163.

Jacobi W. and Eisfield K. (1980) Dose to tissues and effective dose equivalent by inhalation of radon-222, radon-20 another short lived daughters. Gesellschaft fur strahlen - Und Umweltforschung MBH, Institut fur Strahlenschutz, GSF Report S-626.

Junge C.E. (1963) Air Chemistry and Radioactivity. Academic Press, New York.

Kotin P., Courington D. and Falk H.L. (1966) Pathogenesis of cancer in ciliated mucus secreting epithelium. American Review of Respiratory Diseases, 93, 115.

Kruger J. and Nothling J.F. (1979) A comparison of the attachment of the decay products of radon-220 and radon-222 to monodispersed aerosols. J. Aerosol Sci. 10, 571.

Landahl H.D. (1950) On the removal of airborne droplets by the human respiratory tract: I. The Lung. Bull. Math. Biophysics. 12, 43.

Lundin F.E., Wagoner J.K. and Archer V.E. (1971) Radon daughter respiratory cancer quantitative and temporal aspects. National Institute for Occupational Safety and Health Report NIOSH-M-71-1.

Mercer T.T. (1975) Unattached radon decay products in mine air. Health Physics, 28, 158.

Mercer T.T. (1976) The effect of particle size on the escape of recoiling RaB atoms from particulate surfaces. Health Phys, 31, 173.

Porstendorfer J., Wicke A. and Schraub A. (1978) The influence of exhalation, ventilation and deposition processes upon the concentration of radon (^{222}Rn), thoron (^{220}Rn) and their decay products in room air Health Phys. 34, 465.

Raabe O.G. (1969) Concerning the interactions that occur between radon decay products and aerosols. Health Phys 17, 177.

Raabe O.G. (1978) Interactions between radon daughters and aerosols. In Workshop on dosimetry for radon and radon daughters, Oak Ridge National Laboratory April 12-12, 1977. Oak Ridge National Laboratory Report ORNL-5348.

Raghavayya M. and Jones J.H. (1974) A wire-screen filter paper commination for the measurement of fractions of unattached daughters in uranium mines. Health Phys. 36, 417.

Strandeh E. (1979) The influence of variations in the ventilation rate in rooms upon the respiratory dose from inhalation of radon daughters. Phys. Med. Biol. 24, 913.

UNSCEAR (1977) Sources and effects of ionizing radiation. United Nations Scientific Committee on the Effects of Atomic Radiation report to the General Asembly, with annexes. United Nations, New York.

Walsh P.J. (1979) Dose conversion factors for radon daughters. Health Phys. 36, 601.

Weibel E.R. (1963) Morphometry of the human lung. Academic Press, New York.

Wise K.N. (1980) On the radiation dose to lung tissue from radon. Australian Radiation Laboratory Report ARL/TR024.