7 Internal dosimetry of radionuclides

This Chapter reviews the behaviour of radionuclides in the body. It summarises the biokinetic and dosimetric models that have been developed by the International Commission on Radiological Protection (ICRP) for assessing radiation doses, and hence risks, from intakes by different routes, including inhalation and ingestion. These models have been widely accepted around the world for use in radiological protection. They have been incorporated in the European and International Basic Safety Standards as well as in many national regulations and guidance notes around the world. Future developments in this area are also examined. Finally, methods that can be used to assess intakes of radionuclides by direct and indirect monitoring procedures and requirements for dose assessment are summarised.

7.1 Introduction

People may be exposed to radionuclides in a number of ways. They may be taken into the body as a result of occupational exposure or uptake from the environment. They are used extensively in medical diagnosis and treatment as well as in biomedical research. People may also be exposed externally by submersion in a radioactive cloud.

For occupational exposure, the main route of intake is by inhalation, although a fraction of any material deposited in the respiratory system will be transferred to the throat and swallowed, giving the opportunity for absorption in the gastrointestinal tract. Intakes by direct ingestion may occur and some radionuclides may be absorbed through the intact skin. Damage to the intact skin by cuts or other wounds can also result in the entry of radionuclides into the body.

For members of the public, the main route of intake of radionuclides will be by ingestion in food and drinking water although intakes by inhalation may also occur, in particular in the case of accidental releases into the environment. For medical applications the method of administration will depend upon the specific nature of the diagnostic investigation or treatment.

Knowledge of the behaviour of radionuclides in the body is important for assessing radiation doses resulting from intakes or superficial contamination. For occupational and public exposure the calculation of radiation doses provides the basis for controlling exposures to within accepted limits, for assessing the consequences of the presence of radionuclides in the working or natural environment or determining the need for treatment in the case of accidental intakes. In medical situations radiation doses are needed for optimising diagnostic and treatment schedules. In the case of administration of radionuclides for clinical research, for example on the behaviour of radiopharmaceuticals, assessment of radiation doses is needed for estimating risks for ethical considerations.

The radiation dose received by a tissue as a result of the intake of a radionuclide will depend upon a number of factors. These include: the route of intake, the physico-chemical form, its biokinetic behaviour and pathways in the body, organ(s) of accumulation, rate of removal (by physical decay, biological turnover and excretion) and the quality of the emitted radiation (α, β, γ). Biological variation (age, sex, dietary habits etc) will also influence behaviour in the body. Thus the determination of the radiation dose to tissues and the assessment of the possible biological effects resulting from the intake of a particular radionuclide requires a knowledge of all the pertinent physical, physiological, biokinetic and chemical data.

This Chapter reviews the biokinetic behaviour of radionuclides in the body and illustrates the key features through examples. It summarises the models that have been developed by the International Commission on Radiological Protection (ICRP) for assessing doses and hence risks from intakes of radionuclides and examines some future developments in this area. These models have been widely accepted around the world for use in radiological protection. They provide the basis for dose coefficients (doses per unit intakes, Sv Bq^{-1}) for assessing radiation doses from intakes by inhalation and ingestion and have been incorporated in the European and International Basic Safety Standards [96E1, 96I2] as well as in many national regulations and guidance notes around the world. The use of these dose coefficients for assessing the risks from intakes of radionuclides is illustrated.

A recent development by ICRP has been the issue of a report giving dose coefficients for the embryo and foetus following intakes of radionuclides by the mother before or during pregnancy [01I1]. A further report is presently being prepared that will give doses to the newborn child from radionuclides consumed in mothers' milk. The development of the document is summarised. A new dosimetric model for the human alimentary tract is also being prepared. The conceptual basis for this model is reviewed. Finally, methods that can be used to assess intakes of radionuclides by monitoring procedures and the requirements for dose assessment are described in principal; more detailed information is given in Sections 10.3.2 and 10.3.3 of Chapter 10.

7.2 Biokinetics of radionuclides in the body

The principal routes by which radionuclides may enter and move around the body and which must be considered in internal dosimetry are summarised in Figure 7.1. Radionuclides passing through the gastrointestinal tract, or deposited in the air passages of the lungs, in a wound or on the outer layer of skin will irradiate these tissues. Soluble forms of radionuclide(s) that are transportable can readily enter the bloodstream and their subsequent fate depends upon their chemical characteristics. If poorly transportable they will only slowly enter the bloodstream or the lymphatic system. Any insoluble particles entering the systemic circulation will be taken up by the reticuloendothelial cells of the liver, spleen and red bone marrow. Here they may remain for up to the life-span of the individual.

To facilitate calculation of doses to tissues following intakes of radionuclides, the ICRP has developed a number of generalised biokinetic models to describe their movement and behaviour in the body. Specific models were given by ICRP for adult workers in Publication 30 [79I1, 80I1, 80I2, 88I2] to describe the behaviour of radionuclides in the main organs of intake – the lungs and gastrointestinal tract as well as the skin. For radionuclides that have entered the blood and systemic circulation, activity subsequently deposited in tissues was generally assumed to be uniformly distributed throughout them and therefore the radiation dose depends solely on the organ mass and both the physical half-life and the biological half-time of the radionuclide (see Chapters 3 and 4). A specific model was needed for the skeleton, however, because of the morphology of skeletal bone and the heterogeneous distribution of deposited activity. Biokinetic models were also given in the various parts and supplements of Publication 30 to describe the behaviour of radionuclides in the body after their entry into the blood.

More recently, ICRP has provided age-dependent biokinetic models for selected radionuclides in Publications 56, 67, 69, 71 and 72 [89I1, 93I1, 95I1, 95I2, 96I1] and has given dose coefficients (Sv Bq^{-1}) for six ages: 3-month-old infants, 1-, 5-, 10- and 15-year old children and adults. The requirement for age-dependent models and dose coefficients became apparent in the aftermath of the Chernobyl accident when it was realised that, whilst some countries had developed such models there were no models that were generally accepted around the world. Such models are essential for assessing doses to the public from intakes of radionuclides in foods and drinking water, for making comparisons with dose limits and for informing decisions on the acceptability for consumption of foods that may be marketed in many countries. For the development of "age-dependent" models there was a need to include anatomical and physiological information, such as age dependent mass and turnover rate of the skeleton. These, so-called physiologically based models provide a framework in which both human and animal data on the behaviour of radionuclides in the body can be integrated and allow a more realistic approach to the calculation of doses to individuals of different ages. They also have the important advantage that they can take into account excretion and are therefore more appropriate for the interpretation of bioassay data.

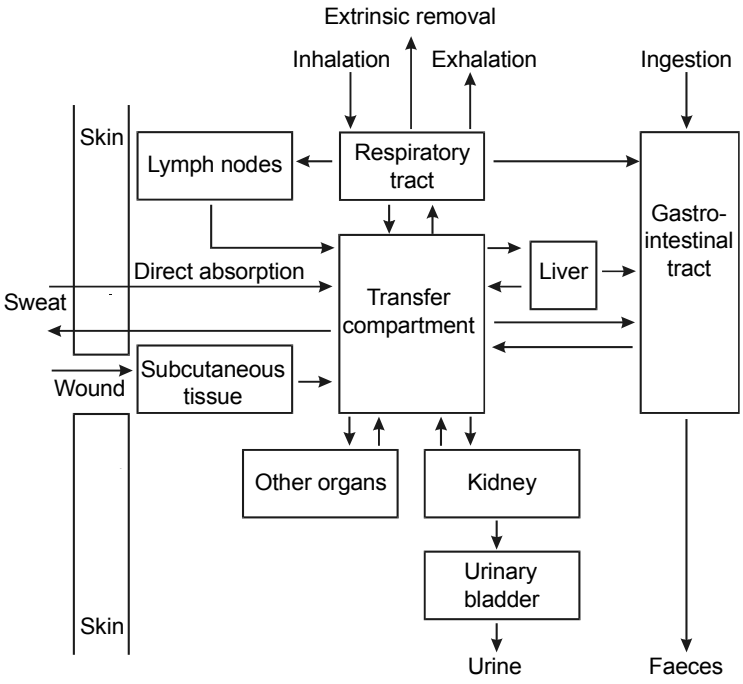


Fig. 7.1. Summary of the main routes in intake, transfers and excretion of radionuclides in the body; [97I2].

In addition to the development of age-dependent biokinetic models a new human respiratory tract model (HRTM) was issued in Publication 66 [94I2]. This model has been applied in all the recent calculations of dose coefficients for workers and the public issued by ICRP (see compilations in 94I1, 96I1, 99I1). Table 7.1 summarises the recent ICRP publications giving revised biokinetic models and dose coefficients.

A further development has been the issue of a report giving dose coefficients for the embryo and foetus following intakes of radionuclides by the mother before or during pregnancy. ICRP Publication 88 gives biokinetic models for 31 elements and also dose coefficients for selected radionuclides [01I1].

Presently being prepared by ICRP is a further report that will give doses to the newborn child from radionuclides consumed in mothers' milk. The development of the document is summarised in Section 7.2.8. A new dosimetric model for the human alimentary tract is also being developed that will also be age-dependent. The conceptual basis for this new model is summarised in Section 7.2.2.

7.2.1 Inhalation

A model for describing the deposition and clearance of inhaled radionuclides in adults who are occupationally exposed was first given in Publication 30 of ICRP [79I1]. This lung model separated the respiratory system into three distinct regions, the naso-pharynx (NP), the tracheo-bronchial region (TB) and the pulmonary region (P). It gave information on the deposition of inhaled radionuclides in these regions as a function of the activity median aerodynamic diameter (AMAD) of inhaled particulates and on the rate of clearance of the material from the respiratory system in terms of three default clearance Classes. These had clearance times from the pulmonary part of the respiratory system of days (Class D), weeks (Class W) and years (Class Y). The default particle size was taken to have an AMAD of 1 μm. Information was also given on the transfer of the three Classes of material to lymphatic tissue associated with the respiratory system. A main feature of this lung model is that it calculated only the average dose to the lungs (TB and P regions). Although the model was developed for adults it has been used for younger ages but without any changes to parameter values, other than organ mass.

Table 7.1 Summary of ICRP reports on dose coefficients for workers and members of the public from intakes of radionuclides.

ICRP Publication No. (year)	Application	Intake	Contents
56 (1989)	Public[a]	Inhalation and ingestion	Age-dependent systemic models, and tissue dose coefficients for selected radioisotopes, for H, C, Sr, Zr, Nb, Ru, I, Cs, Ce, Pu, Am and Np. Issued before ICRP Publication 60 [91I1], and hence giving dose equivalents using the tissue weighting factors from ICRP Publication 26 [77I1]. It was also issued before ICRP Publication 66 [94I2] and hence used the ICRP Publication 30 lung model [79I1]. The dose coefficients given in ICRP Publication 56 were superseded by those in ICRP Publications 67 and 71, which used the tissue weighting factors from Publication 60.
67 (1993)	Public[a]	Ingestion	Age-dependent systemic models, and tissue dose coefficients for selected radioisotopes, for S, Co, Ni, Zn, Mo, Tc, Ag, Te, Ba, Pb, Po and Ra. Updated systemic models are given for Sr, Pu, Am and Np.
68 (1994)	Workers	Inhalation and ingestion	Effective dose coefficients for workers, for about 800 radionuclides: selected radioisotopes of the 91 elements covered in ICRP Publication 30, Parts 1-4 [79I1, 80I1 and I2, 88I2]. The inhalation dose coefficients for workers exposed to ^{226}Ra given in ICRP Publication 68 were revised in Annexe B of ICRP Publication 72. Applies the Human Respiratory Tract Model, HRTM [94I2].
69 (1995)	Public[a]	Ingestion	Age-dependent systemic models, and tissue dose coefficients for selected radioisotopes, for Fe, Sb, Se, Th and U.
71 (1995)	Public[a]	Inhalation	Tissue dose coefficients for selected radioisotopes of elements covered in ICRP Publications 56, 67 and 69, plus Ca and Cm for which age-dependent systemic models are given. Applies the HRTM [94I2].
72 (1996)	Public[a]	Inhalation and ingestion	Effective dose coefficients for members of the public for radioisotopes of the 31 elements covered in ICRP Publications 56, 67, 69, and 71, plus radioisotopes of the further 60 elements covered in ICRP Publications 30 and 68. Applies the HRTM [94I2].
CD-ROM (1998)	Public[a] and workers	Inhalation and ingestion	A database of equivalent doses to individual tissues corresponding to the effective dose coefficients in ICRP Publications 68 and 72. Inhalation dose coefficient for 10 particle sizes.
88 (2001)	Embryo and foetus	Inhalation and ingestion by the mother	Dose coefficients for the offspring for intakes by the mother (worker or public) before or during pregnancy of radionuclides of the 31 elements covered in Publications 68 and 72.
CD-ROM2 (2002)	Embryo and foetus	Inhalation and ingestion by the mother	Database of dose coefficients extending information on radionuclides in Publication 88.

a Age-dependent dose coefficients (3 months, 1-, 5-, 10-, and 15-years and adult)

The Human Respiratory Tract Model (HRTM) described in ICRP Publication 66 [94I2] was developed to replace the lung model given in ICRP Publication 30. It takes into account extensive data on the behaviour of inhaled materials that had become available since the ICRP Publication 30 model was developed. As in the earlier model, *deposition* and *clearance* are treated separately. The scope of the model was extended to apply explicitly to all members of the population, giving reference values for 3-month-old infants, 1-, 5-, 10- and 15-y-old children and male and female adults. The main features of the model are summarised below.

In the new model, the respiratory tract is represented by five regions (Fig. 7.2). The extrathoracic (ET) airways are divided into ET_1, the anterior nasal passage and ET_2, which consists of the posterior nasal and oral passages, the pharynx and larynx. The thoracic regions are bronchial (BB: trachea, generation 0 and bronchi, airway generations 1-8), bronchiolar (bb: airway generations 9-15), and alveolar-interstitial (AI: the gas exchange region). Lymphatic tissue is associated with the extrathoracic and thoracic airways (LN_{ET} and $LN_{TH,}$ respectively). Reference values of dimensions and scaling factors for subjects of different ages are specified in the model. A main feature of the HRTM, compared with the Publication 30 model, is the calculation of doses to these specific tissues in the five regions and allowance for their differences in radiosensitivity.

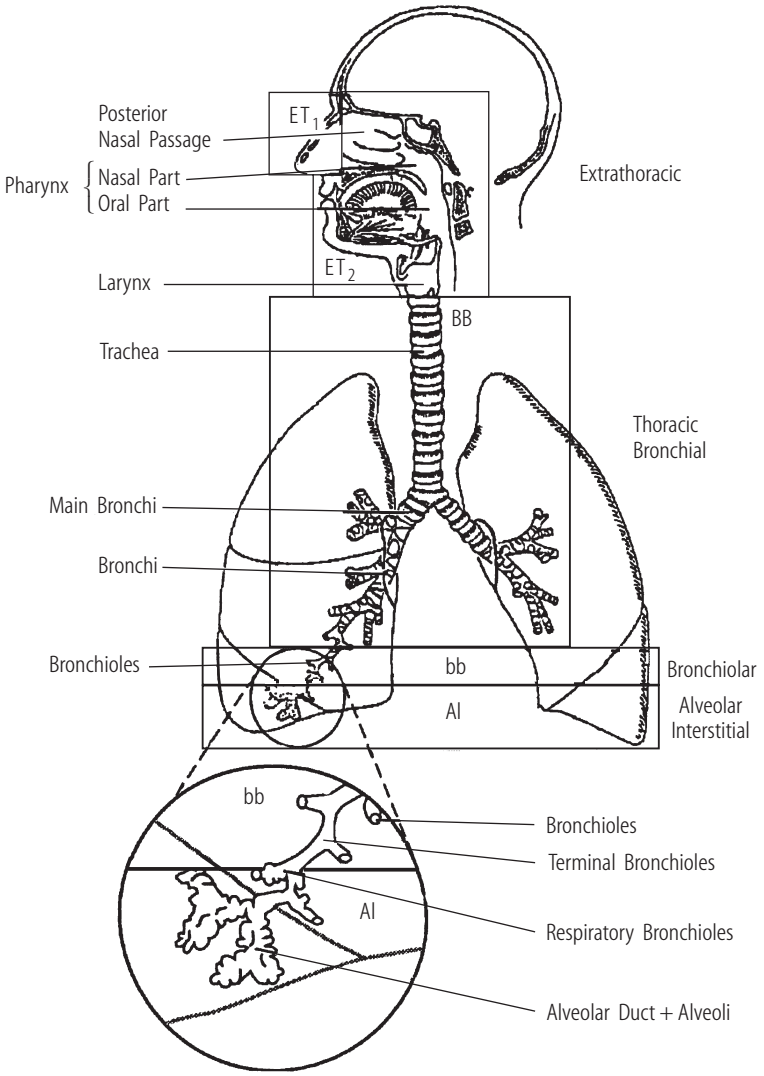


Fig. 7.2. Respiratory tract regions defined in the Human Respiratory Tract Model; [94I2].

7.2.1.1 Deposition

The amount of an aerosol inhaled depends upon breathing parameters and these are influenced by age, body size and level of physical exertion. Deposition of inhaled particles in the HRTM is calculated for each region of the respiratory system, with account taken of both inhalation and exhalation. This is done as a function of particle size, breathing parameters and/or work load, and is assumed to be independent of chemical form. Four standard levels of activity are defined in the HRTM ranging from sleep through to heavy exercise, and the different proportions of time spent at these reference levels are specified for representative individuals at the six standard ages [94I2]. The fraction of inhaled aerosol deposited in the various regions of the lung depends in turn upon the particle size, typically taken as a log-normal distribution. Age dependent default deposition parameters are given for a range of particle sizes from 0.6 nm activity median thermodynamic diameter (AMTD) to 20 μm activity median aerodynamic diameter (AMAD). Previously, a 1 μm AMAD was taken as the default particle size for occupational exposure, but ICRP Publication 66 now recommends 5 μm AMAD as being more typical of the workplace. For members of the public the default is taken as 1 μm AMAD. Table 7.2 compares regional deposition in the respiratory system for the models of ICRP Publications 30 and 66. For the old and new defaults for workers, total deposition is about 30 % higher in the new model (82 % c.f. 63 %), with the extrathoracic region dominating, although a large fraction of this is in the ET_1 region and thus unavailable for systemic uptake. Conversely, deposition in the deep lung (bronchiolar and alveolar-interstitial regions) is a factor of four higher in the ICRP 30 model (25 % c.f. 6.4 %).

Table 7.2 Comparison of regional deposition for ICRP 30 Lung and ICRP 66 Respiratory Tract Models.

Publication 30 model		Publication 66 model			
	Adult		Adult member of public	Worker	
	1 μm[a]		1 μm[a]	1 μm[a]	5 μm[a]
Region	[%]	Region	[%]	[%]	[%]
Nasal passage (NP)	30	Extrathoracic (ET_1)	15	17	34
		Extrathoracic (ET_2)	19	21	40
		Total	(34)	(38)	(74)
Trachea and bronchial tree (TB)	8	Bronchial (BB)	1.3	1.2	1.8
		Bronchiolar (bb)	2.0	1.7	1.1
		Total	(3.3)	(2.9)	(2.9)
Pulmonary region (P)	25	Alveolar-interstitial (AI)	11	11	5.3
Total	63	Total	48	52	82

a Activity Median Aerodynamic Diameter (AMAD)

The variation in deposition parameters between individuals, depending upon age, gender and habits, is an important difference from the Publication 30 model [79I1] for which particle size was the only factor that influenced deposition (see 1 μm entries of Table 7.2). Previously, this distinction was not needed, since the Publication 30 model was intended only for reference adults who were occupationally exposed. In contrast, the ICRP Publication 66 model [94I2] has been designed for application to all members of the population. In the new model, deposition, but not clearance, is strongly influenced by age.

7.2.1.2 Clearance

Subsequent to deposition, material is cleared from the respiratory tract. For material deposited in the anterior nose ET_1, clearance is affected extrinsically by such means as nose-blowing or sneezing. The ET_1 deposit is cleared directly from the body and makes no subsequent contribution to gut or systemic tissue doses. Removal from all other regions is treated as two competing processes: particle transport (by mucociliary clearance to the throat or translocation to lymph nodes) and absorption to blood.

It is assumed that these clearance processes compete independently with each other and have no age or gender dependence. Transport processes include mechanical transport to the gut by mucociliary action and removal by macrophages to the lymph nodes. Particle transport rates are taken to be fixed for all materials and a single compartment model describes clearance by this mechanism (Fig. 7.3). Absorption rates, however, are determined by solubility of inhaled materials and default parameters are recommended for Fast (Type F), Moderate (Type M) and Slow (Type S) absorption. This corresponds roughly to the ICRP Publication 30 classification scheme and chemical forms previously assigned to Class D, W or Y are now provisionally treated as Type F, M or S, respectively. The correspondence between the two schemes is not exact, e.g. the D, W or Y classification refers to whether total pulmonary lung clearance (by absorption to blood or clearance to the throat and then through the gut) is of the order of days, weeks or years, whereas Type F, M or S refers only to the absorption component. The mechanical clearance of the deposited activity is not dependent on the chemical form. The main clearance components for the two models, in the form of approximate biological half-times, are summarised in Table 7.3. Qualitatively, residence times in the lung are reduced, quite drastically, for Type F compared to Class D and elevated for Types M and S relative to Classes W and Y.

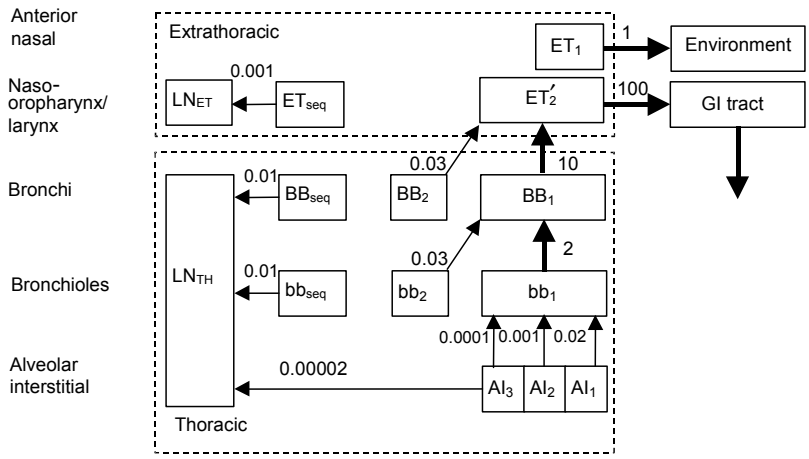


Fig. 7.3 Compartment model representing time-dependent particle transport from each respiratory tract region. Rates shown alongside arrows are reference values in units of d^{-1}. It is assumed that (i) the AI deposit is divided between AI_1, AI_2 and AI_3 in the ratio 0.3:0.6:0.1; (ii) the fraction of the deposit in BB and bb that is cleared slowly (BB_2 and bb_2) is 50 % for particles of physical size <2.5 μm and decreases with diameter >2.5 μm, and the fraction retained in the airway wall (BB_{seq} and bb_{seq}) is 0.7 % at all sizes; (iii) 0.05 % of material deposited in region ET_2 is retained in its wall (ET_{seq}) and the rest in compartment ET_2' which clears rapidly to the GI tract. The model as shown above would describe the retention and clearance of a completely insoluble material. However, there is in general simultaneous absorption to body fluids of material from all the compartments except ET_1; [94I1].

Table 7.3. Comparison of approximate clearance half-times for Publication 30 Lung and Publication 66 Respiratory Tract Model

ICRP 30		ICRP 66	
Class	Pulmonary clearance $T½$	Type	Dominant absorption $T½$
D	0.5 d (100 %)	F	10 min (10 %)
W	50 d (60 %)	M	140 d (90 %)
Y	500 d (60 %)	S	7000 d (99.9 %)

By considering the relative rates of the two independent clearance processes (mechanical and absorption) in the HRTM and the amount absorbed in the gut after clearance to the throat it is possible to calculate the fraction of material ultimately transferred to the blood and systemic circulation, both directly from the lungs and indirectly via the gut. Because particle transport rates are fixed for all lung Types, the proportion of material escalating to the gut increases as the classification changes from Type F to Type S. It is interesting to compare the amounts transferred to the circulation for inhalation of the different Types. This is illustrated in Fig. 7.4, ignoring the effect of radioactive decay. For Type F material, such as soluble (transportable) forms of radioisotopes of caesium and iodine (e.g. ^{137}Cs, ^{131}I) there is rapid translocation to the blood with about 25 % of the intakes being taken up by within a day. In contrast for Type S materials, such as $^{239}PuO_2$, transfer to the blood is much slower with about 0.15 % transferred after 1000 days. Examples of the lung clearance Types adopted for various chemical forms of a selection of radionuclides are given in Table 7.4. Although the HRTM model provides these default clearance Types there is also provision for including material specific absorption parameters when information is available. ICRP has issued a guidance document on this application of the HRTM [02I1].

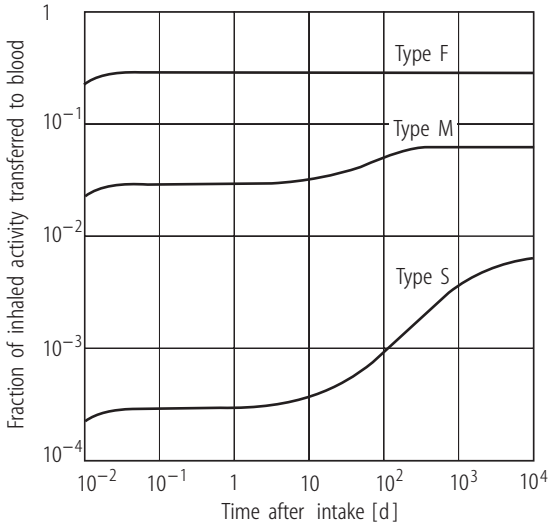


Fig. 7.4. Cumulative fraction of inhaled activity absorbed into blood directly from the respiratory tract as a function of time after intake for each default absorption Type (in the absence of radioactive decay), for a reference worker; [02I1].

Table 7.4 Examples of lung clearance Types adopted by ICRP for workers; [94I1].

Radionuclide	Chemical form	Inhalation Type
Tritium	Tritiated water	SR2[a]
Cobalt	Unspecified compounds,	M[b]
	Oxides, hydroxide, halides and nitrate	S
Strontium	Unspecified compounds	F[b]
	Strontium titanate	S
Zirconium	Unspecified compounds	F
	Oxides, hydroxide, halides and nitrate	M
	Zirconium carbide	S
Niobium	Unspecified compounds	M
	Oxides and hydroxide	S
Ruthenium	Unspecified compounds	F
	Halides	M[b]
	Oxides and hydroxides	S
Iodine	All compounds	F[b]
	Vapour	V[b]
Caesium	All compounds	F[b]
Cerium	Unspecified compounds	M[b]
	Oxides, hydroxides and fluorides	S
Polonium	Unspecified compounds	F
	Oxides, hydroxides and nitrate	M
Radium	All compounds	M
Thorium	Unspecified compounds	M
	Oxide and hydroxides	S
Uranium	Most hexavalent compounds, e.g. UFO_6, UO_2F_2 and $UO_2(NO_3)_2$	F
	Less soluble compounds, e.g. UO_3, UF_4, UCI_4 and most other hexavalent compounds	M[b]
	Highly soluble compounds, e.g. UO_2, U_3O_8	S
Plutonium	Unspecified compounds	M[b]
	Insoluble oxides	S[b]
Americium	All compounds	M[b]
	Trace contaminant	S[c]
Curium	All compounds	M[b]

a Excretion and retention functions for inhalation of ^3H_2O given in Figure 7.15.
b Excretion and retention functions for inhalation of 5 μm AMAD aerosols given in Figures 7.16-7.25.
c Trace contaminant formed from ^{241}Pu in matrices of nuclear fuel in insoluble (Type S) forms (Fig. 7.25).

7.2.1.3 Gases and vapours

For radionuclides inhaled in particulate form, it is assumed that entry into and deposition in the respiratory tract is governed by the size distribution of the aerosol particles [94I2]. The situation is different for gases and vapours, for which the radionuclide has a specific behaviour at its site of entry into the respiratory tract, depending on the chemistry of the compound. Almost all inhaled molecules contact airway surfaces, but usually return to the air unless they dissolve in, or react with, the surface lining. The fraction of an inhaled gas or vapour that is deposited in each region thus depends on its solubility and reactivity. Generally, however, the regional deposition of a gas or vapour cannot be predicted on a mechanistic basis, from knowledge of its physical and chemical properties, but has to be obtained from an *in vivo* experimental study.

The HRTM assigns gases and vapours to three classes:

- Class SR-1 (soluble or reactive). Deposition may occur throughout the respiratory tract. Retention in respiratory tract tissues and uptake to the systemic circulation may be less than 100 % of the inhaled activity, although this is the default assumption; e.g. tritium gas and tritiated methane, carbon monoxide.
- Class SR-2 (highly soluble or reactive). Total deposition occurs in the extrathoracic airways (ET2). Subsequent retention in the respiratory tract and absorption to body fluids are determined by the chemical properties of the specific gas or vapour; e.g. tritiated water, organically bound tritium and carbon dioxide.
- Class SR-0 (insoluble and non-reactive). Negligible deposition in the respiratory tract. External irradiation from submersion in the cloud of gas, and internal irradiation from gas within the respiratory tract. e.g. from all radioisotopes of argon, krypton and xenon (except ^{37}Ar, 94I1). Subsequent retention in the respiratory tract and absorption to body fluids are determined by the chemical properties of the specific gas or vapour.

ICRP Publications 68 and 71 as well as the guidance document [94I1, 95I2, 02I1] give information on the assignment of gases and vapours to these three classes, and for selected Class SR-1 compounds information on fractional deposition and subsequent clearance.

As an alternative to any of the three default Types defined in ICRP Publication 66, very fast uptake to body fluids (Type V) may be recommended. Although consideration has to be given to the total respiratory tract deposition, regional deposition does not need to be assessed for such materials, since, for the purposes of dose calculation, they can be treated as if they were injected directly into body fluids. Examples are tritiated water and tritiated methane, methyl iodide and methane.

7.2.1.4 Dosimetry

In the ICRP Publication 30 lung model, doses to the respiratory system were averaged over 1 kg of lung tissue and the energy of charged particle emissions in the TB, P and respiratory lymph node regions was assumed to be completely absorbed within the lung, i.e. the absorbed fractions were unity for charged particles. In the HRTM model, doses are calculated to several specific regions of the lung and account is taken of variations in radiosensitivity. Absorbed fractions are energy dependent and prescribed functions are given for all source and target combinations and particle types.

The target cells identified for the assessment of doses are: basal cells of the epithelium in both extrathoracic regions; basal and secretory cells in the bronchial epithelium; Clara cells (a type of secretory cell) in the bronchiolar epithelium; and endothelial cells, such as those of capillary walls and type II epithelial cells, in the alveolar-interstitial (AI) region.

The overall dose to the lung is then taken to be a weighted sum of the doses to the following regions: bronchial, bronchiolar, pulmonary and lymphatic with weighting factors of 0.333, 0.333, 0.333 and 0.001, (the sum is 1), respectively. These weights are known as regional apportionment factors to distinguish them from the tissue weighting factors used in the calculation of effective dose (Chapter 4). They represent the contribution from each region towards the total radiation detriment associated with irradiation of the lung.

7.2.2 Ingestion

The model of the gastrointestinal tract (GI) presently used by ICRP to describe the behaviour of ingested radionuclides is that given in ICRP Publication 30 [79I1]. Radionuclides contaminating food or drink, or cleared from the lung by mucociliary action are swallowed, pass down the oesophagus and enter the gastrointestinal (GI) tract, which is treated as four compartments (Fig. 7.5). Absorption is usually described by f_1 values which give fractional absorption into the systemic circulation (e.g. f_1 = 1.0, absorption = 100 %, f_1 = 0.01, absorption = 1 %). The transport of material through the GI tract is described in terms of movement through the four regions.

Stomach (*ST*)*:* Its contents are acidic, and little absorption takes place other than for very soluble radionuclides such as caesium or iodine for which absorption from the stomach is assumed to be complete (f_1 = 1.0). All other radionuclides are assumed to be absorbed in the small intestine. The residence time for food in the stomach varies from minutes to hours depending on many factors – the amount and composition of food, exercise and emotions. In the dosimetric model the mean residence time is taken to be 1 h.

Small Intestine (*SI*)*:* The principle site of absorption. The contents are alkaline, so that elements which hydrolyse such as rare earths and actinides are not normally readily absorbed. The mean residence time is assumed to be 4 hours. Recommended f_1 values are given in ICRP publications for specific radionuclides (e.g. ^{226}Ra: f_1 = 0.2, ^{144}Ce: f_1 = 0.0005, $^{239}PuO_2$: f_1 = 0.00001).

Upper Large Intestine (*ULI*)*:* Water is absorbed here from the semi-liquid contents. The mean residence time is taken to be 13 hours.

Lower Large Intestine (*LLI*)*:* This region acts as a store for food residues and often forms the critical organ for long-lived non-transportable ingested radionuclides. The mean residence time is taken to be 24 hours.

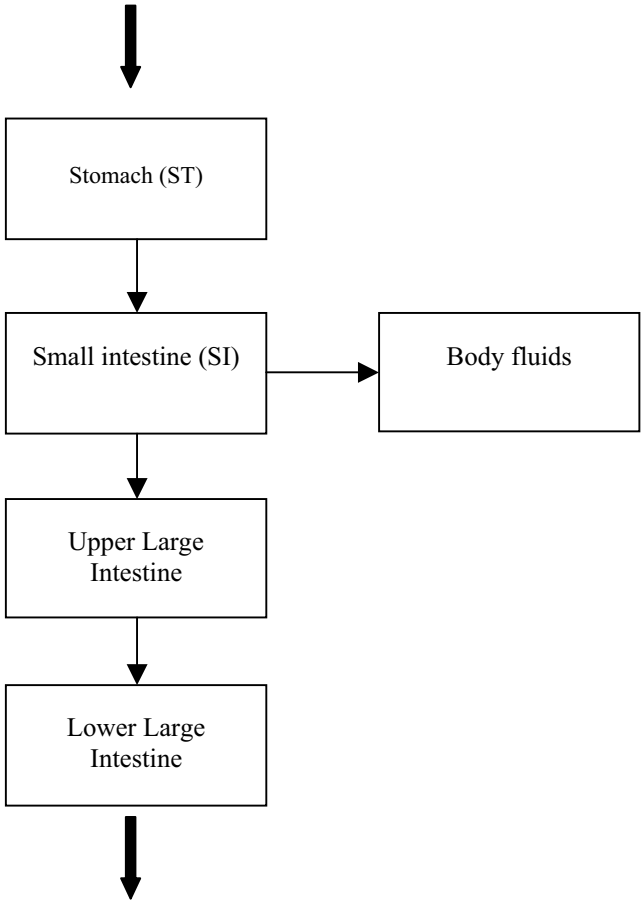


Fig. 7.5. Biokinetic model for the gastro-intestinal tract (based upon 79I1).

Table 7.5 gives the transit time and mass of the contents for the different regions of the GI tract that are assumed for dose calculations. Typical f_1 values are given in Table 7.6 for some important elements.

Table 7.5 Regional masses and residence times in the ICRP Publication 30 dosimetric model for the gastrointestinal tract; [79I1].

Portion of GI tract that is the critical tissue	Mass of contents [g]	Mean residence time [days]	K[a] $days^{-1}$
Stomach (ST)	250	1/24	24
Small Intestine (SI)	400	2/24	6
Upper Large Intestine (ULI)	220	13/24	1.8
Lower Large Intestine (LLI)	135	24/24	1

a Transfer rate

Table 7.6 Examples of f_1 values adopted by ICRP for workers; [94I1].

Radionuclide	Chemical form	f_1
Tritium	Tritiated water	1.0[a]
	Organically bound tritium	1.0[a]
Cobalt	Unspecified compounds	0.1[a]
	Oxides, hydroxides and inorganic compounds	0.05
Strontium	Titanate	0.01
	Unspecified compounds	0.3[a]
Zirconium	All compounds	0.002
Niobium	All compounds	0.010
Ruthenium	All compounds	0.05[a]
Iodine	All compounds	1.0[a]
Caesium	All compounds	1.0[a]
Cerium	All compounds	5×10^{-4} [a]
Polonium	All compounds	0.10
Radium	All compounds	0.20
Thorium	Oxide and hydroxides	2.0×10^{-4}
	Unspecified compounds	5.0×10^{-4}
Uranium	Unspecified compounds	0.02[a]
	Most tetravalent compounds, e.g. UO_2, U_3O_8, UF_4	0.002
Plutonium	Nitrate	1×10^{-4} [a]
	Insoluble oxides	1×10^{-5} [a]
	Unspecified compounds	5×10^{-4} [a]
Americium	All compounds	5×10^{-4} [a]
Curium	All compounds	5×10^{-4}

a Chemical forms of radionuclides for which retention and excretion functions are given in Figures 7.15-7.25.

The f_1 values recommended for workers are not necessarily appropriate for food and drinking water. Moreover the absorption of radionuclides tends to be greater in the newborn although the results of animal studies suggest that gut absorption decreases as age increases, reaching adult f_1 values by about the time of weaning in most cases. An expert group set up by the Nuclear Energy Agency (NEA) within the Organisation for Economic Cooperation and Development (OECD) [88N1] suggested f_1 values to be used as average values for the first year of life. The expert group recommended that for fractional absorption values between 0.01 and 0.5 in adults, an increase by a factor of 2 be assumed for the first year of life; but for elements with a fractional absorption in adults of 0.001 or less, a value 10 times that of the adult should be assumed. This general approach has been adopted in the current ICRP documents when more specific data are not available.

In the dosimetric model for the GI tract doses are calculated to the radiosensitive mucosal cell layer. For low LET radiation (β particles and γ-rays) this is nominally taken to be one half of the average energy absorbed per gram of the contents and for high LET (α) radiation one two-hundredth.

The Publication 30 model for the GI tract has a number of distinct limitations in use and although it has been used for calculating doses to infants and children it does not have specific age-dependent parameter values. For this reason a Task Group of ICRP is developing a new model for the Human Alimentary Tract (HAT). The proposed new model is illustrated in Figure 7.6.

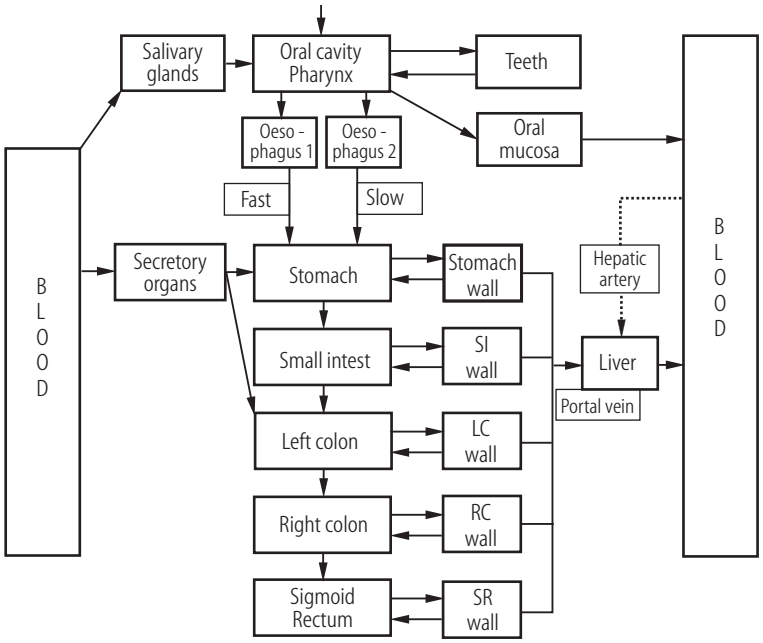


Fig. 7.6. Proposed structure for the new human alimentary tract model; [03M1].

The revision of the Publication 30 model was motivated by a number of developments:

- The 1990 recommendations of ICRP introduced specific risk estimates and tissue weighting factors, w_T for radiation-induced cancer of the oesophagus, stomach and colon, requiring dose estimates for each of these regions [91I1]. The Publication 30 model did not include the oral cavity, or the oesophagus and treated the colon as two regions – upper and lower large intestine (Figure 7.5). The new model for the alimentary tract will comprise the oral cavity, including the mouth, teeth, salivary glands and pharynx, the oesophagus, the stomach, the small intestine, including duodenum, jejunum and ileum, the large intestine, including ascending, transverse and descending colon, rectum and anal canal.
- Since the development of the ICRP Publication 30 model, a considerable body of data has become available on the transit of materials through the different regions of the alimentary tract. These data have been obtained using non-invasive, mainly scintigraphic techniques and include studies of differences between solid and liquid phases, age and gender related differences and the effect of disease conditions. These data are being used to set default transit rates for the defined regions of the alimentary tract for the six age groups given in ICRP Publication 56 (Table 7.1) [89I1].
- Information has become available for morphometrical and physiological parameters and on the location of sensitive cells in different regions of the alimentary tract.
- More information has become available concerning absorption, retention and transfer from different regions of the alimentary tract.
- Extensive age-, gender- and health-dependent information is available.

Development of the new model has been described [03M1]. The new HAT model will be applicable to children and adults under all circumstances of exposure. It considers the movement of radionuclides throughout the alimentary tract from ingestion to elimination. It takes account of sites of radionuclide absorption and retention in the alimentary tract and routes of secretion of absorbed radionuclides into the alimentary tract. Doses will be calculated for sensitive cells in each region: mouth, oesophagus, stomach, small intestine and colon.

The new model is more detailed and morphological than the previous gastro-intestinal tract model [79I1]. The new model is physiologically based. It includes consideration of absorption in regions other than the small intestine when such information is available. The model can also be used for radio-pharmaceuticals. The model provides the flexibility needed to calculate dose to the alimentary tract for a wide range of exposure conditions and for specific individuals. A gut transfer factor, equivalent to the f_1 value, will take account of absorption from the small intestine and from other regions of the alimentary tract where information is available.

An important development in the new model is the calculation of doses to sensitive cells in the different regions of the alimentary tract. The location of sensitive epithelial stem cells in the various regions is considered separately; that is for the mouth, oesophagus, stomach, small intestine and colon. Doses from radionuclides in the gut lumen, retained radionuclides and radionuclides in transit to blood are considered.

It is expected that the report will be used as the basis for future dosimetric calculations for both ingested radionuclides and radionuclides passed through the throat and swallowed after inhalation.

7.2.3 Cuts and wounds

The presence of cuts, abrasions, burns or other pathological damage to the skin may greatly increase the ability of radioactive materials to reach subcutaneous tissues and thence the blood and systemic circulation. Although much of the material deposited at a wound site may be retained at the site, and can be surgically excised, soluble (transportable) material can be transferred to the blood and hence to other parts of the body. These events occur only as a result of accidents, each event will, therefore, be unique and need to be assessed by occupational health physicists and medical staff. To date, ICRP has not given advice on the interpretation of wound monitoring data following accidents involving radionuclides. The biokinetic models that have been developed for various radionuclides are, however, applicable to the soluble component of any deposit in cuts or wounds that enters the blood circulation. Insoluble material will be slowly translocated to regional lymphatic tissue, where it will gradually dissolve and eventually enter the blood. A variable fraction of insoluble material can be retained at the wound site or in lymphatic tissue for the remainder of the individual's life. If particulate material enters the blood it deposits principally in phagocytic cells in the liver, spleen and bone marrow.

The United States National Committee on Radiological Protection and Measurements (NCRP) has established a Committee to review the problem of wound dosimetry. The report it is preparing will contain an extensive compilation of human and experimental data on the behaviour of radionuclides at wound sites. Four default categories for wound retention of deposited material have been proposed as summarised below:

- weakly retained (≤10 % retained at one day, <1 % retained at 16 days);
- moderately retained (11-55 % retained at one day; ≤5 % at 64 days);
- strongly retained (32-85 % retained at one day; 8-40 % at 64 days); and
- avidly retained (>80 % retained in one day; >50 % at 64 days).

In addition default categories are also being considered for colloids, particles and fragments.

In reviewing the experimental data available, various chemical forms of elements/radionuclides are being allocated to these categories on the basis of either the results of studies in experimental animals or their chemical characteristics [03G1]. Once the report is complete it will need to be reviewed by the NCRP and it is possible these categories may change. The ICRP is awaiting publication of the report before deciding on its future work in this area.

7.2.4 Absorption through intact skin

The intact skin provides an effective barrier against the entry of most radioactive materials into the body, exceptions of practical importance being tritium oxide as liquid or vapour, organic carbon compounds and iodine as vapour or in solution. No generalised model has been adopted by ICRP for estimating absorption of radionuclides through the skin although it would be possible to develop specific models. For example, the behaviour of tritiated organic compounds following direct absorption through the skin would be expected to be significantly different from that after inhalation or ingestion. For skin contamination, both the radiation dose to the area of skin contaminated and the dose to the whole body as a result of absorption need to be considered. ICRP [77I1, 91I1] has recommended that for skin contamination doses should be calculated to sensitive cells, assumed to be at a depth of 70 μm (as a reasonable average value). ICRP [79I1] addressed the uptake of tritiated water vapour by assuming the uptake is instantaneously distributed within body water in the same manner as the inhaled water vapour. That is, for airborne HTO vapour, the dose per unit uptake through the intact skin is the same as the dose per unit activity inhaled. For deposited activity doses are to be calculated as an average to each cm^2 of skin tissue. This applies to activity distributed over the skin surface or aggregated in particles. No specific models are recommended by ICRP for calculating doses from β particles deposited on the skin (also see Section 8.2).

7.2.5 Systemic behaviour of radionuclides

The fraction of an intake of a radionuclide entering the systemic circulation is referred to as the uptake. In Publication 30 [79I1, 80I1, 80I2, 86I1] ICRP reviewed biokinetic data for each element for use in the calculation of limits on internal exposure to radionuclides by workers for intakes by inhalation and ingestion. Element-specific biokinetic models were given for the distribution and retention of radionuclides following their entry into the blood. The ICRP 30 models applied specifically to workers and not to members of the public. More recently, Publications 56, 67, 69 and 71 have revised the biokinetic models for selected elements and these have been applied in the calculation of dose coefficients for both workers and members of the public [89I1, 93I1, 94I1, 95I1, 95I2] (Table 7.1).

If a radionuclide that enters the blood is an isotope of an element that is required by the body then it will follow the normal metabolic pathways for that element (e.g. Na, P, K, Ca, Fe). If it has similar chemical properties to an element that is normally present then it will tend to follow the biokinetic pathways of that element, although its rate of movement between the various compartments in the body may be different (e.g. ^{90}Sr and ^{226}Ra behave similarly to Ca, ^{137}Cs and ^{86}Rb similarly to K). For other radionuclides their behaviour in the body will depend upon their affinity for biological ligands and other transport systems in the body and, as a result, the extent of uptake is unpredictable and must be assessed from the available human or animal data (e.g. ^{95}Nb, ^{106}Ru, ^{239}Pu, ^{241}Am).

Radionuclides entering the blood may distribute throughout the body (e.g. ^{3}H, ^{42}K, ^{137}Cs); they may selectively deposit in a particular tissue (e.g. ^{131}I in the thyroid; ^{90}Sr in bone) or they may deposit in significant quantities in a number of tissues (e.g. ^{239}Pu, ^{241}Am, ^{144}Ce). Some examples of the behaviour of selected radionuclides are given below. Limited information is also given on methods of treatment for accidental intakes. More information on decorporation of radionuclides is given in Chapter 9.

7.2.5.1 Elements that distribute widely in body tissues

Hydrogen

Tritium labelled water (HTO), given either orally or by intravenous injection, is rapidly absorbed from the lungs and absorption from the gut is also essentially complete (f_1= 1.0). HTO distributes throughout the body water and is subsequently lost from the body with a biological half-time of about 10 days as a result of excretion in the urine, sweat, faeces and via the lungs (i.e. about 7 % of the total body water is lost per day). The addition of HTO to the body water has been a standard method of determining total body water by isotope dilution. For example, following intravenous injection of 1000 kBq of HTO the activity in a urine sample 6 hours later was 20 Bq ml^{-1}. The total body water is then:

$$\text{Total body water} = \frac{1{,}000{,}000\,\text{Bq}}{20\,\text{Bq/ml}} = 50{,}000\,\text{ml}\,(50\,\text{litres})$$

The total body water in Reference Man is 42 litres [75I1]. The rate of loss of tritiated water can be increased by increasing the fluid intake (see Chapter 9). Doubling the fluid intake from 2 to about 4 litres per day can reduce the half-time of tritiated water to about 5-6 days. This rate of loss of tritiated water is also found in countries with a hot climate, such as India.

In practice, a small fraction of tritium in body tissues becomes incorporated into organic compounds – amino acids, carbohydrates, etc. – and is retained with a longer half-time. For adults, ICRP [89I1] assumes that this fraction is 0.03 (3 %) and is lost with a half-time of 40 days, while the half-time of 10 days applies to the remaining fraction of 0.97 (97 %).

For members of the public, ingesting foods containing tritium, absorption of organically-bound forms and their incorporation into body tissues will lead to longer retention of a larger component. ICRP [89I1] assumes that in the adult, the half-times of 10 days and 40 days apply to equal fractions (0.5) of activity entering blood; values the same as those recommended for organically bound forms of ^{14}C.

Caesium

^{137}Cs, together with ^{90}Sr, is a major component of nuclear fission. As a result of nuclear weapons testing ^{137}Cs has been injected into the atmosphere and weapons fallout has resulted in the contamination of the food chain and man [77U1]. The first observation of the presence of weapons fallout ^{137}Cs in man was reported in 1956 [56M1]. Since then it has been shown to be present in everyone as a result of contamination of the environment by nuclear test explosions, routine releases from nuclear sites and by the accident at Chernobyl in 1986. ^{137}Cs may enter the body either by inhalation or through the foodchain. Absorption from the gut is almost 100 % (f_1 = 1.0, see Section 7.2.2). Once inside the body caesium ions (Cs^+) behave very similarly to potassium ions (K^+) and are rapidly taken up by cells. There is a considerable concentration factor between the cells and plasma. Generally tissue/plasma concen-tration ratios are the same for K^+ and Cs^+ but there are a number of exceptions and some variations between different species. In particular, muscle accumulates Cs^+ more effectively than K^+ and is the main site of long term deposits in the body. At equilibrium the muscle accounts for more than 50 % of the total body ^{137}Cs in man and bone about 8 %. Accumulation of Cs^+ by cells is both by diffusion and by the ion pump that normally accumulates K^+. There is a continual turnover of Cs^+ in body tissues.

For the purposes of dosimetry the retention of ^{137}Cs in man is taken to have two components [89I1]. The first accounts for about 10 % of the administered activity and is excreted mainly in the urine with a half-time($T½$) of about 2 days. The second component (90 %) has a half-time of about 110 days in males (range about 50 to 150 days). The long half-time mainly reflects the slow turnover rate in muscle tissue. The retention half-time for the long-term component in females is less than in males, with a mean value for adults of about 60 - 65 days. The use of the ICRP value of 110 days is therefore likely to be conservative for adult females. In children the half time is less than in adults. Thus for a 5 year-old the half- time is taken to be 55 days [89I1].

In cases of accidental intakes, Prussian Blue (ferric ferrocyanide) can increase the rate of excretion of ^{137}Cs from the body (see Chapter 9). If Prussian Blue is ingested it will accumulate any caesium secreted into the gut preventing it being re-absorbed. The half-time of retention can be reduced to about 40 days; the rate of loss is dependent upon the turnover rate of ^{137}Cs in tissues and its loss into the gastrointestinal tract.

Ruthenium

^{106}Ru is also produced in nuclear fission. Its absorption from the gastrointestinal tract is quite low; the value for gut absorption (f_1) is taken to be 0.05. The distribution of ^{106}Ru in mice, rats, monkeys and dogs is fairly uniform throughout all tissues after an initial period during which the kidneys contain the highest concentration. The animal data have been used by ICRP [89I1] to define a retention function for

ruthenium in man. Any ruthenium entering the blood is assumed to be distributed throughout the body with retention components of 35 % ($T½$ = 8 days); 30 % ($T½$=35 days) and 20 % ($T½$=1000 days). The remaining activity entering the blood (15 %) is taken to be promptly excreted.

Because of the relatively short retention time in the body of most of the intake and a physical half-life of 368 days, the effective dose from ingestion of ^{106}Ru is dominated by the radiation dose to the large intestine from beta-particles emitted while it passes through the GI tract.

7.2.5.2 Elements that deposit mainly in particular organs or tissues

Iodine

Radioactive isotopes of iodine are important components of nuclear fission, particularly in the first few days and weeks after a release into the atmosphere. If taken into the body they are accumulated by the thyroid gland, as was demonstrated particularly after the accident at Chernobyl. The most important isotope is ^{131}I which has a physical half-life of 8.04 days. Radioactive isotopes of iodine are also widely used in medicine. They are used to demonstrate changes in thyroid function, to treat hyperthyroidism or to kill tumour cells in the treatment of thyroid cancer.

The thyroid gland consists of a bi-lobed body in the neck region. It produces the hormones thyroxine and tri-iodothyronine which are important for regulating the body's metabolic rate. Disorders of the gland can result in either an under- or over-active gland (hypo- or hyperthyroidism). The gland weighs about 20 g in the healthy adult (2 g in the newborn child) and is made up of 20 to 40 million spherical vesicles (follicles) per lobe. Each follicle is surrounded by a single layer of cuboidal epithelial cells (acinar cells) lying upon a basement membrane and in proximity to numerous blood capillaries. The vesicles are filled with a structureless semi-fluid material – the so-called “colloid” which contains the active component of the gland (a protein-storage form of hormone). When the thyroid is quiescent, colloid is abundant and the follicles large (about 300 μm in diameter). When the thyroid is active colloid is scanty and follicles small (about 50 μm in diameter). The gland contains about 10,000 μg of iodide in the average normal adult.

For adults in Europe about 225 μg of stable iodide enters the extracellular (iodine) space from the diet each day, absorption occurring across the small intestine within 1-2 hours. About 70 μg of iodide per day is trapped by the thyroid and converted to thyroid hormones while most of the rest is excreted in the urine. The amount of ingested iodide that is taken up by the gland is thus about 30 % [83S1, 87S1]. The fractional absorption varies between different individuals and there are significant differences between countries because of varying levels of stable iodine in the diet. For the purpose of dosimetric modelling ICRP has recommended that the uptake of radioiodine by the gland should be taken to be 30 %. The iodide synthesised into hormone leaves the gland with a half-time of about 80 days (adult) and enters other tissues. From this source most of the iodine (about 80 %) is metabolised back to free iodide with a half-time of about 8 days and re-enters the iodide space, the rest is excreted in the faeces. In adults the total amount of stable iodine excreted is approximately equal to the amount absorbed.

Iodide or elemental radioactive iodine (e.g. ^{131}I) may be ingested (f_1 = 1.0) or the volatile compounds inhaled. As the gland is small, and it takes up about 30 % of radioiodine entering the blood, the concentration of radioiodine in the gland, and hence the radiation dose, is more than a thousand times that to other tissues. The turnover of stable iodine, and hence radioiodine, is low ($T½$ = 80 days in adults) and thus short-lived isotopes (e.g. ^{131}I $T½$ = 8.0 days) will decay mainly in the gland rather than being returned to the blood. In children, although the turnover rate is faster the mass of the gland is smaller and hence for a similar intake of radioiodine the dose can be higher. Milk consumption is the most important pathway for the uptake of radioiodine from the human food chain after a release of radioiodine into the environment and children have a high consumption of milk. As a consequence children are the most sensitive (critical) group following such a release.

Various drugs have been used to reduce the uptake of radioiodine into the thyroid gland after an intake. The safest and most effective procedure is to administer a large single oral dose (20-200 mg) of potassium iodide or iodide (see Chapter 9). It is effective within an hour and reduces the subsequent uptake of radioiodine into the gland. The daily intake is suddenly increased from about 225 μg to

20-200 mg. However, the uptake of iodide into the thyroid remains at about 70 μg d^{-1} so that only a small fraction of the stable iodine and hence radioiodine present in the iodide space is then transferred to the gland. Thus, if 20 mg of stable iodide is given promptly the fractional uptake of radioactive iodine by the gland can be reduced to less than 0.001 (0.1 %). Since the half-time of iodide in the iodide space is about 10 hours, with rapid uptake by the gland or excretion from the body, the effectiveness of administering large amounts of stable iodide diminishes the greater the delay; after 48 hours, it is of little value at all. It has no effect on any radioiodine that has been taken up by the gland. Administration of stable iodide is the treatment of choice for accidental intakes of radioiodine and is recommended by the World Health Organisation [99W1].

Alkaline earth elements

The bone is a highly specialised form of connective tissue. It consists of a soft organic matrix of collagen and ground substance in which is deposited calcium hydroxyapatite (Ca_{10} $(PO_4)_6$ $(OH)_2$). The cells of bone consist of a proliferating population of stem cells which differentiate into: osteocytes – the cells responsible for bone maintenance; osteoblasts – the cells responsible for bone formation; and osteoclasts – the cells responsible for bone removal. There are two types of bone:

- Hard cortical bone, which makes up about 80 % of the bone mass and which is penetrated by blood vessels and Haversian systems; and
- Trabecular bone or spongy bone which makes up the remaining 20 % of the bone mass. The trabeculae are generally 100-200 μm in diameter and usually do not contain blood vessels. The spaces between the trabeculae are large (up to 1000-2000 μm) and contain the vascular (red) marrow. The surface area of trabecular bone is estimated to be about 4 times that of cortical bone [79I1].

The surfaces of bone are covered with non-mineralised layers of connective tissue. This is the periosteum on the external bone surface and the endosteum on the internal surface. The processes of bone turnover, resulting from the laying down of new bone and removal of old bone, continue throughout life although slowing down with increasing age.

Calcium is thus an important component of the skeleton and other alkaline earth elements can be substituted for it in the bone matrix. A number of substitutions are possible in the lattice structure without disturbing the symmetry of the crystal lattice. ICRP has developed age-dependent biokinetic models for the alkaline earth elements (Ca, Ba, Sr and Ra) [93I1]. These models allow for the recycling of radionuclides between the skeleton, blood and soft tissues. They are physiologically based and take account of bone growth and remodelling as a function of age. They can also be applied to the interpretation of bioassay data. These models are essentially dynamic in nature. Material initially deposited on bone surfaces may be buried in bone volume by the formation of new bone or resorbed to red bone marrow. Activity in marrow is either locally or systemically recycled back to bone surfaces, and is also replenished by resorption from bone volume. A fraction will also be excreted from the body. The rate at which some of the processes operate can depend on the type of bone, as well as age, and allowance is made for this by division into cortical and trabecular compartments. This division also accommodates age-dependent dosimetry – in adults most of the active marrow is associated with trabecular bone, whereas for children this is more evenly distributed between both types of bone.

7.2.5.3 Elements that deposit in a number of tissues

Plutonium, americium and curium

Plutonium and other higher actinides are produced in thermal reactors, although the relative amounts generated depend upon the irradiation time. At low irradiation times almost pure $^{239}Pu\backslash^{240}Pu$ are produced (subsequently given as ^{239}Pu). As the irradiation time increases other isotopes of plutonium (e.g. ^{241}Pu)

and higher actinides (e.g. ^{241}Am, ^{242}Cm, ^{244}Cm) are produced. ^{239}Pu is the isotope that has been processed in greatest quantity and for which most biological data are available. ^{238}Pu is also an important isotope and is used as a power source in satellites and in cardiac pacemakers. Plutonium metal is highly reactive, oxidising in moist air if present in a finely divided form. The oxide is chemically inert and insoluble, particularly if produced at a high temperature (1000 °C). Am and Cm metals readily react with oxygen to produce oxides which are much more soluble than PuO_2. In the processing of nuclear fuel soluble nitrates and other soluble complexes are formed.

The behaviour of plutonium in the body depends upon its chemical form. Plutonium dioxide is chemically inert and largely insoluble in biological fluids, particularly if produced at a high temperature. Soluble plutonium compounds (e.g. plutonium nitrate, plutonium citrate) readily hydrolyse at the pH of biological fluids. Following hydrolysis, there is a strong tendency to polymerise, forming a colloidal insoluble compound. Alternatively, soluble compounds can react with naturally occurring complexing agents in body fluids which can readily move around the body. Which of these reactions predominates depends upon the site of entry of plutonium. If into blood, then complex formation is likely; if into a wound or the lung, then colloidal polymers are likely to be formed.

The main route of entry of plutonium into the body is by inhalation, although it can also enter through cuts and wounds. In adults very little is absorbed from the gut (ICRP has adopted gut absorption, f_1, values of 5×10^{-4} for soluble plutonium compounds and plutonium in foodstuffs, 1×10^{-4} for plutonium nitrate, and 1×10^{-5} for plutonium oxide, $^{239}PuO_2$) or across the intact skin. For americium and curium ICRP has adopted an f_1 of 5×10^{-4} for all compounds [94I1, 96I1].

After inhalation, deposition of plutonium in the lungs is determined by particle size as detailed in Section 7.2.1.1. Subsequent clearance from the lungs depends upon its chemical form. Whatever the chemical form inhaled, a fraction, consisting of any soluble material will be rapidly transported to blood and this is excreted through the kidneys or deposited in tissues (mainly the liver and skeleton). The remaining material, consisting of colloidal polymers or material with a low solubility (e.g. PuO_2), is initially retained in the lungs. Material retained in the lungs is largely taken up by scavenger cells (macrophages) in the lungs. These cells may migrate to lymph nodes or reach the muco-ciliary escalator, be swallowed and excreted in the faeces. Alternatively materials in macrophages may gradually dissolve and translocate to blood. In studies with experimental animals retention half-times of the long-term component of retention have varied from 100 to 1000 days or more although soluble compounds are cleared more rapidly. The relative proportions of the long and short retention components depend upon the material initially deposited. For example, in the case of a polydisperse aerosol of high temperature calcined plutonium dioxide deposited in the lungs the amount rapidly moving to blood is normally less than 0.4 % and would be treated as default absorption Type S in the HRTM (Section 7.2.1.2). For a plutonium nitrate aerosol it may be greater than 20 % and would be treated as Type M. Compounds of americium and curium, particularly the oxides, are more soluble than plutonium compounds in the respiratory system and more readily absorbed. They would generally be classified as Type M but any americium or curium trapped in insoluble particles of PuO_2 would behave as Type S.

The principal sites of deposition of Pu in the body after translocation to the blood are the liver and skeleton. This is also the case for Am and Cm. All three actinides are classified as bone-surface seeking elements. That is, they are assumed to be uniformly distributed on endosteal bone surfaces of cortical and trabecular bone after their deposition in the skeleton. In practice, surface deposits of Pu, Am and Cm have been shown to be progressively buried by the formation of new bone. In addition, activity is lost from bone surfaces during resorption and some transfer to bone marrow takes place, particularly for Pu. In ICRP Publication 67, a dynamic model has been adopted to describe their behaviour in the body and to take account of their movement in bone as well as between tissues and excretion (Fig. 7.7 [93I1]).

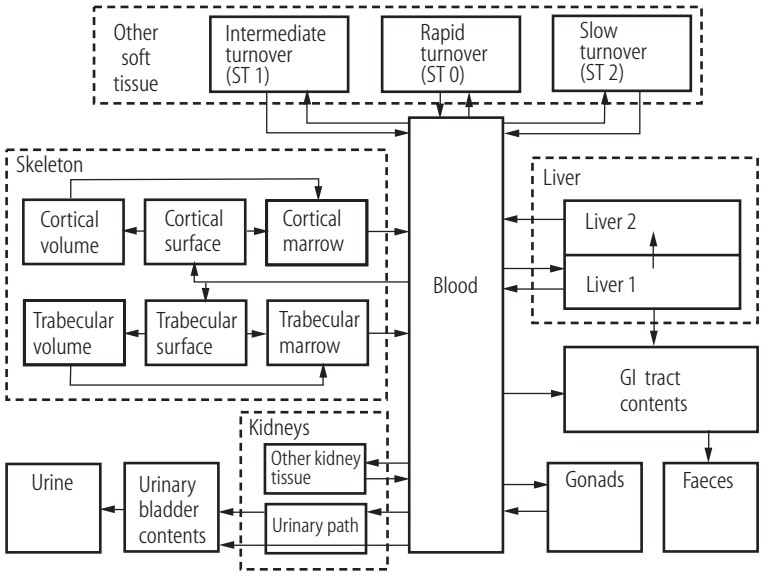


Fig. 7.7. Diagram of the biokinetic model for plutonium and americium; [93I1].

Comparisons of relative skeletal retention in the biokinetic model are complicated by the compound structure of the skeleton and recycling between compartments which gives rise to several retention components. However, calculation shows that after short-term losses are complete it is possible to discern an effective retention half-time in the skeleton of nearly 100 years (Fig. 7.8). The figure also gives the retention of plutonium in the skeleton for 3-month-old infants and 10-year-old children. Because of the faster turnover of bone at younger ages, the initial uptake of plutonium by the skeleton is greater although the rate of loss is also faster. Fig. 7.9 gives comparable data for the liver. Although there are differences in the initial uptake, reflecting the effect of age and deposition in the skeleton, overall liver retention is similar for all age groups. The peak in uptake at 5-10 years reflects the uptake of activity lost to the blood from the skeleton.

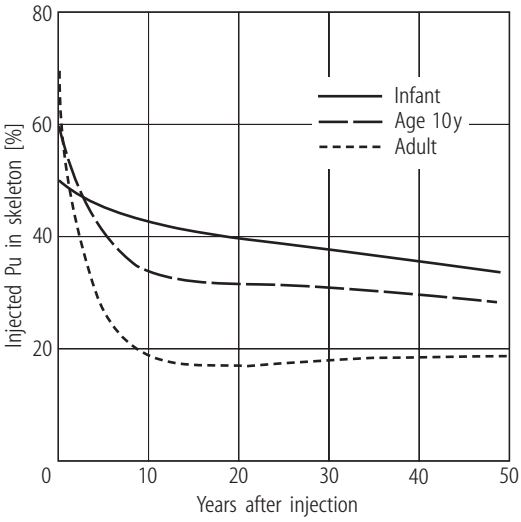


Fig. 7.8. Model predictions of the plutonium content of the skeleton as a function of age and time after injection.

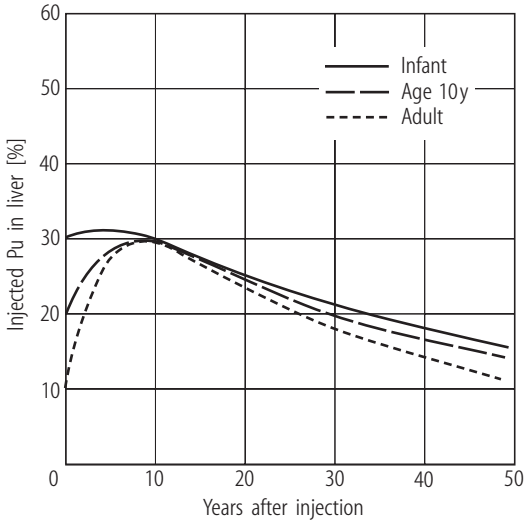


Fig. 7.9. Model predictions of the plutonium content of the liver as a function of age and time after injection.

Intravenous injection of the complexing agent diethylenetriaminapentaacetic acid (DTPA) as the calcium zinc salt is the only accepted therapeutic method for removing soluble forms of actinides from the body. It forms chelate complexes with these actinides which can be excreted in the urine and hence

can effectively clear them from the systemic circulation and some that has recently deposited in bone and other tissues. It is unable to remove intracellular deposits or activity that has been buried in bone and must, therefore, be administered soon after an intake. It can remove some soluble complexes from the lungs. Local injection of DPTA into contaminated wounds can remove more soluble forms of plutonium from the body than the same amount given intravenously, provided the DPTA completely infiltrates the wound site. Further information on DTPA treatment is given in Chapter 9.

7.2.6 Excretion

In the biokinetic model described in ICRP Publication 30, no specific information was given on excretion in urine and faeces, although the models were used in Publication 54 [88I1] for interpreting excretion data. In the 1990 recommendations of the ICRP [91I1], however, the urinary bladder and the colon are given explicit tissue weighting factors w_T (see Section 7.3.1) and the revised biokinetic models given by ICRP now give specific information on excretion pathways in urine and faeces [93I1]. For assessing doses from systemic activity lost into the faeces, the model for the gastrointestinal tract is used (Section 7.2.2) assuming secretion of radionuclides from the blood into the upper large intestine. A model for the urinary bladder has been adapted for calculating doses to the bladder wall [93I1]. The bladder is taken to be of fixed size containing 15, 25, 65, 75, 85 and 115 ml of urine in the 3-month-old, 1-, 5-, 10-, 15-year-old children and adults, respectively. These volumes represent the average content of the bladder during the time period between voids. The rate at which radionuclides enter the bladder is based on their elimination rates from body tissues and the urine to faecal excretion ratio adopted for the biokinetic data for each element. For some elements, the biokinetic data directly address excretion. The number of voids per day for the 3-month-old and 1-year-old are taken as 20 and 16, respectively. For all other ages, 6 voids per day are assumed.

7.2.7 Embryo and foetus

During pregnancy, radionuclides that have entered the mother's body, either before or after conception, can irradiate the developing embryo and foetus. The radiation dose to the offspring will depend upon a number of factors. These include:

- the transfer of radionuclide(s) to the developing offspring from maternal blood and from deposits in the tissues of the mother;
- the distribution and retention in foetal tissues;
- the physical half-life and formation of decay products;
- growth of the offspring; and
- photon irradiation from radionuclides in the placenta and maternal tissues.

Radiation doses will also be received by the newborn child from radionuclides retained at birth.

During the foetal period of development, radionuclides can cross the placenta to reach the tissues of the embryo and foetus from the maternal circulation. The processes involved in this transfer may include simple diffusion, facilitated transport, active transport, movement through pores and channels, and pinocytosis [83S1, 87S1]. Most human data on the placental transfer of radionuclides are available from studies with labelled metabolites, radiopharmaceuticals and other radionuclides used in nuclear medicine, although some data are also available for radionuclides in fallout from weapons testing or for radionuclide releases into the environment as a result of nuclear accidents (e.g. ^{90}Sr, ^{131}I, ^{137}Cs). Analysis of autopsy samples has also given information on both naturally occurring and artificially produced radionuclides. Information is additionally available on levels of stable elements in the placenta and foetal tissues that can be compared with those in the adult. The rather limited amount of human data available has made it

essential to use the results of animal studies in the development of dosimetric models for man, although even here information can be very limited for many elements. Chemical analogy is also of value in model development.

ICRP has issued Publication 88 [01I1] giving dose coefficients for the embryo and foetus following intakes of radionuclides by the mother. It covers selected radionuclides of the 31 elements covered in Publications 56, 67, 69 and 71 (Table 7.1) and applies to the offspring of both members of the general public and workers. In the development of biokinetic and dosimetric models, two approaches have been used. Where sufficient information is available, element-specific models have been given. This applies, for example, to tritiated water, caesium, iodine and the alkaline earths. When appropriate human data are not available, animal studies have provided the main basis for model development using a generic modelling approach.

It has been assumed, in the absence of more specific information, that the dose to all tissues of the embryo, taken to be up to the end of 56 days after conception, (i.e. the end of the second month of gestation), can be approximated by the dose to the maternal uterus. All organs and tissues of the developing embryo thus receive the same dose.

The general approach that has been adopted for calculating equivalent doses to the organs and tissues of the developing foetus from experimental studies in animals is to use average concentrations of a radionuclide in the foetus (C_F) and mother (C_M) obtained shortly after injection. Where possible the value of the C_F:C_M concentration ratio adopted has been based on results obtained in a number of different animal species. The total activity transferred to and retained in the foetus from 57 days of gestation to birth at 38 weeks (266 days) is calculated for each radionuclide from the C_F:C_M ratio. This ratio at the time of the intake is assumed to stay constant for the remaining period of the pregnancy. This is taken to be a conservative assumption. Some examples of (C_F:C_M) concentration ratios adopted are given in Table 7.7. The concentration ratio may depend upon the time of the intake in relation to the start of the pregnancy. Thus for an acute intake of plutonium at any time before pregnancy the C_F:C_M ratio is taken to be 0.03; this ratio is then maintained at this value through the period of gestation. For an acute intake during the first trimester of pregnancy, however, a ratio of 0.1 is adopted, subsequently increasing to 0.3 for an intake at the end of the second trimester (180 days) and 1.0 for an intake at term (266 days). Again this ratio is assumed to be kept constant over the remaining period of the pregnancy.

Table 7.7 Concentration ratios for elements in the foetus and mother (C_F:C_M) following intakes by the mother before or during pregnancy and corresponding ratios for the placenta (C_{Pl}:C_M)

	C_F:C_M		
Element	Intakes prior to pregnancy	Intakes during pregnancy	C_{Pl}:C_M
H in HTO	1.6	1.6	1
Organic carbon	1.5	1.5	1.5
Sulphur	1	2	2
Zinc	2	2	1
Zirconium	0.2	0.2	1
Ruthenium	0.01	0.2	0.1/2[c]
Caesium[a]	1	1	1
Cerium	0.01	0.05	0.1/1[c]
Plutonium	0.03	0.1/0.3/1[b]	0.1/5[c]
Americium	0.01	0.1	0.1/2[c]

a Half-time of long-term component in mother during pregnancy taken to be 50 days
b Intakes in 1st and at the end of the 2nd/3rd trimester (see text)
c Intakes before/during pregnancy

Dosimetric models were developed by ICRP that allowed for the calculation of doses to the embryo and to foetal tissues from radionuclides deposited either in the tissues of the embryo/foetus, in the placenta or in the mother. To provide data that could be used for assessing a range of possible intake scenarios dose coefficients have been given for acute and chronic intakes by the mother at various times

both before conception or during pregnancy. Dose coefficients for the offspring following ingestion of radionuclides by the mother are given for a range of f_1 values in Publication 88, while dose coefficients for the offspring after inhalation of radionuclides by the mother are given for both 1 and 5 μm AMAD aerosols (the default particle sizes for members of the public and workers, respectively) and appropriate lung absorption Types.

For acute exposures, intakes of radionuclides are taken to occur at the time of conception and after 5, 10, 15, 25 and 35 weeks of the pregnancy and at 6 months and 2½ years before conception. For chronic exposures, intakes are taken to occur during the year of pregnancy, starting from conception and for 1 year or for 5 years prior to conception. This range of intake scenarios was selected to allow doses to the offspring to be calculated for any pattern of intake by the mother. Equivalent doses to the date of birth have been given in Publication 88 for the brain, for the most sensitive 8-15 weeks of gestation, and for the tissue receiving the highest dose. The effective dose to birth has also been given using the w_T values recommended by ICRP in Publication 60 [91I1]. Whilst these values are not strictly appropriate for exposures *in utero*, they have been used as no alternative weighting factors are available and the calculation of effective dose provides a useful quantity for comparison with doses to the reference adult. Effective doses (to age 70 years) received after birth are also given, together with the total committed effective dose (before and after birth) received by the offspring.

The total committed effective dose to the offspring, $e_{offspring}$ due to maternal intake of the radionuclide is the sum of the effective dose received during the *in utero* period, $e_{in\ utero}$ and the committed effective dose during the subsequent 70 years of post-natal life, $e_{postnatal}$. That is:

$$e_{offspring} = e_{in\ utero} + e_{postnatal} \tag{1}$$

where

$$e_{in\ utero} = \int_0^{8w} \dot{h}_{uterus}(t)\mathrm{d}t + \sum_T w_T \int_{8w}^{38w} \dot{h}_T(t)\mathrm{d}t \tag{2}$$

and

$$e_{postnatal} = \sum_T w_T \int_{birth}^{70y} \dot{h}_T(t)\mathrm{d}t \tag{3}$$

where the limits of integration in the first two integrals are in weeks and in the last term is in years. The value $\dot{h}_T$ is the equivalent dose rate to individual tissues of the offspring during foetal life and after birth. In the case of the embryo the dose to the tissues of the uterus is taken as a surrogate for the dose to the embryo.

In conjunction with the preparation of ICRP Publication 88 a CD-ROM has been issued which gives much more detailed information than in the publication [02I2]. In addition to the doses given in Publication 88 it provides equivalent doses to 15 organs and tissues in the foetus as well as an average equivalent dose to the remainder tissues. It also gives doses to the offspring to a number of times after birth (10, 20, 40, 70 years). Additionally, doses have been given for a range of ten inhaled particle sizes.

Publication 88 gives only dose coefficients to the offspring and no information is provided on comparative doses to the adult. Such a comparison has, however, been published [02S1]. The main findings are that, in general, doses to the offspring are similar to or less than those to the reference adult. For a few radionuclides the dose to the offspring can exceed that to the adult. This applies to ^{3}H, ^{14}C, ^{35}S and ^{59}Fe, to radioisotopes of I and to radioisotopes of the alkaline earth elements including ^{45}Ca, ^{89}Sr, ^{90}Sr, ^{224}Ra and ^{226}Ra. For radioisotopes of iodine and the alkaline earth elements, the greatest doses result from intakes during the last trimester of the pregnancy when there is the greatest foetal demand for iodine and calcium. Whilst in most cases the doses to the offspring for the radionuclides covered in Publication 88 exceed those to the reference adult by a factor of about 2 to 3, in the case of some bone-seeking radionuclides the difference can be around a factor of 10 for intakes of short lived isotopes towards the end of the period of pregnancy. Some illustrative dose coefficients for the offspring following inhalation of ^{137}Cs by the mother (as a member of the public) are given in Table 7.8. In this case the doses to the

offspring are highest for intakes early in the pregnancy as they reflect doses received by the mother over the period of the pregnancy. For intakes later in the pregnancy doses are lower as, although some ^{137}Cs will be retained in the newborn child, the half-time of retention is less than in the mother.

Table 7.8 Dose coefficients [Sv Bq^{-1}] for the offspring from acute intakes by inhalation of ^{137}Cs (T ½ = 30 y) by the mother, as a member of the public (AMAD = 1 μm, absorption Type F, f_1 = 1.0). Reference adult = 4.6×10^{-9} Sv Bq^{-1}

Scenario [weeks][a]	Highest organ dose (*in utero*)[d]	Brain (8-15 weeks)	$e_{\text{in utero}}$	$e_{\text{post natal}}$	$e_{\text{offspring}}$
–130[b]	7.0×10^{-13}	1.9×10^{-13}	7.0×10^{-13}	1.1×10^{-14}	7.1×10^{-13}
–26	6.2×10^{-10}	1.7×10^{-10}	6.2×10^{-10}	9.6×10^{-12}	6.3×10^{-10}
0[c]	2.5×10^{-9}	6.7×10^{-10}	2.5×10^{-9}	1.5×10^{-11}	2.5×10^{-9}
5	2.4×10^{-9}	1.1×10^{-9}	2.4×10^{-9}	2.5×10^{-11}	2.4×10^{-9}
10	2.3×10^{-9}	9.4×10^{-10}	2.3×10^{-9}	4.1×10^{-11}	2.3×10^{-9}
15	2.2×10^{-9}	na	2.2×10^{-9}	6.7×10^{-11}	2.3×10^{-9}
25	1.7×10^{-9}	na	1.7×10^{-9}	1.8×10^{-10}	1.9×10^{-9}
35	6.1×10^{-10}	na	6.1×10^{-10}	4.7×10^{-10}	1.1×10^{-9}

a Intake at indicated time; negative times are prior to pregnancy
b –130 weeks = acute intake 2.5 years before conception
c 0 = acute intake at time of conception
d For ^{137}Cs all tissues receive the same dose
na Not applicable

7.2.8 Transfer in maternal milk

Models are presently being developed by ICRP for the transfer of radionuclides to mothers' milk that will allow the calculation of dose coefficients for intakes by the offspring [03H1]. The publication will cover a review of biokinetic data relevant to an assessment of the transfer of radionuclides to breast milk following intake by the mother, the development of models, and the calculation of doses to the newborn child resulting from the transfer of radionuclides to milk after inhalation or ingestion by the mother. It is assumed that lactation lasts for a period up to 6 months after birth and that milk consumption increases to 800 ml d^{-1} over the first week and then remains constant to the end of lactation. Doses to the infant will be given for a range of intake scenarios. It is proposed that in the publication dose coefficients will be given for acute intakes by the mother at 26 weeks before conception, for intakes during pregnancy at 5, 15, and 35 weeks after conception and for intakes after birth at 1, 10 and 20 weeks of age. In addition doses from protracted exposures throughout pregnancy and lactation will also be included. These dose coefficients should give a sufficient amount of information to understand the implications for doses to the offspring for intakes at various times either before or after birth. Data for additional acute intake times and for chronic exposures as well as for inhalation of a range of particle sizes will be included on a CD-ROM. The dose coefficients for intakes by the 3-month-old infant given in previous publications [96I1] will be used to calculate the doses from the intakes by the suckling infant in milk.

Some preliminary information is given in Table 7.9 (from 03H1) which gives the ratios of infant (offspring) dose to adult dose for chronic intake of various radionuclides throughout pregnancy and lactation. The values for lactation include contributions from activity retained in the mother from intakes during pregnancy as well as transfer to milk from intakes by her during lactation. The results of preliminary model calculations showed that intakes during pregnancy contribute an estimated 15 % of activity in milk for ^{137}Cs, ^{210}Po and ^{241}Am, about 10 % for ^{45}Ca and ^{90}Sr, 4 % for ^{239}Pu and negligible amounts for ^{131}I. Doses to the infant from milk consumption are estimated to exceed adult doses in the cases of ^{45}Ca and ^{131}I. Very similar ratios of infant to adult dose are obtained when considering acute maternal intake by ingestion, during early lactation; that is, for maximum transfer to milk.

Also shown in Table 7.9 are ratios of dose to the offspring and adult from activity transferred to the embryo and foetus during pregnancy. These ratios are based on the dose coefficients for the offspring given in ICRP Publication 88 [01I1]. These offspring doses from *in utero* exposure include contributions from activity retained in the tissues of the child at birth, ranging from about 90 % of the total "foetus" dose for ^{239}Pu to less than 10 % for ^{137}Cs. Only in the cases of ^{131}I and ^{210}Po do the doses to the offspring from transfer in milk exceed that resulting from *in utero* transfer.

Table 7.9 Comparison of doses following chronic maternal ingestion of radionuclides throughout pregnancy and lactation

Radionuclide	Ratio of offspring : adult dose[a]	
	Foetus[b]	Infant in milk[c]
^{45}Ca	12	2.7
^{90}Sr	1.5	0.8
^{131}I	1.0	2.4
^{137}Cs	0.4	0.4
^{210}Po	0.1	0.2
^{239}Pu	0.04	$<10^{-3}$
^{241}Am	0.01	$<10^{-3}$

a Committed effective dose (environmental exposures).
b Includes doses received *in utero* and from activity retained by the child at birth (based on 01I1)
c Includes doses from activity transferred to milk as a result of maternal intakes during pregnancy and lactation (preliminary calculations).

7.3 Dosimetric models

7.3.1 Introduction

The dose to organs of the body (this set of organs is referred to as target organs or target regions) depends in part on the distribution of the activity within the body (this set of anatomical regions is referred to as source regions) and the transport of the radiations emitted in nuclear transformations (decays) of the radionuclide residing in the source regions. In general, the "target regions" as well as "source regions" will be identified as organs of the body, but this need not be the case when knowledge suggests otherwise. For example, the short-lived decay products of radon, when inhaled, deposit on the surfaces of the respiratory airways from which they irradiate the basal cells of the bronchial epithelium as a target region of interest. Various procedures have been employed in computing the dose to target regions, given the information on the distribution of activity within the body. In the late 1960s - early 1970s, however, efforts were devoted to establishing a unified formulation that would be applicable to all types of radiations. The Medical Internal Radiation Dose (MIRD) Committee of the Society of Nuclear Medicine led efforts to formulate a computational scheme based on physical framework in support of the dosimetry of diagnostic radiopharmaceuticals. The formalism set forth by the MIRD Committee [76L1] deals with the various radiations emitted in nuclear transformation of radionuclides in a consistent manner. This formalism only addressed the absorbed dose quantity and was limited to short-lived radionuclides that emit electron (beta and conversion electrons) and gamma radiations. However, the rigorous physical basis of the system enabled it to be extended to the needs of radiological protection and the calculation of equivalent dose by the ICRP in its Publication 30 issued in 1979.

In Chapter 4 the various radiological quantities are defined. The absorbed dose quantity D is a point dosimetric quantity that can be defined in any material. The absorbed dose within the target region T of the body is generally computed as the mean absorbed dose, denoted by $\overline{D}_{\mathrm{T}}$, and thus is the average energy per unit mass absorbed by the target region. The mean absorbed dose can, of course, be written as the integral of point quantity D over the volume of the target. In radiological protection it is necessary to address all the emitted radiations and to recognize that some radiations, and thus their contribution to dose, are more biologically efficient than other radiations. Thus the basic dosimetric quantity of radiation protection is the equivalent dose in target organ T and is denoted by H_{T}. The equivalent dose is defined as

$$H_{\mathrm{T}} = \sum_{\mathrm{R}} w_{\mathrm{R}} \overline{D}_{\mathrm{T,R}} \tag{4}$$

where the summation extends over all radiations R contributing to the mean absorbed dose $\overline{D}_{\mathrm{T,R}}$ in the target T. The radiation weighting factors w_{R} represent judgments regarding the potential relative biological damage of radiation R without regard to the specific tissue or health consequence. The values in current use in dosimetry are given in Chapter 4, Section 4.5.2.2. The weighting factors reflect, in part, the density of the ionization within the target which is indicated by the linear energy transfer (LET) of the radiation. See Chapter 4 for further discussion.

The tissues of the body, of course, vary in their sensitivity to ionising radiation particularly with respect to stochastic effects (cancer induction and hereditary disease). The effective dose quantity was introduced into the radiological protection system in ICRP Publication 26 and was further amplified and extended in Publication 60. The effective dose reflects the underlying information regarding the risk of stochastic effects among the irradiated tissues and aggregates these contributions into a single dosimetric quantity (see Chapter 4). The effective dose E is defined as

$$E = \sum_{\mathrm{T}} w_{\mathrm{T}} H_{\mathrm{T}} \tag{5}$$

where the summation extends over the organs/tissues assigned tissue weighting factors w_{T} as described in Chapter 4, Section 4.5.2.4. Table 4.3 gives tissue weighting factors recommended in Publication 26 [77I1] and in Publication 60 [91I1]. The effective dose resulting from radionuclides within the body can be added to that from external radiation fields to obtain a single quantity that encompasses both exposures as described in Section 7.7.2.

7.3.2 Absorbed fraction and specific absorbed fraction

The formulation of a computational scheme that enabled the estimation of absorbed dose from all radiations emitted in nuclear transformations of radionuclides was largely achieved by the introduction of the absorbed fraction quantity. Consider a source region r within which radiation of type i is being emitted. If target region v absorbs energy from the radiation emitted in the source region r, then the absorbed fraction in v from r $\phi_i(\mathrm{v} \leftarrow \mathrm{r})$ is defined as the quotient of the energy imparted to the target region v and the energy, exclusive of rest energy, emitted in the source region r. That is, the absorbed fraction can be expressed as

$$\phi_i(\mathrm{v} \leftarrow \mathrm{r}) = \frac{\text{energy absorbed by target region v}}{\text{energy emitted in source region r}} \tag{6}$$

The absorbed fraction is defined only for target regions which are volumes; however, no such constraint is placed on the source regions, i.e., it could be a point, line, surface, or volume. The absorbed fraction quantity embodies not only the geometric variables of size, shape, and spatial relationship of the regions, but also the extent to which the radiation is transported through the medium containing these regions.

The specific absorbed fraction $\Phi_i(\mathrm{v} \leftarrow \mathrm{r})$ is defined as the absorbed fraction per unit mass of the target region; i.e,

$$\Phi_i(\mathrm{v} \leftarrow \mathrm{r}) = \frac{\phi_i(\mathrm{v} \leftarrow \mathrm{r})}{m_\mathrm{v}} \tag{7}$$

where m_v is the mass of the target volume. The specific absorbed fraction has the property that it can be defined for all target regions, including the important case of a point target. Recall from Section 7.3.1 that absorbed dose is a point dosimetric quantity. There can, however, be no points in common between the source and target region unless one of the regions is a volume.

If the source and target regions are in a homogeneous absorbing medium that is sufficiently large (relative to the range of the radiation) that edge effects are negligible, and if the activity is uniformly distributed in the source region, then the *uniform isotropic model* is said to apply. Under this model, the distribution of absorbed energy about the source region is a function only of distance from the source. The fraction of emitted energy absorbed per unit mass at a distance x can then be represented by the *point-isotropic specific absorbed fraction* $\Phi_i(x)$. Since the emitted energy must be absorbed somewhere, the point-isotropic specific absorbed fraction must satisfy the constraint that

$$4\pi\rho \int_0^\infty x^2 \Phi_i(x)\, \mathrm{d}x = 1 \tag{8}$$

where ρ is the density of the homogeneous medium.

The point-isotropic specific absorbed fraction for the various radiations of interest provides the basic means of estimating specific absorbed fractions. Non-point source and target regions can be developed simply as the superposition of the point function. Thus the specific absorbed fraction between any non-point target region r and a point source P is the mean of the values of $\Phi_i(x)$ in the target region

$$\Phi_i(r \leftrightarrow P) = \overline{\Phi}_i(x) \tag{9}$$

Furthermore, the specific absorbed fraction in any region r_1 from a source in another region r_2 is the mean of the values of the point-isotropic specific absorbed fraction for all pairs of points in the regions; i.e.,

$$\Phi_i(\mathrm{r}_1 \leftrightarrow \mathrm{r}_2) = \overline{\Phi}_i(x) \tag{10}$$

where x is the distance between points randomly selected in r_1 and r_2. The doubled-headed arrow in equations 9 and 10 indicates that either region may be the source or target region. Equations 9 and 10 can be expressed in their integral representation, but for regions whose geometry is complex, i.e., other than spherical, recourse is often made to numerical evaluation using Monte Carlo techniques to randomly select the points.

As noted above, the point-isotropic specific absorbed fraction is a function only of distance. Thus if the source and target regions are interchanged in Equations 6 and 7, the numerical value of the specific absorbed fraction does not change. This property of the uniform isotropic model is referred to as the *Reciprocity Theorem*. The conclusion of the theorem is that the specific absorbed fraction Φ_i is independent of which region is designated as the source and which the target is. In symbols

$$\Phi_i(r_1 \leftarrow r_2) = \Phi_i(r_2 \leftarrow r_1) \equiv \Phi_i(r_1 \leftrightarrow r_2) \tag{11}$$

and therefore

$$\frac{\phi_i(v_2 \leftarrow v_1)}{m_2} = \frac{\phi_i(v_1 \leftarrow v_2)}{m_1} \tag{12}$$

Note that these relationships apply to all radiations in the uniform isotropic model.

7.3.3 Computational models of the human anatomy

7.3.3.1 Mathematical phantoms

The application and extension of the above in radiological protection necessitated the formulation of a computational model of the human body – a so called mathematical phantom. Such a mathematical description of the adult human was used to provide estimates of the absorbed fractions for photon emitters distributed in the model [69S1]. Much of the stimulus for this development came from the needs of the MIRD Committee and thus the mathematical model is often referred to as the MIRD phantom although over the years many modifications have been made to the model including the extension to children (Fig. 7.10).

The MIRD phantom consists of three principle Sections: (1) an elliptical cylinder representing the arms, torso, and hips,; (2) two truncated elliptical cones representing the legs and feet; and (3) an elliptical cylinder representing the neck region and lower portion of the head, which is topped by a half ellipsoid. The organs of the body were represented by simple conic sections in some cases cut by planes and rotated. The defining equations were readily evaluated and thus the model was well suited for use with Monte Carlo techniques in the computation of photon absorbed fractions. Limitations in the computational hardware of the early 1970s, not the available anatomical information, restricted the realism of the modelling.

7.3.3.2 Voxel models

Voxel models are human models based on computed tomographic or magnetic resonance images obtained from high resolution continuous scans of a single individual. The greyscale data of the medical images are interpreted into tissues (i.e. organs), a process known as segmentation. Each volume element, called voxel, has an identification number that identifies the discrete organ of that particular voxel. The models, consisting of millions of voxels, provide a three-dimensional representation of the human body and the spatial form of its constituent organs and structures. They were initially developed for radiological protection purposes to estimate the risk to a person or population due to an irradiation. For this purpose, a detailed model of the human body is required, together with computer codes simulating the radiation transport and energy deposition in the human body. They are at present the most precise representation of the human anatomy to be used for computational radiation transport simulation.

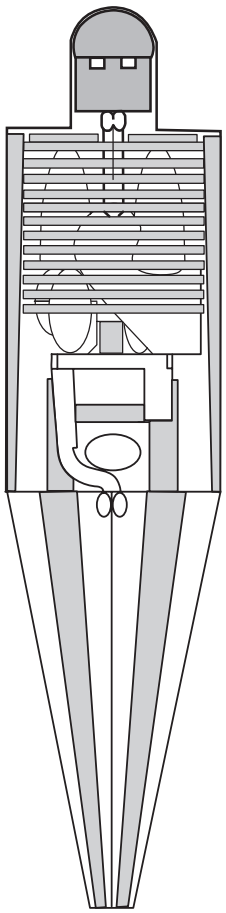


Fig. 7.10. Schematic diagram of the MIRD phantom; [78S1]. Various modifications have been made by other investigators; [87C1].

Among other laboratories, the GSF- National Research Centre in Germany started the development of voxel models covering various ages and anatomical statures [02P1]: an 8 week old baby girl (Baby) and a 7 year old girl (Child) [88Z1], four male models (Golem (38 y), Visible Human (38 y), Frank (48 y) and Otoko (40 y)) and three adult female voxel models named Donna (40 y), Helga (26 y) and Irene (32 y) [02P1]. Fig. 7.11 shows three dimensional reconstructions of some organs of the above female models and demonstrates their anatomic realism. The male adult phantom Golem [01Z1] has height and weight similar to the ICRP Reference Man [75I1]. Otoko (01S1) stems from whole body CT data of a patient whose external dimensions are in good agreement with the Japanese Reference Man [89T1]. The Visible Human was constructed from CT data and photographic data from the Visible Human Project of the American National Library of Medicine, obtained from the CT pictures of a donated body of an executed 38-year-old man from Texas, USA. Both Visible Human and Frank are rather broadly built, the former being also very tall, and are probably more suitable to simulate bigger individuals. Similarly, Donna and Helga are taller and heavier than the reference adult female, as characterised in ICRP Reference Man [75I1] and ICRP Report 89 [02I3], whereas Irene has a weight below the reference female.

Due to their anatomical realism, such models have been the subject of increasing interest and acceptance, and others have been developed elsewhere: Zubal et al [94Z1] at Yale developed a head and torso phantom as well as a head phantom with fine resolution from the CT data of an adult male with dimensions similar to the MIRD-5 mathematical phantom [78S1] who was imaged from head to mid-thigh. Dimbylow and his colleagues [96D1] at NRPB, UK, have developed the Norman model based on whole-body MRI scan data of a healthy volunteer. The exact dimensions of the voxels were scaled so that height and mass of the segmented model agreed with the values of the Reference Man [75I1]. A body representation that gained recent popularity is the Visible Human Male [98S1]; this data set was assembled using the original colour photographic anatomical slice images of the body mentioned above

which was acquired by the Visible Human Project of the American National Library of Medicine. Xu and co-workers [00X1] at the Rensselear Polytechnic Institute have developed the VIP-Man based on these data, which is the most complete body description so far with respect to number of structures defined and voxel resolution. More recently, Kramer et al [03K1] created MAX based on the Yale model. Regarding paediatric models, Caon et al [99C1] constructed a trunk model of a 14-year-old female and Nipper et al [02N1] developed a newborn model which is based upon a high resolution CT scan of a 6-day female baby.

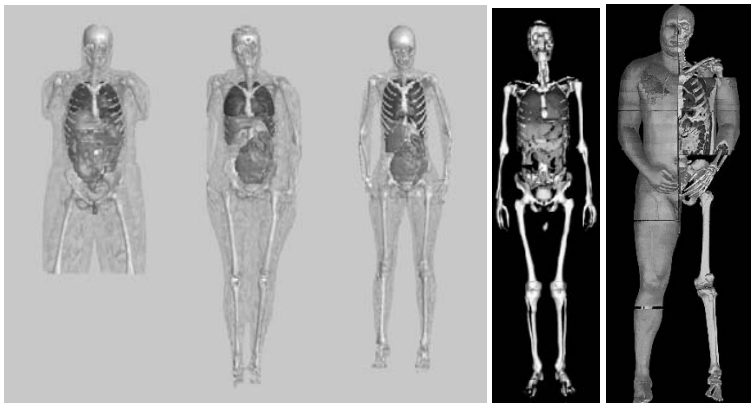


Fig. 7.11. View of the skin and some organs of the female models Helga, Donna and Irene (left) and of the skeleton and some organs of the male model Golem (right); [02Pet]. On the extreme right is an image constructed from the colour photographic anatomical slice images of the Visible Human Project of the American National Library of Medicine; [00X1].

Several image processing functions are used for segmentation of voxel models. However, none of these methods works automatically; but interactive methods controlled by an operator must be applied and thus the segmentation of a whole body model with several organs is a time-consuming procedure. Moreover, it is difficult to obtain a suitable fine, contiguous data set.

The most important advantage of the voxel phantoms over mathematical phantoms, is their realism concerning anatomy: the organ shape as well as the organ location is realistic, since computed tomographic images from an actual subject were employed for their construction. Thus, they offer a clear improvement compared to the older mathematical models whose organs are described by relatively simple geometrical bodies. Furthermore, the distance between the organs, an important parameter, particularly for internal dosimetry where several organs are the so-called source organs and all the others are the targets, are realistically simulated by the voxel models, which is not always the case for the MIRD-type models.

One of the most interesting characteristics of the voxel models is the possibility of varying their size and hence simulating smaller or bigger individuals. Their external dimensions can be adapted for each of the three dimensions independently. All internal dimensions of the resulting scaled-down or scaled-up version of the original model are consequently modified with the same scaling factors. However, the scaling factors should range within reasonable magnitudes, to avoid anatomical errors in the organ proportions.

A limitation of the voxel models is that very small tissues, as for example the eye lenses and the skin, cannot be represented with their correct thickness because it is not possible to segment structures below the voxel resolution. An additional concern is that the supine position of the body during acquisition of the image data results in an upward shift of the abdominal organs and the compression of the lungs. However, for radionuclides with residence times in the body of a few days much of the dose is received under various postures including sitting or lying down. The position of the stomach is well known to be quite varied depending on its content.

Strictly speaking voxel models represent themselves and not a “reference” or an “average” individual. For many situations in radiation protection, this is fully adequate, particularly if their external dimensions comply more or less to the reference or the exposure conditions cannot be accurately described. Moreover, since the models available range from very slim persons to large and heavy persons, they can be used to estimate the doses to an individual by selecting the model fitting best to the person under consideration. However, for certain situations involving regulations, guidelines or dose limitations, for example exposures of a population or radiation workers, dose values are required for a “reference”

individual. As a consequence, there is a need to construct body models that combine a realistic anatomy with organ masses, shapes and locations that are representative. Probably the best way to achieve such an aim is to modify an appropriate voxel model (i.e., one that already resembles Reference Man data in its external body dimensions) to one having reference organ masses as well, retaining its realistic anatomy. This approach was used by Dimbylow [96D1] to construct NORMAN, the model of an adult male, and is now underway at the GSF-National Research Centre in Germany under the supervision of the ICRP. These reference voxel models are then appropriate for calculating organ doses for reference persons and, hence, establishing reference dose conversion coefficients for international recommendations such as those from ICRP.

7.3.4 Dose rate per unit activity, S-factor

Consider an organ T of the body which at time t is absorbing energy from activity in a source region S of the body. Let the activity, i.e., the average rate of nuclear transformations (nt), in the source region be $A_S(t)$ and denote the mean energy emitted as radiation of type R per nuclear transformation by Δ_R; that is $\Delta_R = Y_R\, E_R$, where Y_R and E_R are the yield (number per nt) and energy of radiation R, respectively. The rate at which energy is absorbed in T per unit mass at time t, which is by definition the mean absorbed dose rate, $\overline{\dot{D}_R}(\mathrm{T} \leftarrow \mathrm{S}, t)$ due to the activity in S is

$$\overline{\dot{D}}_R(\mathrm{T} \leftarrow \mathrm{S}, t) = A_S(t)\, \Delta_R\, \Phi_R(\mathrm{T} \leftarrow \mathrm{S}) \tag{13}$$

In general, more than one type of radiation will be associated with the nuclear transformation process of a particular radionuclide, and thus the mean absorbed dose rate is

$$\overline{\dot{D}}(\mathrm{T} \leftarrow \mathrm{S}, t) = \sum_R \overline{\dot{D}_R}(\mathrm{T} \leftarrow \mathrm{S}, t) = A_S(t) \sum_R \Delta_R\, \Phi_R(\mathrm{T} \leftarrow \mathrm{S}) \tag{14}$$

If the activity is present in a number of source organs, then an additional summation must be considered to derive the mean absorbed dose rate in T

$$\overline{\dot{D}}(\mathrm{T}, t) = \sum_S A_S(t) \sum_R \Delta_R\, \Phi_R(\mathrm{T} \leftarrow \mathrm{S}) \tag{15}$$

where the first summation is over all source regions S.

Examination of the above equations reveals that the factors within the inner summation, i.e., Δ_R and $\Phi_R(\mathrm{T} \leftarrow \mathrm{S})$, reflect the physical data on the nuclear transformation process and the transport of the emitted radiations between S and T which depend in part on nature of the radiations and the anatomical relationship of these two regions. Given an agreed-upon analog of the human body for estimation of Φ_R, then considerable effort can be saved through consideration of the additional quantity S. If we define $S(T \leftarrow \mathrm{S})$ as

$$S(\mathrm{T} \leftarrow \mathrm{S}) = \sum_R \Delta_R \Phi_R(\mathrm{T} \leftarrow \mathrm{S}) = \sum_R Y_R\, E_R\, \Phi_R(\mathrm{T} \leftarrow \mathrm{S}) \tag{16}$$

where Y_R is the yield of radiation R of energy E_R. Then the expression for the mean absorbed dose rate reduces to

$$\dot{\overline{D}}(\mathrm{T},t) = \sum_{\mathrm{S}} A_{\mathrm{S}}(t)\, S(\mathrm{T} \leftarrow \mathrm{S}) \tag{17}$$

The quantity S represents the mean absorbed dose rate in T per unit radioactivity in S. If S is considered to be invariant with time, that is, if the analog of the human body and its implied geometric relationships are independent of age, then integration of Equation 17 yields the mean absorbed dose in T.

$$\overline{D}(T) = \sum_{\mathrm{S}} \tilde{A}_{\mathrm{S}}\, S(\mathrm{T} \leftarrow \mathrm{S}) \tag{18}$$

where $\tilde{A}_{\mathrm{S}}$ denotes the time integral of the activity in the source region (the *cumulated activity).* Thus S may also be defined as the mean absorbed dose per unit cumulated activity. Methods for deriving the *cumulated activity* are discussed in Section 7.4.1.

The S factor can be expressed in terms of equivalent dose by inclusion of the radiation weighting factors in its defining equation; i.e,

$$SEE(\mathrm{T} \leftarrow S) = \sum_{\mathrm{R}} \Delta_{\mathrm{R}}\; w_{\mathrm{R}}\; \Phi_{\mathrm{R}}(\mathrm{T} \leftarrow \mathrm{S}) = \sum_{\mathrm{R}} Y_{\mathrm{R}}\; E_{\mathrm{R}}\; w_{\mathrm{R}} \Phi_{\mathrm{R}}(\mathrm{T} \leftarrow \mathrm{S}) \tag{19}$$

where w_{R} is the radiation weighting factor (Section 7.3.1). The quantity *SEE* is a radiological protection quantity introduced by ICRP (1979) as the *specific effective energy*. However a more appropriate name would be the *specific equivalent energy*.

The equivalent dose rate to tissue T can be written in terms of *SEE* as follows,

$$\dot{H}(\mathrm{T},t) = \sum_{\mathrm{S}} A_{\mathrm{S}}(t)\, SEE(\mathrm{T} \leftarrow \mathrm{S}) \tag{20}$$

and the effective dose rate is defined as the sum of the weighted equivalent dose rates in a number of tissues (Section 4.5.2.3 and Equation 5, this Chapter).

$$\dot{E}(t) = \sum_{\mathrm{T}} w_{\mathrm{T}}\, \dot{H}(\mathrm{T},t) \tag{21}$$

7.3.5 Specific absorbed fractions for various radiations

The three principal modes of nuclear transformation are beta decay, alpha decay, and isomeric transition. An additional process, spontaneous fission, is available to some heavy nuclides. The principle radiations involved in these modes of nuclear transformation are alpha particles, electrons (either negative or positive charge) and photons (electromagnetic radiation). The latter two radiations may arise from the nucleus as well as the orbital electrons of the newly formed radionuclide. These radiations differ significantly in their energy deposition pattern, as a result of different mechanisms through which they interact with matter, further details are given in Section 3.5.

7.3.5.1 Electrons

A continuous energy spectrum of electrons is associated with beta decay. The spectrum ranges in energy from zero to the maximum energy permitted by the difference in the energy level of the parent and daughter nucleus. Electrons of discrete energy are also observed in nuclear transformation, as a result of processes involving the orbital electrons.

Under the auspices of the MIRD Committee, Berger has tabulated point-isotropic specific absorbed fraction data for monoenergetic electron sources ranging in energy from 0.025 to 5 MeV [71B1]. To facilitate numerical use, Berger presented the data in terms of a scaled point kernel $F(r/r_0)$ where r_0 is the continuous slowing down approximation (*csda)* range. The point-isotropic specific absorbed fraction $\Phi(r)$ in terms of Berger's scaled point kernel is

$$\Phi(r) = \frac{1}{4\pi\rho r^2 r_0} F(r / r_0) \tag{22}$$

where ρ is the density of the medium. The tabulations were prepared for water as a surrogate medium for soft tissue. Table 7.10 presents the 90-percentile distance (x_{90}) in water as a function of electron energy. The 90-percentile distance is defined to be the radius of a sphere around a point source within which 90 % of the emitted energy is absorbed. As can be seen from Table 7.10, electrons of energy up to about 2 MeV deposit their energy within a distance of less than 1 cm.

Table 7.10. Deposition of electron energy (range-energy relationships). Percentile distance x_{90} in water for electrons from monoenergetic sources. The results for $E_0 \leq 0.020$ MeV are extrapolated (based on Table 9, page 15, 71B1).

E_0 [MeV]	x_{90} [cm]	E_0 [MeV]	x_{90} [cm]
4.0	1.57	0.70	0.207
3.5	1.36	0.60	0.169
3.0	1.16	0.50	0.131
2.6	0.99	0.40	0.096
2.2	0.82	0.30	0.063
2.0	0.74	0.20	0.0334
1.5	0.53	0.10	0.0106
1.2	0.41	0.05	0.00318
1.0	0.328	0.010	0.000194
0.90	0.287	0.005	0.000060
0.80	0.247	0.001	0.000008

Organs of the body are of dimensions sufficiently large relative to the electron range that the electron absorbed fraction may be taken as unity if the source is uniformly distributed in the organ. Thus the specific absorbed fraction for electrons is

$$\Phi(\mathrm{T} \leftarrow \mathrm{S}) = \begin{cases} 1/m_T\,, & \text{if } \mathrm{T} = \mathrm{S} \\ 0\,, & \text{if } \mathrm{T} \neq \mathrm{S} \end{cases} \tag{23}$$

A notable exception to the above occurs for walled organs where the source resides in the contents, e.g., urinary bladder and the segments of the gastrointestinal tract.

For organs whose contents contain an electron emitter, the specific absorbed fraction in the wall of the organ from its contents is usually taken to be given by

$$\Phi(\text{wall} \leftarrow \text{contents}) = \frac{1}{2\, m_{\text{contents}}} \tag{24}$$

where $m_{contents}$ is the mass of the contents. This relationship is derived from the fact that the dose rate at the surface of a half space containing a uniform distribution of activity is one-half the equilibrium dose rate at locations within the contaminated half space far from the interface. It should be noted that the approach for walled organs may be very conservative, in that the critical cells are typically considered to be the basal cells of the epithelial layer, which lie at some depth in the tissue; in the gastrointestinal tract, they are further shielded by a mucus layer. Thus the dose rate in the wall may decrease rapidly from the value at the surface, particularly for low-energy electrons. Consideration of these details in the dosimetric models must await further description of the location of the cells at risk.

7.3.5.2 Alpha particles

The point-isotropic specific absorbed fraction $\Phi(x)$ has not been tabulated in the literature for alpha particles since the range of alpha particles in tissue is sufficiently small that for organs of the body, an absorbed fraction of unity can be assumed. However, in some specific instances – such as alpha emitting short-lived radon daughters deposited on the airways of the lung – consideration must be given to the energy deposition pattern.

The point-isotropic specific absorbed fraction $\Phi(x)$ can be defined as

$$\Phi(x) = \frac{1}{4\pi\rho x^2 E_\alpha}(\mathrm{d}E/\mathrm{d}x)_x \tag{25}$$

where $(\mathrm{d}E/\mathrm{d}x)_x$ is the stopping power of the alpha particle at the energy it has after travelling a distance x, and E_α is the initial energy of the alpha emission. In order to avoid the discontinuity at $x = 0$, the quantity $4\pi x^2 \rho \Phi(x)$ is tabulated for the point-isotropic specific absorbed fraction. The mass stopping power $\frac{1}{\rho}(\mathrm{d}E/\mathrm{d}x)$ and the *csda* range as a function of energy in soft tissue are shown in Figure 7.12. These data can be used with Equation 25 to compute the point-isotropic specific absorbed fraction.

Specific absorbed fractions for source-target pairs in the body are the same as employed for beta radiation, that is Equation 23 is applicable to solid organs. For walls of the gastrointestinal tract Equation 24 is applied, however only 1 % of the alpha particles' energy is considered to penetrate the mucous lining of the tract.

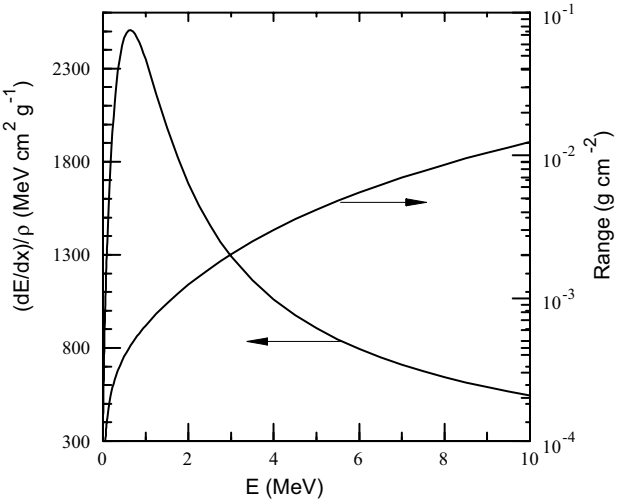


Fig 7.12. Mass stopping power and range of alpha particles in soft tissue.

7.3.5.3 Gamma-rays and characteristic X-rays

Gamma-rays and X-rays are electromagnetic radiations of short wavelength, orders of magnitude shorter than visible light. A nucleus in an excited state from which it is energetically impossible to de-excite through emission of particulate radiation (emission of alpha or beta particles) may de-excite through the emission of one or more photons of electromagnetic radiation. Many nuclides formed in beta or alpha decay may be in an excited state, and thus gamma-ray emission often accompanies these decays. Electromagnetic radiations associated with changes in nuclear state are referred to as gamma radiation.

A measure of the probability per unit distance (density distance) travelled by a photon that an interaction occurs is the mass attenuation coefficient. As the three interaction events, photoelectric effect, Compton effect and pair formation (see Chapter 3) are independent and mutually exclusive, the total mass attenuation coefficient μ/ρ is given as

$$\mu/\rho = \tau/\rho + \sigma/\rho + \kappa/\rho \tag{26}$$

where τ/ρ, σ/ρ and κ/ρ are the mass attenuation coefficient for the photoelectric effect, Compton effect, and pair formation interactions. Tabulations of these coefficients for various elements and compounds of general interest have been given [95H1]. Values for other compounds or absorbing media can be computed as

$$\mu/\rho = \sum w_i(\mu/\rho)_i \tag{27}$$

where $(\mu/\rho)_i$ is the tabulated value for the i^{th} element, and w_i is the fraction by weight of the i^{th} element in the medium of interest. The equation is valid because the chemical binding energies between atoms in a molecule are very small, thus not significantly altering the electronic binding energies. The transfer of energy from the photon to secondary electrons is given by the mass energy-transfer coefficient and is denoted by μ_{en}/ρ. The mass energy-transfer coefficient is the weighted sum of the mass attenuation coefficients; i.e.,

$$\mu_{en}/\rho = f_\tau(\tau/\rho) + f_\sigma(\sigma/\rho) + f_\kappa(\kappa/\rho) \tag{28}$$

The weights f_τ, f_σ, and f_κ indicate, for their respective interactions, the fraction of the photon energy which is converted into kinetic energy of secondary electrons and dissipated in the medium by collision losses. It is beyond the scope of this Chapter to detail the prescription for estimation of the weights. It is important to note that the weights reflect only the energy transferred as kinetic energy of charged particles and thus energy emitted as X-rays following photoelectric effect and the rest mass energy of the positron-electron pair in the pair formation process are excluded from the weight. It should be further noted that for the composition of body tissues and typical photon energies, the correction for bremsstrahlung energy loss by the secondary electrons is rather small.

7.3.5.4 Point-isotropic specific absorbed fraction

The fraction of the energy emitted by a point- isotropic source that is absorbed per unit mass at a distance x from the source the point isotropic specific absorbed fraction $\Phi(x)$ can be expressed as

$$\Phi(x) = \frac{\mu_{en}}{\rho}\frac{e^{-\mu x}}{4\pi x^2}B_{en}(\mu r) \tag{29}$$

where x is the distance from the point source, μ is the linear attenuation coefficient at the source energy, μ_{en} / ρ is the mass energy-transfer coefficient at the source energy, and $B_{en}(\mu r)$ is the energy-absorption buildup factor. The mass attenuation and mass energy-transfer coefficients for soft tissue as a function of photon energy are shown in Fig. 7.13.

The build up factor is defined as the ratio of the absorbed dose obtained from a measurement to the absorbed dose calculated to be due to "uncollided" photons at the location. The scattered photons are of lower energy than the uncollided photons and hence subject to increased absorption as seen in Fig. 7.13. Several tabulations of energy-absorption buildup data are available in the literature for application to body tissues. Berger presented such data in MIRD Pamphlet No. 2 [68B1] for a point source in water. Published values applicable to 40 mean free paths (μr = 40) have been published by Spencer and Simmons [73S1], whereas Berger's data were applicable to only 20 mean free paths. For small values of μr, $B_{en}(\mu r)$ is approximately unity and increases rapidly with increasing values of μr. The maximum value of the buildup factor occurs for photons of about 100 keV.

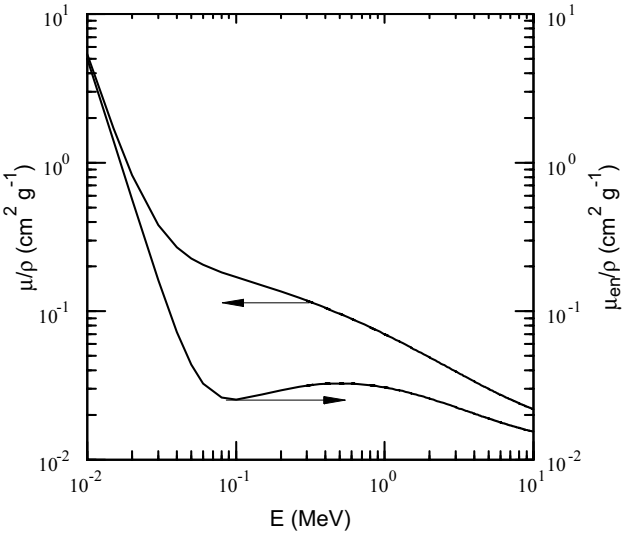


Fig 7.13. Mass attenuation and mass energy-transfer coefficients for photons in soft tissue; [95H1].

7.3.6 Calculation of doses to soft tissues and the skeleton

For the majority of tissues in which radionuclides deposit, following their entry into the blood, the assumption is made that both they and the sensitive cells are essentially uniformly distributed. On this basis the average tissue dose is calculated. This applies for example to the liver, spleen, kidneys, muscle, gonads and glands. In some cases, particles or colloids containing radionuclides may enter the blood and these will result in a heterogeneous distribution of activity in tissues in which they deposit, particularly for alpha emitters with a short path length (~ 50 μm). Most experimental evidence suggests, however, that a heterogeneous distribution of activity is no more likely to produce long term damage, such as cancer, than a homogeneous distribution of activity and the calculation of average tissue dose is justified [80I3, 99S1, 03M1]. In the case of the skeleton, however, it has been necessary to take account of the way radionuclides deposit in order to assess the dose to sensitive tissues.

A generalised model to assess doses from bone seeking radionuclides was given in ICRP Publication 30 [79I1] (Table 7.11). The target tissues in the skeleton are taken to be the active red bone marrow (RBM), which is present in cavities in trabecular bone, and endosteal and epithelial cells assumed to lie within 10 μm of bone surfaces (BS). Energy deposited in the yellow marrow of cortical bone is not considered to cause any radiation effects. The radiation dose to the RBM and BS depends upon the pattern of deposition of the radionuclides in the bone (volume or surface deposition), the radiation it emits (α, β, γ) and the effective half-time. Some bone surface seeking elements are Ga, Zr, Th, Pu, Am and Cm and some volume seekers Ca, Sr, Ra and U. The most recent biokinetic models for Pu, Am and Cm allow for their progressive burial in the skeleton (Section 7.2.5.3).

The total endosteal area of the skeleton over which the dose is calculated is taken as 12 m^2, half associated with cortical bone and half with trabecular bone. The layer on bone surfaces over which the equivalent dose is averaged has a mass of 120 g. The mass of the red bone marrow in cavities within trabecular bone is taken to be 1500 g. Table 7.11 gives the fraction of energy absorbed for α- and β-emitters deposited in the skeleton. Thus, for an α-emitter such as ^{239}Pu, which initially deposits on the bone surface, half the activity deposited in the skeleton is taken to be associated with trabecular bone surfaces and half to cortical bone surfaces. For activity on trabecular bone surfaces 25 % of the energy released will be absorbed by the sensitive 10 μm cell layer on bone surfaces (BS), 50 % will be absorbed in the red bone marrow (RBM); the remaining energy will be harmlessly dissipated in bone mineral. For activity on cortical bone surfaces 25 % of the energy released will be absorbed by cells on bone surfaces; the rest will be dissipated in yellow-marrow or in mineral bone. For radionuclides deposited in bone volume the fraction of energy deposited in sensitive cells, and hence the dose, will be less than for bone surface seekers as much of the energy will be dissipated in bone mineral. It should be noted that some recent analyses of radiation-induced bone tumours and information on bone cell development suggests that sensitive cells for bone tumour induction may reside at distances of more than 10 μm from bone surfaces and that a depth of 50 μm may be a more appropriate depth over which to calculate doses [00G1]. Preliminary calculations suggest that this may reduce the dose to cells near trabecular bone surfaces by a factor of about 2.

Table 7.11. Fraction of energy deposited in target organs for α- and β-emitters deposited on bone surfaces or in bone volume

Source organ	Target organ	α-emitter uniform in volume	α-emitter on bone surfaces	β-emitter on bone surfaces $E_\beta \geq 0.2$ MeV	β-emitter on bone surfaces $E_\beta < 0.2$ MeV	β-emitter uniform in bone
Trabecular bone	BS[a]	0.025	0.25	0.025	0.25	0.025
Cortical bone	BS	0.01	0.25	0.015	0.25	0.015
Trabecular bone	RBM[b]	0.05	0.5	0.35	0.5	0.35
Cortical bone	RBM	0.0	0.0	0.0	0.0	0.0

a BS = Bone surfaces; b RBM = Bone marrow

7.4 Dose coefficients

The previous Section described some of the fundamental principles used in the calculation of radiation dose. In practice many radiation protection professionals will not need to implement these methods in full, but will use published values of *dose coefficients* as a means of estimating doses to individuals or populations.

A dose coefficient is defined as a committed dose per unit intake (Sv Bq^{-1}), the term can be applied to either committed equivalent doses or committed effective doses. Dose coefficients are sometimes referred to as "dose per unit intake values". ICRP has adopted lowercase letters to denote dose coefficients. Thus, a committed equivalent dose to a tissue T is denoted H_T and the corresponding dose coefficient is denoted h_T, similarly the dose coefficients for effective doses (E) are denoted e. Dose coefficients provide a means of converting intakes (Bq) into committed doses (Sv) from internal emitters and are therefore used in many branches of radiological protection such as environmental assessments, the protection of workers and nuclear medicine.

As referred to earlier in this Chapter, ICRP has, over the last decade or so, published a number of documents giving dose coefficients for workers and members of the public, including children (Table 7.1). These Publications provide a consistent source of reference values. ICRP has also issued a publication giving doses to the embryo and foetus from intakes by the mother. In addition ICRP has given dose coefficients (usually expressed in mGy MBq^{-1}) for use in nuclear medicine. More details are given in Section 7.4.2.4.

7.4.1 Method of calculation

The biokinetic models recommended by ICRP are described in Section 7.2. These models describe the fate of an inhaled or ingested radionuclide as it enters the body, its subsequent absorption and distribution among systemic tissues and its elimination from the body. Therefore, in most cases these models are combined to form a complete model for predicting the behaviour of the radionuclide from initial entry into the body until excretion or decay. For example, the complete model for inhaled plutonium would require that the Human Respiratory Tract Model (HRTM) be combined with the systemic model for plutonium (for activity taken up to blood) and the model for the gastrointestinal tract (for activity mechanically cleared from the lung and swallowed and for activity excreted into the GI tract in bile). These model constructs are referred to as compartment models, each compartment representing an apparent influence on the kinetic behaviour of the radionuclide.

All the models recommended by ICRP to date describe the transfer of radionuclides (activity) between compartments of the model by linear first order processes. That is, the rate of biological removal of activity from a compartment at time t is taken to be the product of the activity in the compartment at time t and a transfer rate coefficient, usually denoted by k. Consider an isolated compartment i as in Fig. 7.14. Compartment i receives activity from and transfers activity to all other compartments in the model. The element $k_{j,i}$ of the transfer rate coefficient matrix describes the fraction of the activity in compartment i transferred to compartment j per unit time. The model is completely described by the transfer rate coefficients matrix and the initial activity (content at time zero) in each compartment. The nuclear transformations of many radionuclides form radioactive nuclei which must be considered in computing the dose coefficient. Thus the intake of a radionuclide, the parent, may result in a series of radionuclides being formed within the body. The kinetics of radioactive series must be superimposed on the kinetics described by the biokinetic models. The activity A_i^m of member m of the decay chain in compartment i is given by a set of coupled linear differential equations of the form of Equation 30.

$$\begin{aligned}\frac{d}{dt}A_i^m(t) &= \text{inflow} + \text{ingrowth} - \text{outflow} \\ &= \sum_{j=1,\, j\neq i}^{n} k_{i,j}^m\, A_j^m(t) + \lambda_m \sum_{m'=1}^{m-1} F_{m',m}\, A_i^{m'}(t) - A_i^m(t)\,(\lambda_m + \sum_{j=1,\, j\neq i}^{n} k_{j,i}^m)\end{aligned} \tag{30}$$

where $m = 1$ for the parent radionuclide, $k_{j,i}^m$ is the fraction of the activity of chain member m in compartment i transferred to compartment j per unit time, λ_m is the decay constant of chain member m, $F_{m',m}$ is the fraction of the decays of chain member m' which form member m (often referred to as the branching fraction) and by definition $A_i^{m'}(t) = 0$ if $m = 1$. In computing dose coefficients the set of differential equations are generally solved as an initial value problem such as specified in Eq. 31.

$$A_i^m(0) = \begin{cases} 0 \text{ for all } i \text{ and } m > 1 \\ 0 \text{ for } m = 1 \text{ and } i \text{ not a compartment of intake} \\ \text{nonzero for } m = 1 \text{ and } i \text{ as a compartment of intake} \end{cases} \tag{31}$$

For example, an intake by ingestion would assume that at time zero one unit of activity of the parent radionuclide is present in the stomach content and zero activity of the parent and daughter products is present in all other compartments of the model.

Two approaches have been applied to describe the biokinetics of the daughter products. The so-called *shared kinetics* assumes that the behaviour of the daughter is the same as the parent. That is, $k_{i,j}^m = k_{i,j}^1$, $m = 2,..., n$. If information indicates that the daughter product behaves differently from the

parent radionuclide in the body then *independent kinetics* are applied in Equation 30. The well recognized independent kinetic cases include the formation of noble gas radionuclides in the decay of solid daughter parents, e.g., ${}^{83}_{35}\mathrm{Br} \rightarrow {}^{83m}_{36}\mathrm{Kr}$ and radioiodine formed in the decay of radioisotopes of tellurium, e.g, ${}^{133}_{52}\mathrm{Te} \rightarrow {}^{133}_{53}\mathrm{I}$. However other cases of importance include the long decay series associated with some of the natural decay chains (such as ${}^{228}_{90}\mathrm{Th}$) where fundamentally different behaviours of the chain members in the skeleton are encountered.

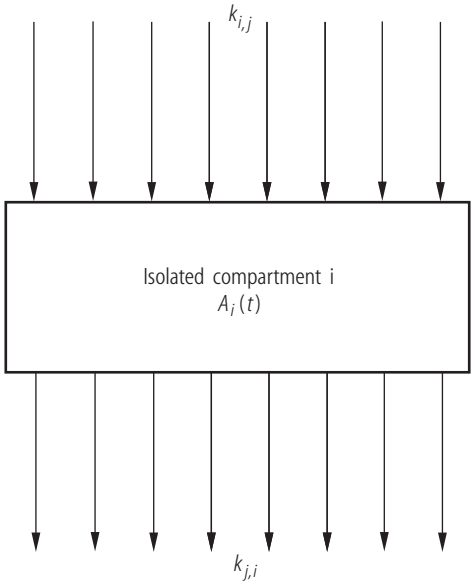


Fig 7.14. Isolated compartment exchanging activity with other compartments in the model.

The biological removal coefficient of the activity of chain member m from i^{th} compartment is given by $\sum_{j=1,\, j\neq i}^{n} k_{j,i}^{m}$ which can be stated as a half-time by dividing ln(2) by the removal coefficient. This would be the half-time for removal if there were no input into the compartment from other compartments. If the compartment is subject to continued input then a plot of the compartment activity as a function of time will reflect the removal rates from all compartments feeding the i^{th} compartment. Thus in general one has to be careful in speaking about biological half-times for complex models.

With the exception of a catenary model (a model in which the compartments only communicate with adjacent compartments; that is, the nonzero members of transfer coefficient matrix are $k_{1,2}$, $k_{2,3}$, $k_{3,4}$, ..., $k_{n-1,n}$) no closed form solution exists for Equation 30. The catenary form of Equation 30 is well-known in radiation protection as it describes the serial kinetics of a decay chain and its solution was formulated in earlier work by Bateman in 1910 [10B1] who was assisting Lord Rutherford in the early investigations of radioactivity. Catenary models were used in ICRP Publication 2 [59I1] and their solution has been extensively investigated by Skrable [74S1].

Since only the catenary system or very small systems (less than four compartments) can be solved exactly, it is necessary to approximate the solutions numerically. Applicable numerical approaches include analysis of eigenvalues and discrete variable methods (also called step-by-step methods or difference methods). With the advent of powerful desktop computing the discrete variable Runge-Kutta method can be readily applied to Equation 30 (see for example 88B1). However well-written variable-order, variable-step routines such as the solver developed by Gear [71G1] are more efficient than a fixed-order, variable-step Runge-Kutta routine. The calculations of Publication 30 [80W1] were carried out using Hindmarsh's coding of the Gear method [74H1]. A collection of state-of-the-art solvers for the initial value problem for ordinary differential equation systems, including the Gear method, are contained in the widely available ODEPACK package [83H1]. Classical methods from linear algebra involve the

eigenvalues and eigenvectors of the matrix of transfer rate coefficients as described in good texts on linear algebra (for example 85J1). The methods are referred to as the matrix exponential method and eigenanalysis. Birchall and James [89B1] described the matrix exponential method and listed the source code for effective implementation of this method on a desktop computer. The eigenanalysis method has been implemented by, among others, Killough and Eckerman [84K1], Bertelli [87B1] and Polig [01P1]. A transportable library of state-of-the art numerical routines for eigenvalue problems is available in the LAPACK Package [00A1]. An advantage of the eigenanalysis approach is that the solution can be expressed analytically even though it is a numerical approximation. An imaginative hybrid numerical-analytical method has been developed by Leggett and co-workers that is extremely simple, flexible and highly efficient [93L1]. Some of these methods have been reviewed by Peace [03P1]. With the memory and speed available in today's desktop computers and the availability of well written solvers, such as those within ODEPACK, the numerical aspects of compartment modelling are no longer a significant issue. However, having available a number of different solvers is useful in ensuring an appropriate approximation to the solution.

The method of calculation of dose is described in Section 7.3 and is encapsulated in Equation 15. The number of nuclear transformations of the k^{th} chain member in the i^{th} compartment during the period t_1 to t_2 $U_i^k(t_1,t_2)$ is given as

$$U_i^k(t_1,\ t_2)=\int_{t_1}^{t_2} A_i^k(u)\,\mathrm{d}u \tag{32}$$

where $A_i^k(t)$ is obtained as the solution to Equation 30. For protection of workers the integral of Equation 32 is evaluated from time zero (the time of the intake) to 50 years post intake and the integral is denoted as $U(50)$. The distinction between $\tilde{A}$ typically used in the dosimetry of radiopharmaceuticals and U is that former has no restriction on the upper limit of the integration since it is typically short-lived radionuclides. The committed equivalent dose coefficient for tissue T, h_T, for the worker is thus computed as

$$h_T=\sum_k\sum_S U_S^k(50)\ \ SEE(\mathrm{T}\leftarrow\mathrm{S})^k \tag{33}$$

where the outer summation extends over the parent radionuclide and all members of its decay chain and the inner summation extends over all source regions in which activity may reside. Note that the compartments in the model must be assigned to the anatomical source regions S in computing U_S. For children both the parameters of the biokinetic models and S-factors (or SEE values) change with time. They must therefore be varied in an effectively continuous manner from the time of intake to age 70 years.

Absorbed fraction data for photons and charged particles are available for six specified ages enabling sets of S-factors to be generated for newborn, 1, 5, 10, 15-years-olds and adults. At intermediate ages S-factors can be derived using an interpolation scheme. ICRP has used a linear interpolation in the inverse total body mass domain. Biokinetic models are specified by ICRP for six standard ages, 3-month-old, 1, 5, 10, 15-year-old, and adult. These models specify the transfer coefficients ($k_{i,j}$) which determine the rates at which material is transferred between the different parts of the body (Equation 30). Transfer coefficients at intermediate ages can be derived using linear interpolation. Numerical methods, which are characterised by the variable time steps inherent in the method, can easily accommodate time-varying transfer coefficients and S-factors. For methods which apply an analytical solution the continuous variation of transfer rates must be modelled discretely using a time-stepping method. Starting at the age of intake the calculation is advanced by a time-step small enough for the interpolated rates to be held constant without undue loss of accuracy. The computed activities at the end of one time-step are used as the initial conditions for the next step. In this way the number of transformations within each step or interval is computed. S-factors calculated at the beginning or mid-point of a step are taken to apply to the

whole interval. The committed dose is then the sum of the doses computed in each time step. In essence the numerical methods are integrating dose rate (Equation 15) while analytical methods apply Equation 18 to a series of larger time steps. One of the advantages of the analytical methods is in calculations for adults where rate constants and *S*-factors are constant; the time step can then be the whole of the period of interest, e.g. 50 years. Numerical methods are advantageous, however, where the intake is complicated, perhaps varying in a difficult manner with time.

7.4.2 Sources of dose coefficients

The recommendations of ICRP and other international bodies advance on different fronts at different rates. This means that it is not always straightforward to identify the most appropriate dose coefficients for a particular application (e.g. workplace, environment, nuclear medicine). For example, for inhalation by members of the public, dose coefficients are given for three lung absorption Types (F, M and S) for 31 important elements (Publication 72, 96I1). A default Type is specified for use when the chemical form is not known. For workers a comprehensive review of lung absorption characteristics for various chemical forms of radionuclides has not been undertaken since Publication 30 although updated dose coefficients using the HRTM have been issued [94I1]. The following subsections aim to help the reader identify the most appropriate dose coefficients at the time of publication of this review.

The results given in ICRP dose compendia take into account the ingrowth of decay products in all regions of the body following an intake of unit activity of the parent nuclide. They do not take into account any activity of decay products in the initial intake. Thus doses from any radioactive decay products present at the time of the intake (perhaps in equilibrium with the parent) may need to be added to the dose from the parent nuclide.

7.4.2.1 Workers

A compendium of dose coefficients (committed effective dose to 50 years after the intake) for workers for over 800 nuclides based on the HRTM model (Section 7.2.1), the ICRP Publication 30 model for the GI tract (Section 7.2.2) and the most recent ICRP systemic models (Section 7.2.5) has been issued in ICRP Publication 68 [94I1] which implemented the tissue weighting factors in Publication 60 [91I1] (Table 7.1). These results are also given in the Euratom Directive [96E1] and the IAEA Basic Safety Standards [96I1]. The previous complete source of doses to workers was ICRP Publication 30, published in four parts between 1979 and 1988 [79I1, 80I1, 80I2, 88I1]. Publication 30 concentrated on giving results as Annual Limits on Intake (Bq) rather than as dose coefficients.

For workers, inhalation dose coefficients are based on an AMAD of 5 μm and specified distributions of time spent at two levels of exercise – sitting and light exercise [94I2].

In support of Publication 68, ICRP has issued Publication 78 [97I2] which contains bioassay predictions, such as daily urinary excretion and retention in lung, skeleton and whole-body, based on the same models used in Publication 68 for a limited range of radionuclides. This enables health physicists to use a consistent set of models in dose assessments. This document fills the role for ICRP Publication 68 that Publication 54 filled for Publication 30. Phipps et al [98P1] provide extended results and fitted functions for predicting bioassay quantities at times not addressed in the ICRP report. For particle sizes other than 5 μm AMAD, Ishigure et. al. calculated bioassay quantities for 0.1, 0.3, 1, 3 and 10 μm and have uploaded the results onto the National Institute of Radiological Sciences web site [02I4]. Further details of the methods of monitoring and dose assessment in the workplace are given in Sections 7.4-7.7 and in Chapter 10, Sections 10.3.2. and 10.3.3.

A CD-ROM of dose coefficients for both members of the public and workers has been issued by ICRP [99I1]. It is consistent with the Publication 68 and it extends the results given in Publications 68 and 72 by giving inhalation dose coefficients for ten particle sizes (0.001, 0.003, 0.01, 0.03, 0.1, 0.3, 1, 3, 5, 10 μm AMAD) and ingestion coefficients. Effective doses and equivalent doses for all important tissues for a

range of integration periods (1, 7, 30 days, 1, 5, 10, 20, 30 and 45 years) are given, together with the dose coefficients to age 70 years. The package also contains extensive help files and the text of Publications 68 and 72.

7.4.2.2 Members of the public

Following the Chernobyl accident it was acknowledged that a set of internationally-agreed dose coefficients was required. Thus, ICRP provided age-specific biokinetic models for selected radionuclides in Publications 56, 67, 69, 71 and 72 [89I1, 93I1, 95I1, 95I2, 96I1] together with dose coefficients (committed effective dose to age 70 years) for six age groups: 3-month-old infants, 1-, 5-, 10-, 15-year old children and adults (Table 7.1). More details about the models themselves are given in Section 7.2. ICRP Publication 56 [89I1] considered H, C, Sr, Zr, Nb, Ru, I, Cs, Ce, Pu, Am and Np. Dose coefficients for selected radionuclides were based on the tissue weighting factors of ICRP Publication 26 [77I1] and the lung model of ICRP Publication 30. These results have now been superseded by other Publications. ICRP Publication 67 [93I1] considered S, Co, Ni, Zn, Mo, Tc, Ag, Te, Ba, Pb, Po, and Ra, and in addition revised some of the models given earlier in Publication 56. In particular the model for Sr was revised substantially and made consistent with the generic model structure used for Ba and Ra. By the time of the issue of ICRP Publication 67 the tissue weighting factors given in Publication 60 were available thus the dose coefficients are consistent with the most recent ICRP recommendations. Both equivalent (h_T) and effective (e) dose coefficients were given and the results of ICRP Publication 56 were updated whether or not the systemic model was updated in Publication 67. Only ingestion was considered as a route of intake. Publication 69 [95I1] gave a similar range of dose coefficients for radionuclides of Fe, Se, Sb, Th and U.

Following the publication of the model for the Human Respiratory Tract [94I2] ICRP Publication 71 reviewed the lung absorption characteristics of environmental forms of the 31 elements considered in Publications 56, 67, 69. ICRP Publication 71 also introduced biokinetic models for Ca and Cm. Inhalation dose coefficients based on the new model were given for all three default Types (F, M and S) and one of these three was recommended as a default for situations where the chemical form is unknown. Details of the HRTM model are given in Sction 7.2.1. For members of the public, dose coefficients are based on an Activity Median Aerodynamic Diameter, AMAD, of 1 μm and specified distributions of time spent at four levels of exercise (sleep, sitting, light exercise and heavy exercise [96I2]). Dose coefficients for ingestion were not given in Publication 71.

A large compendium of inhalation and ingestion dose coefficients (effective dose only) for members of the public for over 800 nuclides based on the HRTM model (Section 7.2.1), the ICRP Publication 30 model for the gastrointestinal tract (Section 7.2.2) and the most recent systemic models (Section 7.2.5) was published in ICRP Publication 72 [96I1]. These results are also given in the Euratom Directive [96E1] and the IAEA Basic Safety Standards [96I2]. The CD-ROM of dose coefficients (Section 7.4.2.1, 99I1) also contains doses for members of the public consistent with ICRP Publication 72 [99I1]. As noted above, for 31 elements dose coefficients are given for three default absorption Types (F, M and S), while for the remaining elements it is assumed that compounds assigned to the Publication 30 classes (D, W and Y) are categorised as F, M and S respectively; thus for guidance about individual chemical forms of these elements one must refer to ICRP Publication 30.

7.4.2.3 Embryo and foetus

ICRP has issued dose coefficients for the embryo and foetus in Publication 88 [01I1]. More details on the biokinetic models used for the dose calculations are given in Section 7.2.7, this subsection therefore covers only the phantoms used in foetal dosimetry and some aspects of the dosimetry.

The pattern of energy deposition within the foetus is modelled in Publication 88 [01I1] using results from two separate sets of computer phantoms developed at Oak Ridge National Laboratories, USA (ORNL). The first is a series for the pregnant female developed by Stabin et al [95S1]; the second is for the foetus itself, developed by Eckerman [03E1]. Energy-dependent specific absorbed fractions (SAFs)

are given for both electrons (including beta particles and positrons) and photons. Due to the small organ masses and short ranges between organs, for beta radiation the beta spectrum is used and not the mean energy. In calculating organ and tissue doses for infants, children and adults, electrons have, in most cases, been assumed to be non-penetrating, i.e. their energy is taken to be absorbed entirely in the organ or tissue in which they are emitted (exceptions are the skeleton and walled organs). In the case of the foetus, however, the extremely small size of some tissues can mean that a substantial fraction of electron energy can be deposited outside the tissue where the electron is emitted [98U1]. Thus, activity in the foetal thyroid could deliver an electron dose to nearby tissues such as the brain. However, these so-called cross-fire doses are generally much lower than the doses to the source tissues themselves.

7.4.2.4 Nuclear medicine patients and volunteers in clinical research

ICRP Publication 53 [87I1] presented biokinetic models and best estimates of biokinetic data for some 120 individual radiopharmaceuticals. It included absorbed dose coefficients for organs and effective dose equivalent coefficients (Publication 30 [79I1] terminology) calculated up to infinity, due to the administration of short-lived radionuclides for diagnostic or experimental purposes. The calculations used ICRP's Publication 26 tissue weighting factors [77I1]. Some information on the range of variation to be expected in pathological states, for adults, children and the foetus were given. The absorbed dose coefficients are used in clinical diagnostic work for judging the risk associated with the use of specific radiopharmaceuticals, both for comparison with the possible benefit of the investigation and to help in giving adequate information to the patient. These estimates also provide guidance to ethics committees having to decide upon research projects involving the use of radioactive substances in volunteers who receive no individual benefit from the study. ICRP Publication 53 supplemented ICRP Publication 52, Protection of the Patient in Nuclear Medicine [88I3].

In Publication 80 [98I1], ICRP provided biokinetic models, absorbed doses to organs and effective dose coefficients, using the tissue weighting factors of ICRP Publication 60, for 10 new radiopharmaceuticals. It also provided recalculated dose coefficients for the 19 most frequently used radiopharmaceuticals from ICRP Publication 53, using ICRP Publication 60 dosimetry. An integrated index to all radiopharmaceuticals treated in ICRP Publications on nuclear medicine up to 1998 gave a listing of effective dose coefficients for adults.

Recently Stabin and Siegel [03S1] have used the best current radiation decay data and computer phantoms to calculate dose coefficients for use in nuclear medicine. Decay data for over 800 radionuclides from the data service at Brookhaven National Laboratory were combined with absorbed fraction data from a number of currently available mathematical whole body and organ models to calculate the dose coefficients. Many more (816) radionuclides are considered than in the ICRP compendia and some alpha emitters are included. New models are also employed, and dose coefficients for bone and marrow have been updated with recently suggested modifications.

7.4.3 Dose coefficients for selected radionuclides

7.4.3.1 Doses to tissues following intakes of radionuclides

Equivalent doses to tissues following inhalation of $^{239}PuO_2$ by an occupationally exposed worker are given in Table 7.12. These doses are calculated using the HRTM [94I1], assuming inhalation Type S and a 5 μm AMAD aerosol, and the biokinetic model for plutonium given in Publication 67 [91I1]. The highest committed (50 year) doses are to the lung, as the site of entry into the body, and the skeleton (cells near bone surfaces) and liver, as the main sites of deposition from the blood. ICRP also calculates the committed effective dose which provides a method for comparing doses, and hence risks, from intakes of radionuclides with those from external radiation. This is discussed in detail in Chapter 4.

Table 7.12. Committed equivalent doses to tissues, weighted committed equivalent doses and committed effective doses from inhalation by a worker of $^{239}PuO_2$ (5 μm AMAD)[a]

Organ or tissue	Committed equivalent dose [Sv Bq^{-1}]	Tissue weighting factor	Weighted committed equivalent dose [Sv Bq^{-1}]
Cells near bone surfaces	9.1×10^{-5}	0.01	9.1×10^{-7}
Colon	1.7×10^{-7}	0.12	2.0×10^{-8}
Liver	1.9×10^{-5}	0.05	9.5×10^{-7}
Lungs	4.7×10^{-5}	0.12	5.6×10^{-6}
Red bone marrow	4.5×10^{-6}	0.12	5.4×10^{-7}
Remainder	2.0×10^{-7}	0.05	1.0×10^{-8}
Skin	1.5×10^{-7}	0.01	1.5×10^{-9}
Stomach	1.5×10^{-7}	0.12	1.8×10^{-8}
Gonads	1.2×10^{-6}	0.20	2.4×10^{-7}
Committed effective dose			8.3×10^{-6}

a Inhalation Type S

Examples of equivalent dose coefficients to tissues from inhalation intakes of ^{131}I, ^{137}Cs and ^{239}Pu by workers are given in Table 7.13. For an intake of ^{131}I the main dose is to the thyroid, with a dose that is rather more than 1000 times that to other tissues. For ^{137}Cs, which moves rapidly from the lungs to blood (Type F) and distributes throughout the body, most tissues receive a very similar dose. In the case of ^{239}Pu oxide, which has a longer retention time in the lung (Type S) and deposits principally in the liver and skeleton there is a greater range of doses although the long retention time in the skeleton results in the highest tissue dose to cells near bone surfaces, as described above.

Table 7.13. Doses to tissues following inhalation by a worker of ^{131}I, ^{137}Cs and ^{239}Pu

Committed equivalent dose [Sv Bq^{-1}]			
Tissue	Iodine-131 (Type F)	Caesium-137 (Type F)	Plutonium-239 (Type S)[b]
Thyroid	2.1×10^{-7}	6.3×10^{-9}	1.5×10^{-7}
Red bone marrow	5.5×10^{-11}	6.3×10^{-9}	4.5×10^{-6}
Cells near bone surfaces	6.9×10^{-11}	6.6×10^{-9}	9.1×10^{-5}
Colon	5.1×10^{-11}	8.1×10^{-9}	1.7×10^{-7}
Lungs	8.1×10^{-11}	6.1×10^{-9}	4.7×10^{-5}
Liver	2.4×10^{-11}	6.5×10^{-9}	1.9×10^{-5}
Committed effective dose	1.1×10^{-8}	6.7×10^{-9}	8.3×10^{-6}

a For inhalation of 5 μm AMAD aerosol

b Assumes $w_R = 20$ for α particles

Some examples of dose coefficients for different radionuclides following intakes by inhalation and ingestion are shown in Table 7.14. The variation in doses reflects differences in behaviour after intakes by inhalation or ingestion, variations in distribution and retention in tissues as well as the use of a radiation weighting factor w_R of 20 in the calculation of equivalent dose to tissues from deposited α emitters. The lowest doses are from intakes of tritiated water and the highest doses from inhalation of the α emitters ^{224}Ra, ^{226}Ra and ^{241}Am.

Table 7.14. Comparison of dose coefficients following inhalation or ingestion of various radionuclides by a worker.

Radionuclide	Lung Type[a]	f_1[c]	Dose coefficient [Sv Bq^{-1}]	
			Inhalation	Ingestion
$^{3}H_2O$	–[b]	1.0	1.8×10^{-11}	1.8×10^{-11}
^{60}Co	M	0.1	7.1×10^{-9}	3.4×10^{-9}
^{90}Sr	F	0.3	3.0×10^{-8}	2.8×10^{-8}
^{95}Zr	F	0.002	3.0×10^{-9}	8.8×10^{-10}
^{95}Nb	M	0.01	1.3×10^{-9}	5.8×10^{-10}
^{106}Ru	F	0.05	9.8×10^{-9}	7.0×10^{-9}
^{131}I	F	1.0	1.1×10^{-8}	2.2×10^{-8}
^{134}Cs	F	1.0	9.6×10^{-9}	1.9×10^{-8}
^{137}Cs	F	1.0	6.7×10^{-9}	1.3×10^{-8}
^{144}Ce	M	5×10^{-4}	2.3×10^{-8}	5.2×10^{-9}
^{210}Po	F	0.1	1.1×10^{-6}	6.8×10^{-7}
^{224}Ra	M	0.2	2.4×10^{-6}	6.5×10^{-8}
^{226}Ra	M	0.2	1.2×10^{-5}	2.8×10^{-7}
^{232}Th	M	5×10^{-4}	2.9×10^{-5}	2.2×10^{-7}
^{234}U	M	0.02	2.1×10^{-6}	4.9×10^{-8}
^{235}U	M	0.02	1.8×10^{-6}	4.6×10^{-8}
^{238}U	M	0.02	1.6×10^{-6}	4.4×10^{-8}
^{239}Pu	S	1×10^{-5}	8.3×10^{-6}	9.0×10^{-9}
^{241}Am	M	5×10^{-4}	2.7×10^{-5}	2.0×10^{-7}
^{242}Cm	M	5×10^{-4}	3.7×10^{-6}	1.2×10^{-8}

a Inhaled materials are classified as Type F, M or S (Fast, Moderate or Slow) which refer to their rates of absorption to blood from the respiratory tract (Section 7.2.1.2). AMAD = 5 μm.
b Tritiated water is assumed to be completely absorbed from the lungs
c Fractional absorption from the gut

7.4.3.2 Application of dose coefficients in risk calculations

The doses to tissues calculated using the dosimetric models developed by ICRP together with risk coefficients for fatal cancer recommended by ICRP for the different tissues [91I1] can be used for assessing the consequences of intakes of radionuclides. An example is given in Table 7.15 for ^{239}Pu inhaled as the oxide (inhalation Type F, AMAD = 5 μm) by a worker.

Table 7.15. Estimation of risk of cancer death following inhalation of $^{239}PuO_2$ (AMAD 5 μm)

Tissue	Cancer	Sv Bq^{-1} inhaled	Risk coefficient [cancer deaths Sv^{-1}][a]	Risk for 100 kBq inhaled
Lungs	Lung	4.7×10^{-5}	6.8×10^{-3}	1 in 31
Liver	Liver	1.9×10^{-5}	1.2×10^{-3}	1 in 430
Cells near bone surfaces	Bone tumour	9.1×10^{-5}	4.0×10^{-4}	1 in 280
Red bone marrow	Leukaemia	4.5×10^{-6}	4.0×10^{-3}	1 in 550
Colon	Colon	1.7×10^{-7}	6.8×10^{-3}	1 in 8,300
Committed effective dose		8.3×10^{-6}	5.0×10^{-2}	1 in 26[b]

a Risk coefficients for workers [91I1]
b Based on risks calculated to individual tissues

The ICRP dosimetric model for plutonium can be used to estimate the committed equivalent dose to tissues following an intake by inhalation. By weighting these doses by the appropriate risk coefficients [91I1], the consequences of an intake of $^{239}PuO_2$ can be estimated (Table 7.15). The calculations have been undertaken using the HRTM [94I2] and assuming an AMAD of 5 μm (the default value for workers). The biokinetic model for plutonium given in ICRP Publication 67 (93I1, Figure 7.7) has also been used.

Following the inhalation of 100 kBq of $^{239}PuO_2$, for example, the risk of developing lung cancer would be $(4.7\times10^{-5}) \times (6.8\times10^{-3}) \times 10^5 = 3.2\times10^{-2}$ i.e. a 1 in 31 risk. The risks of leukaemia, liver and bone cancer would all be lower, about 1 in 550, 1 in 430 and 1 in 280 respectively. The overall risk of developing cancer would be about 1 in 26. These risks are clearly a maximum as they are based upon committed doses and depend upon the full risk to the tissues being expressed. They would therefore apply only to intakes received early in life. A similar, but approximate calculation could be carried out using the committed effective dose (8.3×10^{-6} Sv Bq^{-1}) multiplied by the risk coefficient for whole body radiation exposure recommended by ICRP (5×10^{-2} Sv^{-1}) given in Publication 60 [91I1]. For 100 kBq inhaled this would give an overall risk of 4.15×10^{-2} (i.e an overall risk of 1 in 24). This is very similar to the value given in Table 7.15, the difference being mainly due to the fact the ICRP uses rounded values of tissue weighting factors w_T to calculate effective doses. If a more specific calculation was needed for an individual then it would be necessary to consider the accumulation of dose by the individual over time (i.e. year on year) and how that risk would be expressed.

Using the same approach the overall risk of developing cancer following either the inhalation or ingestion of 100 kBq of some of the radionuclides for which dose coefficients are given in Table 7.14 are given in Table 7.16. There is a significant difference in the risk from intakes of α-emitters compared with β/γ-emitters, reflecting the much higher w_R value of 20 for α particles. In general the risks of inhalation intakes are higher than for ingestion because of the lower absorption and faster rate of clearance from the gut than from the respiratory system. This is not the case for radionuclides such as tritium (as HTO) or caesium that are readily absorbed from the gut. For the same activity, the risk associated with ingestion of $^{239}PuO_2$ is nearly three orders of magnitude less than that following inhalation because of the low absorption in the gut ($f_1 = 10^{-5}$). For ingestion of 100 kBq of ^{131}I the risk is about 1 in 38,000, the majority of the risk of radiation-induced cancers predicted being in the thyroid (more than 99 %). Following inhalation of the same amount of activity the risk is somewhat lower (1 in 78,000) as only about half of the activity inhaled is deposited in the respiratory system. Similar considerations apply to the inhalation and ingestion of ^{137}Cs, although in this case, as the radionuclide distributes throughout body tissues the risk of cancer will be distributed amongst a number of tissues, with the greatest risks being for leukaemia and lung cancer. Of the radionuclides considered, ^{226}Ra and ^{232}Th are the most toxic with risks of about 1 in 7.4 and 1 in 10 respectively of developing fatal cancer following inhalation of 100 kBq. At the other extreme ^{3}H is the least toxic with a risk of less than 1 in a million.

7.5 Internal monitoring

This Section describes the general principles for individual monitoring. Sections 10.3.2 and 10.3.3 in Chapter 10 give more detailed information on *in vivo* measurements by whole and partial body counting as well as by analyses of excreta. This Section is reproduced from ICRP Publication 78, 97I2, paras. 58 - 76.

Table 7.16. Comparison of risk of radiation-induced cancer death associated with inhalation or ingestion of 100 kBq of some radionuclides.

Radionuclide	Lung[a] Type	f_1[c]	Risk of cancer death	
			Inhalation	Ingestion
$^{3}H_2O$	-[b]	1.0	1 in 1.4×10^{7}	1 in 1.4×10^{7}
^{90}Sr	F	0.3	1 in 9,300	1 in 9,700
^{95}Zr	F	0.002	1 in 86,000	1 in 170,000
^{95}Nb	M	0.01	1 in 150,000	1 in 310,000
^{106}Ru	F	0.05	1 in 22,000	1 in 18,000
^{131}I	F	1.0	1 in 78,000	1 in 38,000
^{137}Cs	F	1.0	1 in 37,000	1 in 19,000
^{144}Ce	M	3×10^{-4}	1 in 9,600	1 in 22,000
^{210}Po	F	0.1	1 in 310	1 in 870
^{224}Ra	M	0.2	1 in 64	1 in 3,000
^{226}Ra	M	0.2	1 in 7.4	1 in 1,200
^{232}Th	M	5×10^{-4}	1 in 10	1 in 1,300
^{239}Pu	S	1×10^{-5}	1 in 26	1 in 18,000
^{241}Am	M	5×10^{-4}	1 in 12	1 in 1,100
^{242}Cm	M	5×10^{-4}	1 in 46	1 in 15,000

a Inhaled materials are classified as Type F, M or S (Fast, Moderate or Slow) which refer to their rates of absorption to blood from the respiratory tract (Section 7.2.1.2). AMAD = 5 μm

b Tritiated water is assumed to be completely absorbed from the lungs

c Fractional absorption from the gut

7.5.1 Methods of individual monitoring

The purpose of this Section is to describe briefly the main measurement techniques, their advantages and their limitations. In most cases, individual monitoring for intakes of radionuclides may be achieved by body activity measurements, excreta monitoring, air sampling with personal air samplers, or a combination of these techniques. The choice of measurement technique will be determined by several factors: the radiation emitted by the radionuclide; the biokinetic behaviour of the contaminant; its retention in the body taking account of both biological clearance and radioactive decay; the required frequency of measurements; and the sensitivity, availability, and convenience of the appropriate measurement facilities.

Routine monitoring programmes usually involve only one type of measurement if adequate sensitivity can be achieved. For some radionuclides, only one measurement technique is feasible, e.g. urine monitoring for intakes of tritium. For radionuclides, such as plutonium isotopes, that present difficulties for both measurement and interpretation, a combination of techniques has to be employed. If different methods of adequate sensitivity are available, the general order of preference in terms of accuracy of interpretation is: body activity measurements; excreta analysis; personal air sampling. Results of monitoring of the working environment (area monitoring) may provide information that assists in interpreting the results of individual monitoring, e.g. information on particle size, chemical form and solubility, time of intake. The results of workplace monitoring for air contamination may sometimes be used to estimate individual intakes. However the interpretation of the results of measurements from air sampling in terms of intake is not simple and may be misleading. The most common form of representative sampling is by using fixed samples at a number of selected locations intended to be reasonably representative of the breathing zone of the worker. When such a method is routinely used for quantitative determinations of intake, the representativeness of the results should be determined using a special monitoring programme, often involving personal air samples.

Monitoring in relation to a particular task or event may often involve a combination of techniques so as to make the best possible evaluation of an unusual situation, for example, a programme of both body activity and excreta measurements and, in some circumstances, personal air sampling. In some cases of suspected incidents, screening techniques (such as measuring nose blow samples or nasal smears) may be employed to give a preliminary estimate of the seriousness of the incident. In these cases the regional deposition for ET_1 can be used to confirm that an intake has occurred and to give a rough estimate of the intake.

7.5.1.1 *In Vivo* measurements

The IAEA [96I2] has given guidance on the direct measurement of body content of radionuclides. Direct measurement of body or organ content provides a quick and convenient estimate of activity in the body. It is feasible only for those radionuclides emitting radiation that can escape from the body. In principle, the technique can be used for radionuclides that emit: X- or γ-radiation; positrons, since they can be detected by measurement of annihilation radiation; energetic β-particles that can be detected by measurement of bremsstrahlung; some α-emitters that can be detected by measurement of the characteristic X-rays.

Many facilities for the measurement of radionuclides in the whole body or in regions of the body consist of one or a number of high efficiency detectors housed in well-shielded, low-background environments [96I2]. The geometrical configuration of the detectors is arranged to suit the purpose of the measurement, e.g. the determination of whole-body activity or of activity in a region of the body such as the thorax or the thyroid. The skull or knees may be used as a suitable site for measurement of radionuclides in the skeleton.

Care must be taken to remove surface contamination before body activity is measured. For routine measurements, determination of whole-body content is often adequate for radiological protection purposes. Total body activity will then consist of systemic activity and activity in the gastrointestinal and respiratory tracts. However, in special investigations, or in interpretation of unusual measurements, it may be advantageous to determine the distribution within the body either by profile scanning or by analysis of the relative response of detectors placed at different positions along the body.

Commonly encountered fission and activation products, such as ^{131}I, ^{137}Cs and ^{60}Co, can be detected with comparatively simple equipment at levels that are adequate for radiation protection purposes. Such simple equipment may consist of a single detector, viewing the whole body or a portion of the body, or, for iodine isotopes, a small detector placed close to the thyroid. The advantage of simple equipment is that it may be operated at the place of work, thereby avoiding the time required to visit a remote whole-body monitoring facility. Measurements may then be made more frequently so that any unusually large intake would be recognised soon after it had occurred.

In contrast, high sensitivity techniques are needed for monitoring a few radionuclides at the levels that are required for protection purposes. Examples are the α-emitting radionuclides such as plutonium isotopes.

Until recently, most body activity measurement facilities, whether high-sensitivity or simple systems, used thallium-activated sodium iodide detectors. These have the advantage that crystals of large volume can be manufactured and so provide high efficiency for detection of γ-rays. Interpretation of a γ-ray energy spectrum obtained from a mixture of radionuclides may, however, raise some difficulties. The components of the spectrum can be resolved by a multiple linear regression analysis technique, but this requires previous calibration of the detection equipment with standard sources of the required radionuclides dispersed in a matrix in such a way as to simulate the distribution and attenuation within the body. The increasing availability of high-efficiency germanium detectors is leading to their use in situations where workers may be exposed to mixtures of γ-ray emitting radionuclides. The superior energy resolving power of these detectors simplifies the interpretation of spectra obtained from complex mixtures of radionuclides.

The activity present in a wound can be easily detected with conventional β-γ detectors if the contaminant emits energetic γ-rays. In the case of contamination with α-emitting radionuclides, detection is more difficult since the low energy X-rays that follow the α-decay will be severely attenuated in tissue;

this effect is more important the deeper the wound. It is often necessary to localise the active material and this requires a well-collimated detector. Wound monitors most have an energy discrimination capability if a good estimate is to be made of contamination with mixtures of radionuclides.

7.5.1.2 Analysis of excreta and other biological materials

In some cases, excreta monitoring may be the only measurement technique for those radionuclides which have no γ-ray emission or which have only low energy photon emissions. Excreta monitoring programmes usually involve analysis of urine, although faecal analysis may be required in some circumstances, for example where an element is preferentially excreted via faeces or to assess clearance of Type-S material from the respiratory tract. Other samples may be analysed for specific investigations. Examples are nose blow or nasal smears as routine screening techniques or blood, in the case of suspected high level contamination.

The collection of urine samples involves three considerations. Firstly, care must be taken to avoid adventitious contamination of the sample. Secondly, it is usually necessary to assess the total activity excreted in urine per unit time from the sample provided. For most routine analyses, a 24 h collection is preferred but, if this is not feasible, it must be recognised that smaller samples may not be representative. Tritium is a particular case for which it is usual to take only a small sample and to relate the measured activity concentration to the concentration in body water. Thirdly, the volume required for analysis depends upon the sensitivity of the analytical technique. For some radionuclides, adequate sensitivity can be achieved only by analysis of several days' excreta.

The analysis of faecal samples for routine monitoring involves uncertainty in interpretation owing to daily fluctuations in faecal excretion. Ideally, therefore, collection should be over a period of several days. However, this may be difficult to achieve in practice and interpretation may need to be based on a single sample. Faecal monitoring is more often used in special investigation, particularly following a known or suspected intake by inhalation of Type M or insoluble S compounds. In these circumstances measurement of the quantity excreted daily may be useful in the evaluation of clearance from the lungs and in the estimation of intake. Early results may be useful in identifying exposed individuals.

Radionuclides that emit γ-rays may be determined in biological samples by direct measurement with scintillation or semiconductor detectors. Analysis of α- and β-emitting radionuclides requires chemical separation followed by appropriate measurement techniques. Measurement of so-called total α or β activity may occasionally be useful as a simple screening technique, but there is no method that will determine accurately all the α and β activity in the sample. The technique may be used in routine monitoring situations where intakes are expected to be very low compared with annual limits. The results would not be interpreted quantitatively, but would be used to provide confirmation of satisfactory conditions, an unusual result indicating the need for further investigation which would include radiochemical analysis. Total activity measurements may also be useful following a known contamination event or to identify those samples that merit early attention. Measurements of total α or β activity cannot be used in quantitative evaluations of intake or committed effective dose, unless the radionuclide composition is known.

Measurement of activity in exhaled breath is a useful monitoring technique for some radionuclides such as ^{226}Ra and ^{228}Th since the decay chains of both these radionuclides include gases which may be exhaled. It can also be used to monitor $^{14}CO_2$ formed *in vivo* from the metabolism of ^{14}C-labelled compounds.

7.5.1.3 Air sampling

A Personal Air Sampler (PAS) is a portable device specifically designed for the estimation of intake by an individual worker from a measurement of time-integrated concentration of activity in air in the breathing zone of the worker. A sampling head containing a filter is worn on the upper torso close to the breathing zone. Air is drawn through the filter by a calibrated air pump carried by the worker. Ideally, sampling

rates would be representative of typical breathing rates for a worker (~1.2 m^3 h^{-1}). However, sampling rates of current devices are only about 1/10 of this value. The activity on the filter may be measured at the end of the sampling period to give an indication of any abnormally high exposures. The filters can then be retained, bulked over a longer period, and the activity determined by radiochemical separation and high sensitivity measurement techniques. An estimate of intake during the sampling period can be made by multiplying the measured integrated air concentration by the volume breathed by the worker during the period of intake.

There are three important requirements for a PAS device. Firstly, the sampler should collect sufficient material for the activity corresponding to a significant intake to be measurable in a reasonable counting time. This will depend mainly on the lowest committed effective dose that the PAS is required to detect. Typically, in a routine monitoring programme, the requirement will be to detect annual intakes that in total give rise to committed effective doses greater than 1/10 of the annual dose limit. Secondly, the volume of air aspirated by the sampler should be sufficient to provide a statistically accurate representation of the activity concentration in the breathing zone of the worker. PAS monitoring is most often used for radionuclides such as plutonium, for which a very small number of particles may contain activities that would correspond to a significant intake. The statistics of sampling small numbers of events then becomes the critical factor in determining sampling accuracy. Thirdly, the particle collection characteristics of the sampler should be known. These depend on the aspiration efficiency of the sampling head and the collection efficiency of the filter. The aspiration efficiency is the ratio of the particle concentration in the air entering the sampler to that in the ambient air. It is usually close to unity for particles of aerodynamic diameter less than about 1 μm, but the inertia of larger particles will give a tendency to under- or over-sample according to conditions. Similar effects apply to particles entering the nose and mouth and are taken into account in the ICRP Human Respiratory Tract Model [94I2] (the aspiration efficiency of the respiratory tract is termed inhalability).

A PAS does not provide information on particle size. Nevertheless, it is important either to determine the particle size distribution of the inspirable material or to make realistic assumptions about it, since it can have a marked effect on deposition fractions in the respiratory tract, and hence on dose estimates. This is particularly important now that the recommended default AMAD of 5 μm is intended to be realistic rather than conservative in terms of dose estimation [95D1, 97A1]. All samplers are size selective to a greater or lesser extent, under- or over-sampling at particular particle sizes, and this can result in errors in intake estimation. The aspiration efficiency of a PAS should therefore be determined to indicate whether corrections are necessary. An investigation of the aspiration efficiency of a PAS gave values close to unity up to an aerodynamic diameter of 30 μm under workplace conditions [86M1]. It has been suggested that samplers should be designed to collect the inspirable fraction rather than the total aerosol [81V1]. Use of such samplers would be acceptable, but would require modification of analysis procedures, since the ICRP Respiratory Tract Model implicitly assumes that the *total* aerosol concentration is known.

Static air samplers (SAS) are commonly used to monitor workplace conditions, but can underestimate concentrations in air in the breathing zone of a worker, typically by a factor of up to about 10 [80M1]. Nevertheless, if SAS devices are sited appropriately, a comparison of PAS and SAS measurements can be used to define a PAS:SAS air concentration ratio which can be used in the interpretation of SAS measurement for dose assessment purposes. It should however be recognised that the use of SAS is a relatively indirect method for assessing doses, and use of the results to estimate individual dose requires a careful assessment of exposure conditions and working practices. Apart from their potential use for dose estimation, SAS devices can also provide useful information on radionuclide composition, and on particle size if used with a size analyser such as a cascade impactor.

7.6 Monitoring Programme

(Section 7.6 is reproduced from ICRP Publication 78, 97I2, paras. 81 - 88)

7.6.1 Need for a monitoring programme

ICRP Publication 75 [97I1] recommends that the emphasis in any particular monitoring programme should be on the formal assessment of doses to those workers who are considered likely to receive routinely a significant fraction of the relevant dose limit or who work in areas where exposures could be significant in the event of an accident.

The results of workplace monitoring should give an indication of the likelihood of doses from intakes exceeding 5 mSv a year. Experience has shown that workers involved in the following operations would normally require individual monitoring:

- handling large quantities of gaseous and volatile materials, e.g. tritium and its compounds in large scale production processes, in heavy water reactors, and in luminising;
- uranium mining and processing and fabrication of uranium and mixed oxide fuels;
- processing of plutonium and other transuranic elements;
- processing and use of thorium, and
- production of large quantities of radionuclides and radiopharmaceuticals.

Selection of the type of monitoring programme depends upon the frequency of contamination of the workplace. In situations where contamination events are very infrequent, it is unlikely that routine individual monitoring would be required. Workplace monitoring should be undertaken and the results used to trigger a programme of individual monitoring in relation to special events. However, for the processes listed above, if contamination of the workplace occurs frequently, a routine individual monitoring programme would be appropriate.

For workers who are not routinely employed in areas that are designated as controlled areas in relation to the control of airborne contamination and who are unlikely to have significant intakes of radionuclides, routine monitoring of the workplace will usually be sufficient to provide assurance that intakes are adequately controlled.

7.6.2 Routine monitoring

The required frequency of measurements in a routine monitoring programme depends upon the retention and excretion of the radionuclide, the sensitivity of the measurement techniques available, and the acceptable uncertainty in the estimate of intake and committed effective dose. The measurement technique should be selected so that uncertainties in the measured value are small in relation to the major source of uncertainty which usually lies in the unknown times of intakes. The frequency of measurements within a routine monitoring programme should be chosen so as to reduce the uncertainty arising from the unknown time of intake to an acceptable level.

7.6.3 Special or task-related monitoring

Special monitoring refers to monitoring carried out in actual or suspected abnormal situations. Task-related monitoring is carried out to provide information about a particular operation. Since both special and task-related monitoring relate to distinct events, either real or suspected, one of the problems encountered in interpretation of routine monitoring results does not apply, viz. the time of intake is known. Furthermore, there may be more information about the physical and chemical form of the contaminant.

7.6.4 Confirmatory monitoring

One method of confirming that working conditions are satisfactory is to carry out occasional individual monitoring. Such measurements can be interpreted only qualitatively, but unexpected findings would give grounds for further investigation. Confirmatory monitoring of this type is most useful for those radionuclides that are retained in the body for long periods, and occasional measurements provide a check on the build-up of the activity within the body.

7.6.5 Wound monitoring

When skin is broken, punctured or abraded, radioactive material can penetrate to subcutaneous tissue and thence be taken up by body fluids. Depending upon the radionuclides and the amount of activity it may be necessary to undertake a medical investigation and a programme of special monitoring. In these circumstances, the amount of radioactive material at the site of the wound should be determined taking into account self-attenuation of the radiation in the foreign material and in tissue, as an aid to decisions on excision. If an attempt is made to remove material from the wound, measurements should be made of the removed material and of any activity remaining at the wound site, so as to maintain an activity balance. Subsequently, a series of measurements should be made to determine uptake to body tissues. These measurements may consist of *in vivo* measurements, or urine or faecal excreta monitoring, as appropriate for the particular radionuclides. If whole-body measurements are made, it may be necessary to shield any activity remaining at the wound site. Uptake can be assessed from the data given in Section 7.8.

If medical intervention to prevent uptake or enhance excretion is considered, then it should be noted that any treatment will modify the biokinetic behaviour described by the models given in Section 7.1 and the data in Section 7.7 cannot be used directly to assess committed effective doses when treatment has been administered. When therapy is used following an accidental intake, a programme of special monitoring should be undertaken to follow the distribution and retention of the particular contaminant in the person, and these data should be used to make a specific assessment of committed effective dose for that person.

7.7 Dose Assessment

(Section 7.7 is reproduced from ICRP Publication 78, 97I2, paras. 103 - 109).
Examples for dose estimation from results of *in vitro* measurements are given in Section 10.3.3.9.

7.7.1 Estimation of intake and dose

For special or task-related monitoring when the time of intake is known, the intake can be estimated from the measured results using the predicted values of measured quantities as illustrated by Figures 7.15 to 7.25. If only a single measurement is made, the intake can be determined from the measured quantity M by

$$\text{Intake} = \frac{M}{m(t)} \tag{34}$$

where $m(t)$ is the predicted value at the time of intake t. The intake can be multiplied by the dose coefficient to give the committed effective dose; this can then be compared with the dose limit or any pre-determined investigation level based on dose.

If the measurement indicates that an investigation level has been exceeded, further investigation is required. The nature of the investigation will depend upon the circumstances and the extent to which the investigation level is exceeded. The following should be considered:

- repeated measurements to confirm or refine the initial evaluation, and
- the use of additional monitoring techniques.

If a series of measurements is available, the data in Figures 7.15 to 7.25 provide the time course of the predicted activity (at least over a period of 10 days). The predicted values can then be scaled to obtain the best fit to the measured data points. The best fit is usually taken to be that fit which minimizes the sum of the squares of the residuals, a residual being defined as the number of standard deviations separating a measurement from the fitted curve. The intake is then equal to the value by which the predicted values are scaled.

For routine monitoring, it is assumed that intake took place in the middle of the monitoring interval of T days. For a given measured quantity M obtained at the end of the monitoring interval, the intake is

$$\text{Intake} = \frac{M}{m(T/2)} \tag{35}$$

and the dose from intake in the monitoring interval is obtained by multiplying the intake by the dose coefficient. The dose or intake can be compared with the dose limit or of the activity corresponding to that limit. Alternatively, the dose or intake can be compared with pre-determined investigation levels.

An intake in a preceding monitoring interval may influence the actual measurement result obtained. If more than about 10 % of the actual measured quantity may be attributed to intakes in previous intervals, for which intake and dose have already been assessed, a correction should be made. For a series of measurements in a routine monitoring programme, the following procedure may be observed:

- determine the magnitude of the intake in the first monitoring interval;
- predict from the graphs in Section 7.8 the contribution to the subsequent measurement from this intake;
- subtract this contribution from all subsequent data, and
- repeat above for the next monitoring interval.

Alternative techniques for assessing committed effective dose from a series of measurement values are described in the literature, e.g. [96P1].

If a measured value in a routine monitoring programme exceeds a pre-determined investigation level, further investigation is required. The nature of the investigation will depend upon the circumstances and the extent to which the investigation level is exceeded.

The following should be considered:

- repeated measurements to confirm or refine the initial estimate;
- the use of additional monitoring techniques;
- review of the working conditions and the circumstances of the exposure;
- if default parameter values were used in the original assessment, investigation of the particle size and chemical form of the actual contaminant and selection of more appropriate values, if necessary, and
- in cases of substantial intakes, removal of the contaminated person from work with radioactive materials and investigation of the actual retention and excretion characteristics, in order to refine the dose assessment.

7.7.2 Control of worker doses

The limit on the annual effective dose to a worker applies to the sum of the effective doses from external exposure and committed effective dose from intakes of radionuclides. For practical purposes, the total effective dose E_T can be calculated from the formula:

$$E_T = H_p(d) + \sum_j e_{j,inh}(50) \cdot I_{j,inh} + \sum_j e_{j,ing}(50) \cdot I_{j,ing} \tag{36}$$

where $H_p(d)$ is the personal dose equivalent at a depth d in the body, normally 10 mm for penetrating radiation, $e_{j.inh}(50)$ is the committed effective dose per unit activity intake by inhalation from radionuclide j, integrated over 50 years, $I_{j.inh}$ is the intake of radionuclide j, by inhalation, $e_{j,ing}(50)$ is the committed effective dose per unit activity intake by ingestion from radionuclide j, integrated over 50 years , and $I_{j,ing}$ is the intake of radionuclide j by ingestion.

Strictly, personal dose equivalent is an operational quantity measured in the workplace using personal dosemeters, whereas the committed effective dose quantities are calculated using measurements of other parameters (e.g. air concentrations) in the workplace. However, for practical purposes the two kinds of quantity can be combined in the assessment of the total effective dose.

In the assessment of committed effective doses from internal radionuclides it is often helpful to work in terms of the secondary quantities: Annual Limit on Intake (ALI, Bq); and Derived Air Concentration (DAC, Bqm^{-3}). The ALI is the intake which would lead to a committed effective dose of 20 mSv (the average annual limit on effective dose).

$$\mathrm{ALI} = \frac{0.02}{e(50)} \tag{37}$$

The DAC is the activity concentration in air which would lead to an intake of one ALI assuming a standard breathing rate (1.2 $m^3\,h^{-1}$) and annual working hours (2,000).

$$\mathrm{DAC} = \frac{\mathrm{ALI}}{1.2 \times 2000} \tag{38}$$

These values should not be seen as limits in the way that the 5-year-averaged effective dose is limited, but rather as helpful guides to whether the limits are likely to be approached or exceeded.

7.8 Monitoring data for radionuclides

In this Section illustrative graphs of predicted values of measured quantities (whole-body retention, specific organ retention, daily urinary or faecal excretion) are given in Figs 7.15 to 7.25 as a function of time following a single intake by inhalation, ingestion and injection. The data for the following radionuclides are included: ^{3}H, ^{60}Co, ^{90}Sr, ^{106}Ru, ^{131}I, ^{134}Cs, ^{137}Cs, ^{144}Ce, $^{234, 235, 238}U$, $^{239, 240}Pu$, and ^{241}Am. For inhalation, results are generally given for a single lung clearance Type which is representative of chemical forms present in the workplace (see Table 7.4). For tritium, a graph for inhalation of tritiated water is given which is treated as Class SR-2. In the case of $^{239, 240}Pu$ and ^{241}Am graphs for both Type M and Type S forms are given. For ingestion, f_1 values recommended for materials in the workplace are applied (see Table 7.6). For direct entry into the blood the graphs are applicable to soluble (transportable) forms of radionuclides that have been directly injected into the bloodstream or have entered the body by inhalation, ingestion or through skin/wound contamination.

The Human Respiratory Tract Model in ICRP Publication 66 [94I2] was used to calculate particle deposition and respiratory tract clearance of the deposited particles. The subject exposed to the aerosols was the ICRP reference worker doing light work: defined as light exercise with the ventilation rate of 1.5 $m^3\,h^{-1}$ for 5.5 h + sitting with the rate of 0.54 $m^3\,h^{-1}$ for 2.5 h. The following ICRP default values [94I2] for the physical characteristics of the radioactive aerosols were used.

- Activity Median Aerodynamic Diameter (AMAD) = 5 μm
- geometric standard deviation of particle size = 2.5
- particle density = 3 g cm^{-3}
- particle shape factor = 1.5

The GI tract model in ICRP Publication 30 [79I1] was used. The rate constant λ_B for the absorption of the materials from the small intestine to the blood was obtained from the value of f_1, the fraction of materials absorbed into blood from the small intestine, using the equation:

$$\lambda_B = f_1 \lambda_{SI} / (1 - f_1) \tag{39}$$

where λ_{SI} is the rate constant of material transfer from the small intestine to the upper large intestine. If f_1 value is 1, 0.99 was taken for calculation, which is in line with the ICRP publications.

The latest ICRP biokinetic models at 2003 were used, which are given in the ICRP publications listed in Table 7.1.

In the graphs of Figs 7.15 to 7.25 body or organ retention for day 1 means the content at the end of day 1 etc. For excreted activities, the value at day 1 represents the activity excreted during the first day after intake, corrected for radioactive decay to the end of day 1. One exception to this is for the intake of tritiated water; the activity concentration in urine was calculated by dividing the whole body activity at the time of sampling by the volume of body water, 42 litres. In the context of *in vivo* measurements, the following definitions are relevant. Whole-body retention is the sum of systemic material (including that in the urinary bladder) and material retained within the respiratory and gastrointestinal tracts. The lung retention is taken to be the sum of the contents of the thoracic lymph nodes and the bronchial, bronchiolar, and alveolar-interstitial regions.

Hydrogen-3 (half-life = 12.3 y)

Possible chemical forms of ^{3}H to which workers are exposed include tritium gas (HT), tritiated water (HTO) and organically bound tritium (OBT) [89I1].

Tritium emits low energy β^--particles (0.0057 MeV in average) with 100 % yield and is readily detected by liquid scintillation counting of a urine sample. A typical detection limit readily achievable in monitoring programme is 100 Bq/l for urine samples [97I2]. Since the activity concentration of HTO in urine is assumed to be equal to that in body water, the analysis of HTO in a urine sample is used to give activity concentration in body water at the time of sample collection.

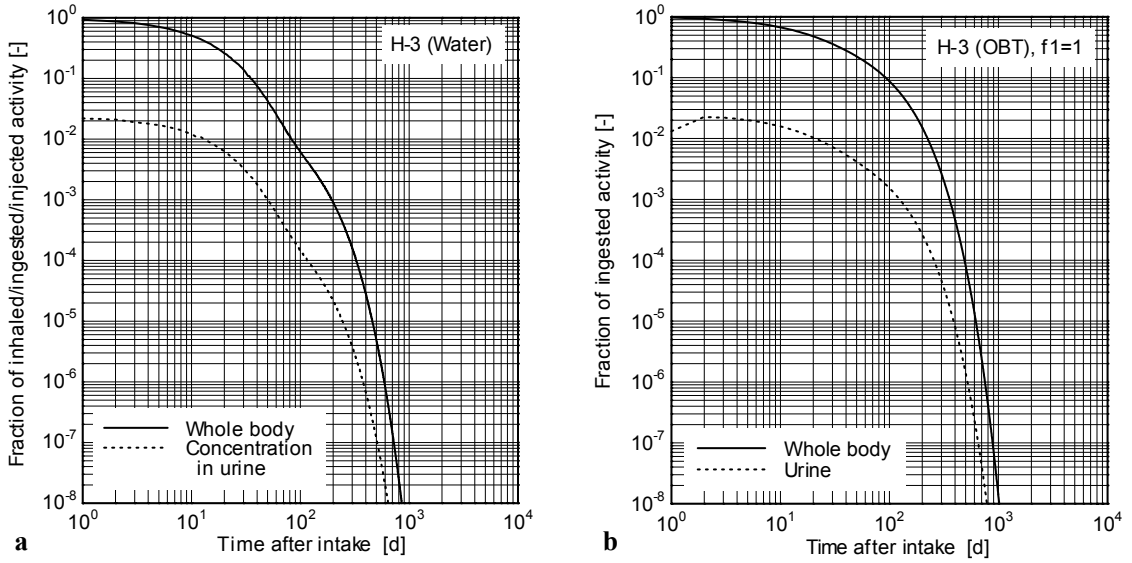


Fig. 7.15. Predicted whole-body retention, daily urinary excretion or concentration in urine of ^{3}H as a function of time following acute intake of unit activity of ^{3}H via **a** all intake routes for tritiated water, **b** ingestion of organically bound tritium (OBT), the f_1 value of which is 1.0.

Cobalt-60 (half-life = 5.27 y)

Insoluble compounds of cobalt, e.g. oxides, hydroxides, halides and nitrates are assigned to Type S (f_1=0.05 for workers, 0.01 for adult members of the public) and all other compounds to Type M (f_1=0.1) by ICRP [94I1].

Cobalt-60 emits two high-energy γ-rays (1.173, 1.332 MeV) per disintegration, which are highly penetrable radiations and therefore readily detected by photon detectors positioned outside the body. A typical detection limit readily achievable in monitoring programme is 50 Bq of ^{60}Co in the whole body and 100 Bq in the lungs [97I2]. Gamma-ray spectrometry on biological samples permits detection of 1 Bq/l of ^{60}Co in urine and 1 Bq per sample of faeces [97I2].

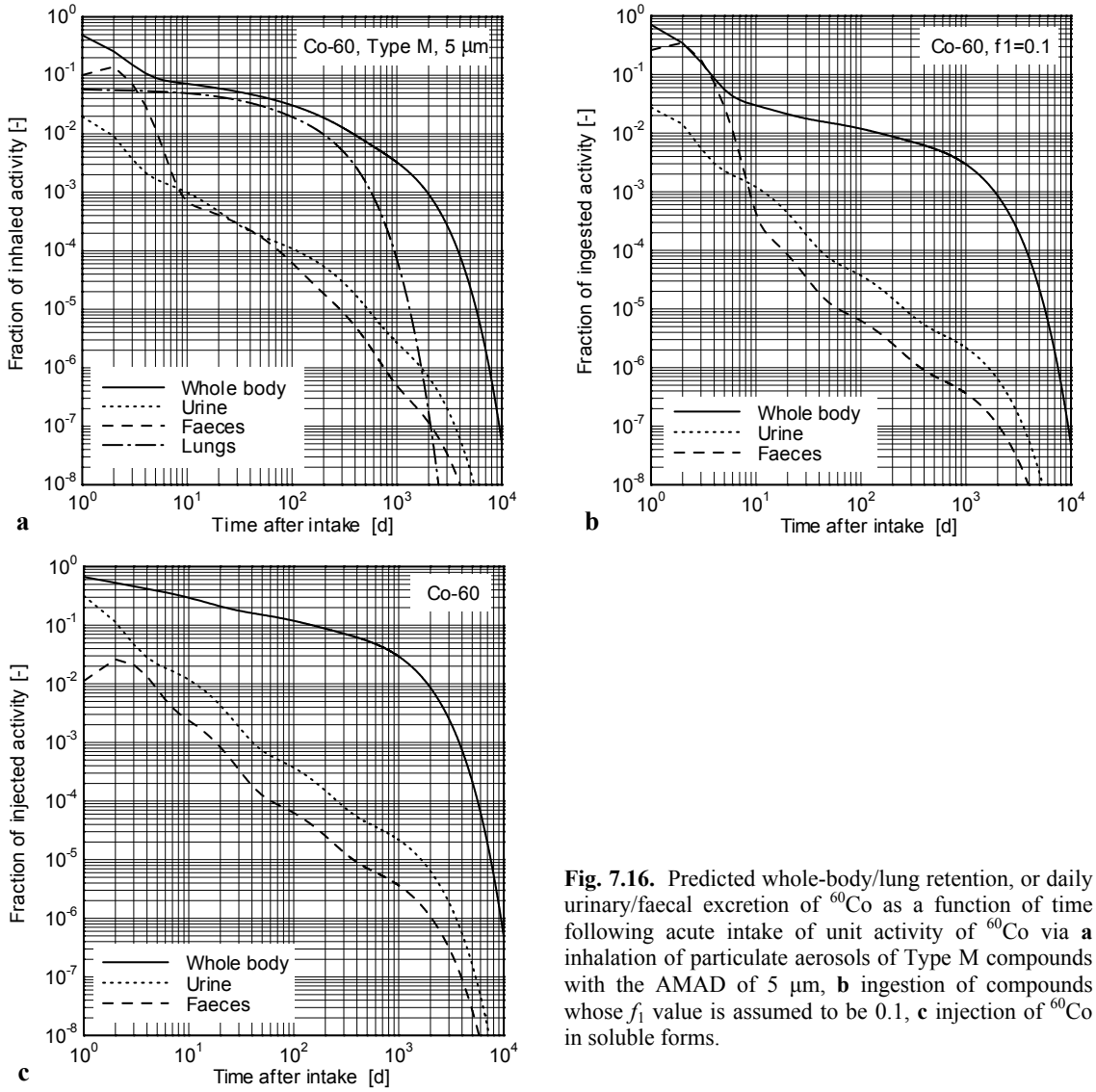


Fig. 7.16. Predicted whole-body/lung retention, or daily urinary/faecal excretion of ^{60}Co as a function of time following acute intake of unit activity of ^{60}Co via **a** inhalation of particulate aerosols of Type M compounds with the AMAD of 5 μm, **b** ingestion of compounds whose f_1 value is assumed to be 0.1, **c** injection of ^{60}Co in soluble forms.

Strontium-90 (half-life = 29.1 y)

All compounds of strontium possibly present in the work place, except for strontium titanate ($SrTiO_3$), are assigned to Type F (f_1=0.3) by ICRP [94I1]. Strontium titanate is assigned to Type S (f_1=0.01) [94I1].

Strontium-90 emits β^--particles (0.20 MeV in average) with 100 % yield but does not emit energetic photons. Internally deposited Sr-90 is therefore measured by β counting of a urine sample following chemical separations. A typical detection limit readily achievable in monitoring programme is 1 Bq/l of ^{90}Sr in urine [97I2].

The decay product of ^{90}Sr, ^{90}Y is radioactive (half-life = 64 h), which emits high-energy β^--particles (0.99 MeV in average) with 100 % yield per disintegration of ^{90}Sr. Strontium-90/yttrium-90 in the body can sometimes be measured by photon detectors positioned outside the body via the bremsstrahlung produced, though the minimum detectable activities are relatively high [99I2].

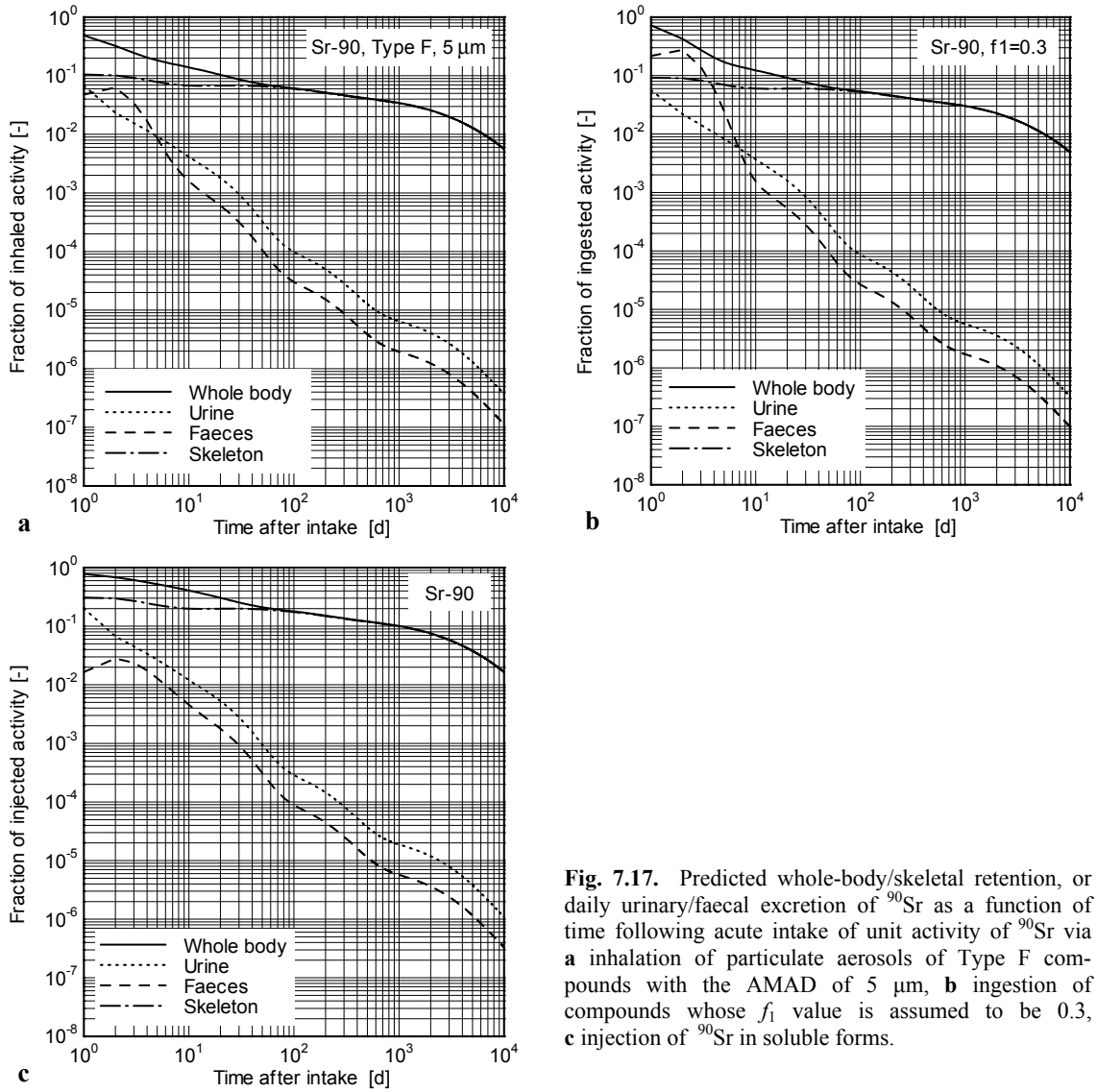


Fig. 7.17. Predicted whole-body/skeletal retention, or daily urinary/faecal excretion of ^{90}Sr as a function of time following acute intake of unit activity of ^{90}Sr via **a** inhalation of particulate aerosols of Type F compounds with the AMAD of 5 μm, **b** ingestion of compounds whose f_1 value is assumed to be 0.3, **c** injection of ^{90}Sr in soluble forms.

Ruthenium-106 (half-life = 1.01 y)

Oxides and hydroxides of ruthenium are assigned to Type S (f_1=0.05), halides to Type M (f_1=0.05) and all other compounds to Type F (f_1=0.05) by ICRP in Publication 68 for workers [94I1].

Though ^{106}Ru does not emit energetic photons, the radioactive decay product ^{106}Rh (half-life = 30 s) emits γ-rays of 0.512 MeV (20.6 % per disintegration of ^{106}Ru), 0.622 MeV (9.8 %) and 1.050 MeV (1.5 %). They are penetrable radiations and therefore readily detected by photon detectors positioned outside the body. A typical detection limit readily achievable in monitoring programme is 200 Bq of ^{106}Ru in the whole body [97I2]. Gamma-ray spectrometry on biological samples permits detection of 5 Bq/l of ^{106}Ru in urine [97I2].

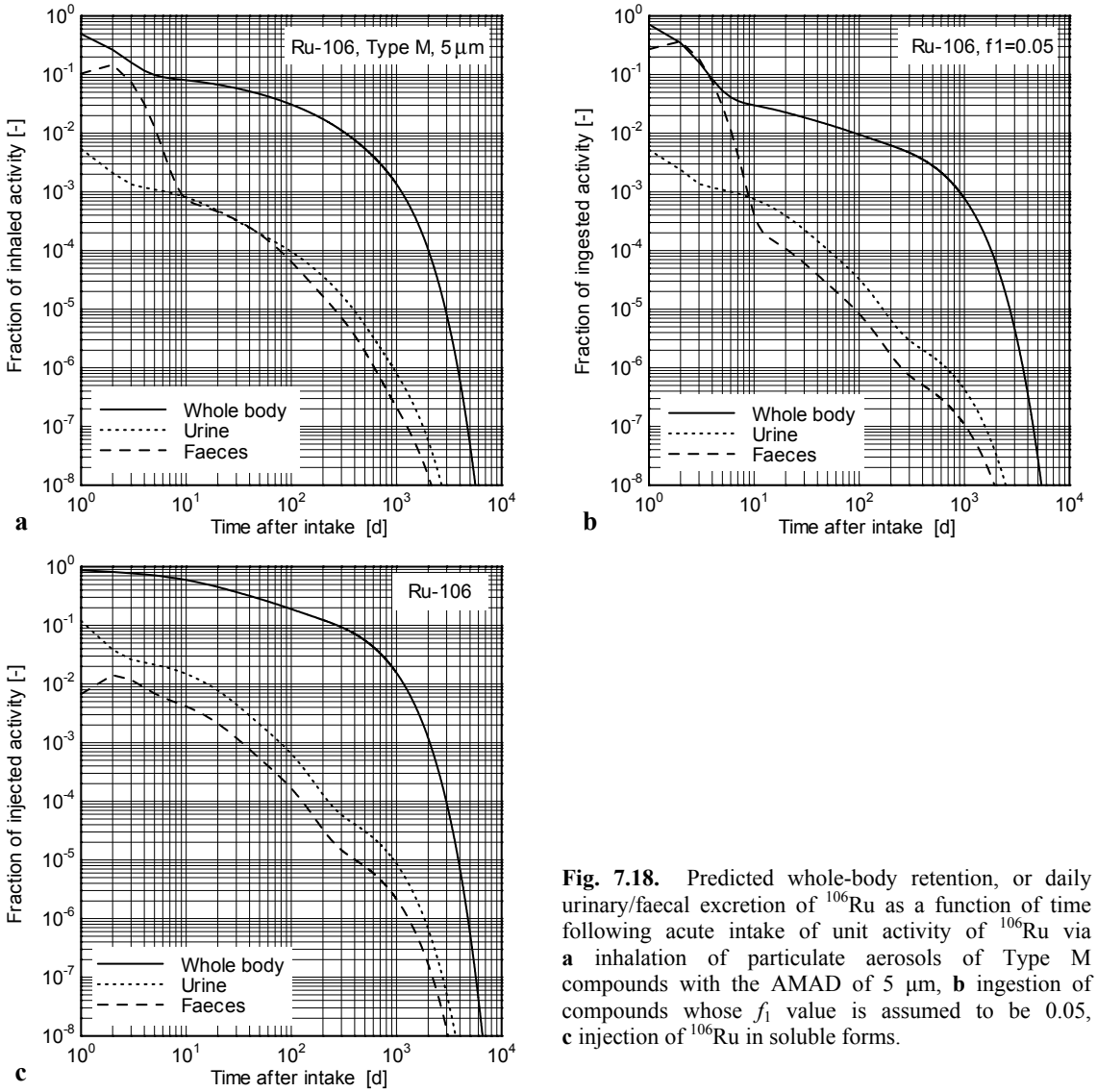


Fig. 7.18. Predicted whole-body retention, or daily urinary/faecal excretion of ^{106}Ru as a function of time following acute intake of unit activity of ^{106}Ru via **a** inhalation of particulate aerosols of Type M compounds with the AMAD of 5 µm, **b** ingestion of compounds whose f_1 value is assumed to be 0.05, **c** injection of ^{106}Ru in soluble forms.

Iodine-131 (half-life = 8.04 d)

Elemental iodine vapour is assigned to Class SR-1 (10 % deposition in ET_1, 40 % in ET_2, 50 % in BB), with Type F clearance [94I1, 95I2]. Methyl iodide gas is assigned to Class SR-1 (70 % deposition in ET_2 and the lungs), with Type V clearance [95I2]. For workers particulate aerosols of iodine compounds are all assigned to Type F (f_1=1.0) [94I1].

Iodine-131 emits γ-rays of 0.284 MeV (6.1 % yield), 0.364 MeV (81.2 %), and 0.637 MeV (7.3 %). The principal γ-ray emissions at 0.364 MeV are used for measurement of ^{131}I by photon detectors positioned just outside the thyroid, in which iodine is highly accumulated. A typical detection limit readily achievable in monitoring programme is 100 Bq of ^{131}I in the thyroid [97I2]. Gamma-ray spectrometry on biological samples permits detection of 1 Bq/l of ^{131}I in urine [97I2].

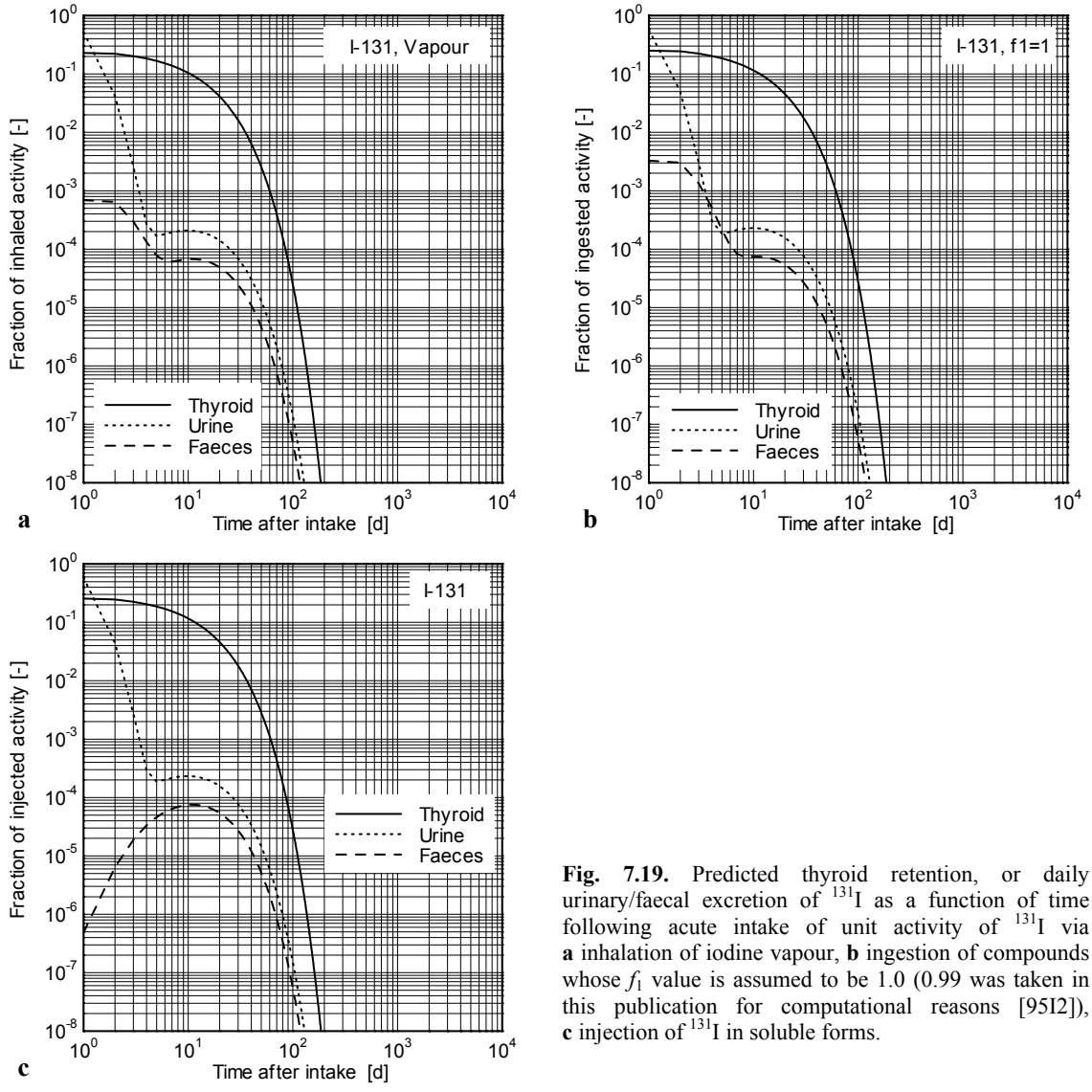


Fig. 7.19. Predicted thyroid retention, or daily urinary/faecal excretion of ^{131}I as a function of time following acute intake of unit activity of ^{131}I via **a** inhalation of iodine vapour, **b** ingestion of compounds whose f_1 value is assumed to be 1.0 (0.99 was taken in this publication for computational reasons [95I2]), **c** injection of ^{131}I in soluble forms.

Caesium-134 (half-life = 2.06 y)

All compounds of caesium possibly present in work place are assigned to Type F (f_1=1.0) by ICRP [94I1] although it is recognised that other forms may be present in the environment [95I2].

Caesium-134 emits γ-rays of 0.563 MeV (8.4 % yield), 0.569 MeV (15.4 %), 0.605 MeV (97.6 %), 0.796 MeV (85.4 %) and 0.802 MeV (8.7 %), which are penetrable radiations and therefore readily detected by photon detectors positioned outside the body. A typical detection limit readily achievable in monitoring programme is 50 Bq of ^{134}Cs in the whole body [97I2]. Gamma-ray spectrometry on biological samples permits detection of 1 Bq/l of ^{134}Cs in urine [97I2].

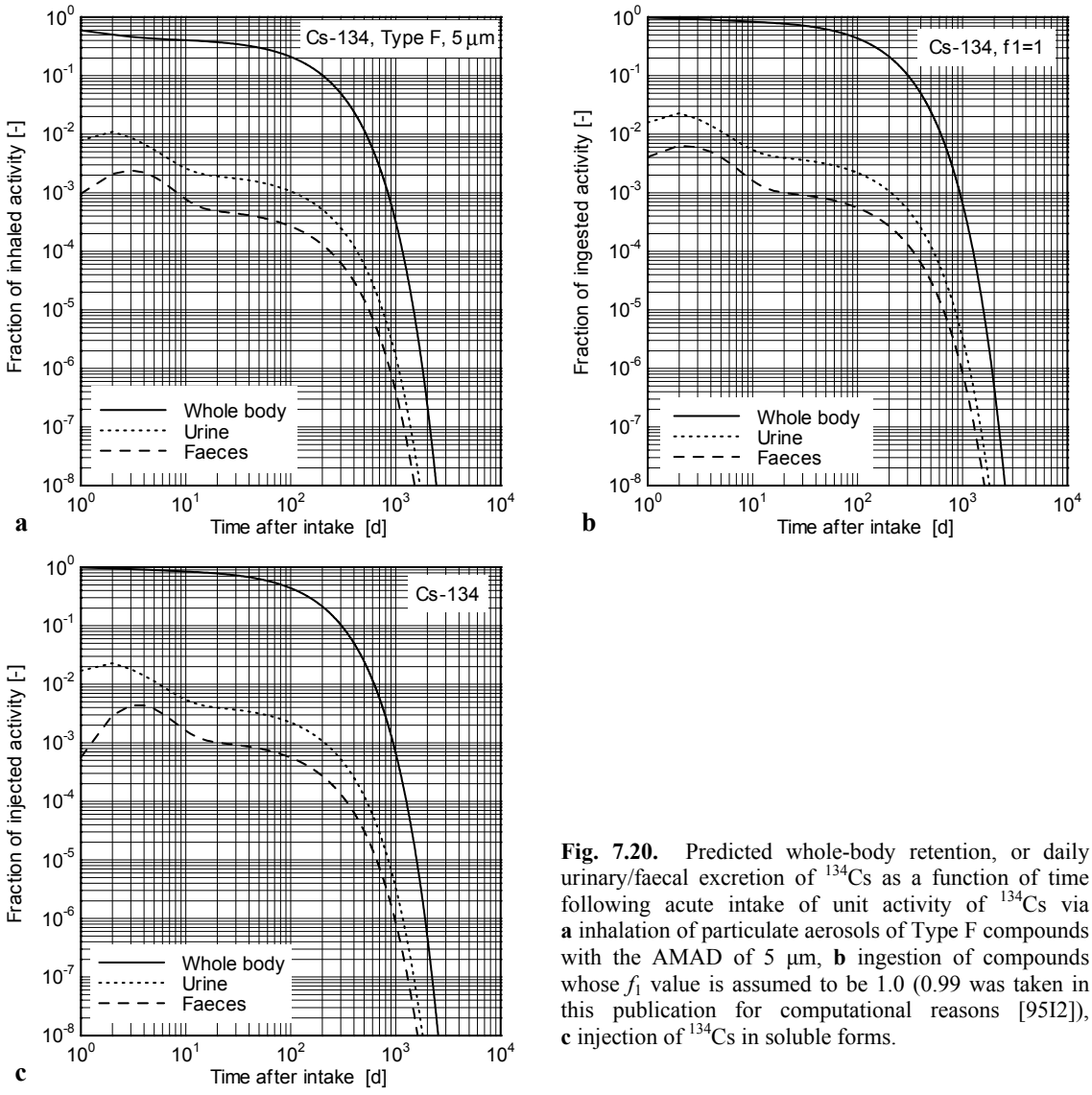


Fig. 7.20. Predicted whole-body retention, or daily urinary/faecal excretion of ^{134}Cs as a function of time following acute intake of unit activity of ^{134}Cs via **a** inhalation of particulate aerosols of Type F compounds with the AMAD of 5 µm, **b** ingestion of compounds whose f_1 value is assumed to be 1.0 (0.99 was taken in this publication for computational reasons [95I2]), **c** injection of ^{134}Cs in soluble forms.

Caesium-137 (half-life = 30.0 y)

All compounds of caesium possibly present in work place are assigned to Type F (f_1=1.0) by ICRP [94I1] although it is recognised that other forms may be present in the environment [95I2].

Though ^{137}Cs does not emit energetic photons, the radioactive decay product ^{137m}Ba (half-life = 2.55 min) emits γ-rays of 0.662 MeV (85.0 % per disintegration of ^{137}Cs), which are penetrable radiations and therefore readily detected by photon detectors positioned outside the body. A typical detection limit readily achievable in monitoring programme is 50 Bq of ^{137}Cs in the whole body [97I2]. Gamma-ray spectrometry on biological samples permits detection of 1 Bq/l of ^{137}Cs in urine [97I2].

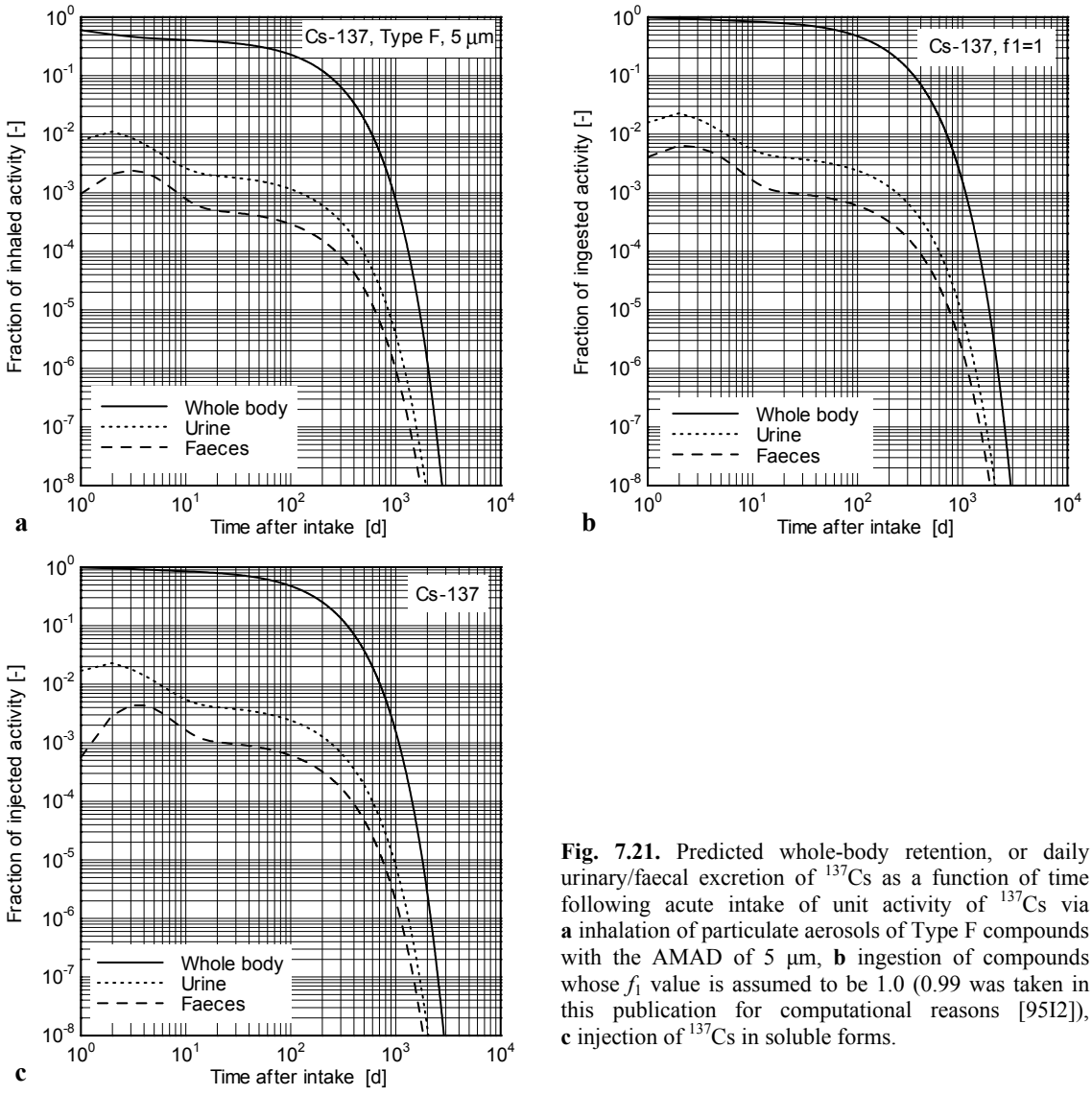


Fig. 7.21. Predicted whole-body retention, or daily urinary/faecal excretion of ^{137}Cs as a function of time following acute intake of unit activity of ^{137}Cs via **a** inhalation of particulate aerosols of Type F compounds with the AMAD of 5 µm, **b** ingestion of compounds whose f_1 value is assumed to be 1.0 (0.99 was taken in this publication for computational reasons [95I2]), **c** injection of ^{137}Cs in soluble forms.

Cerium-144 (half-life = 284 d)

Oxides, hydroxides and fluorides of cerium are assigned to Type S (f_1=0.0005) and all other compounds to Type M (f_1=0.0005) by ICRP in Publication 68 [94I1].

Cerium-144 emits γ-rays of 0.080 MeV (1.6 % yield) and 0.134 MeV (10.8 %). The radioactive decay product of ^{144}Ce, ^{144}Pr emits γ-rays of 0.697 MeV (1.5 % per disintegration of ^{144}Ce). Because of their low abundances, detection limits of *in vivo* counting for ^{144}Ce are relatively high; a typical detection limit that can be readily achieved is 10 kBq of ^{144}Ce in the whole body [88I1]. Detection limits lower than this value are required for routine monitoring. Urine monitoring is not recommended, because cerium in the body is tenaciously retained and hardly excreted (biological half-life = 3500 d [79I1]).

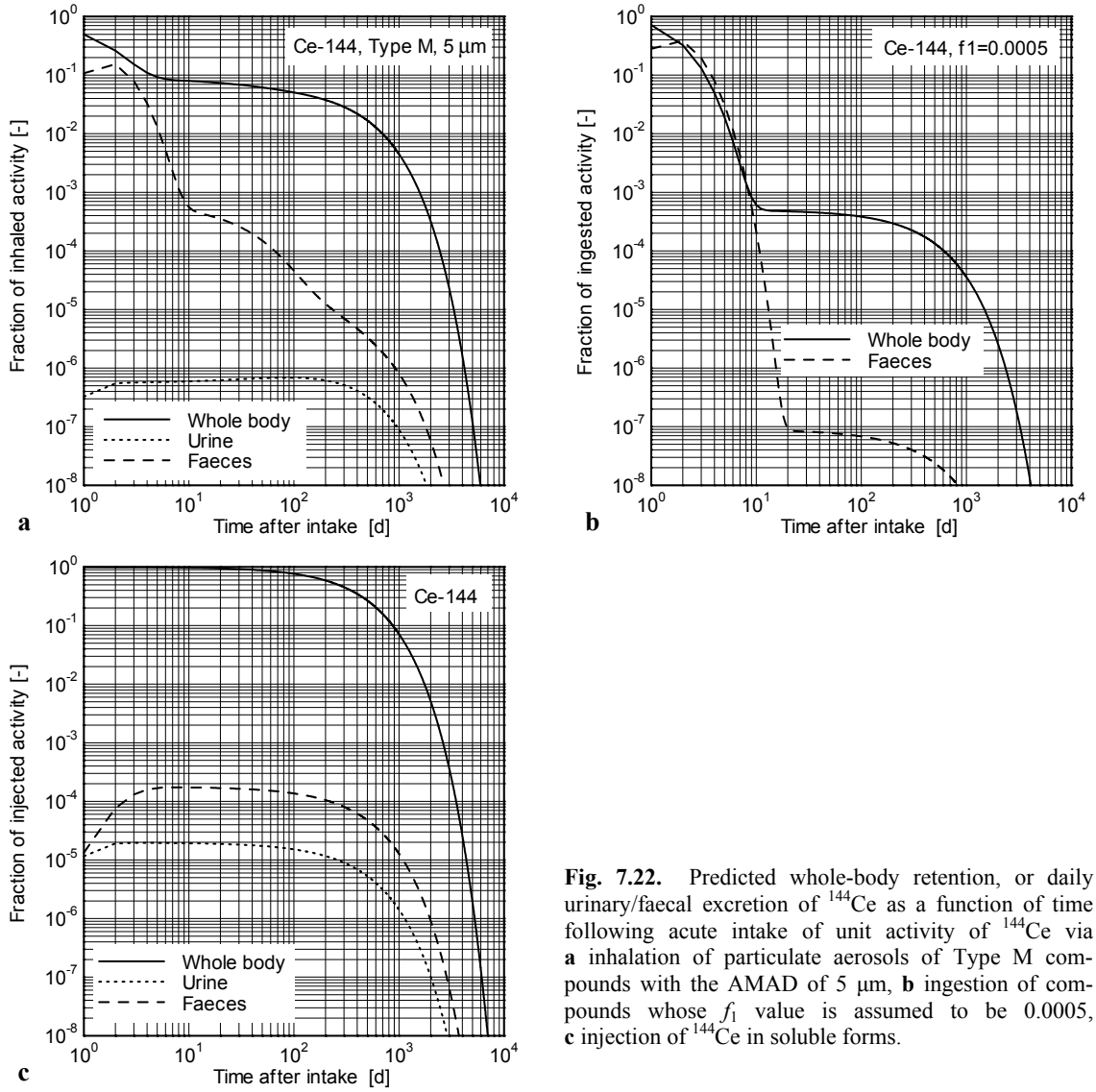


Fig. 7.22. Predicted whole-body retention, or daily urinary/faecal excretion of ^{144}Ce as a function of time following acute intake of unit activity of ^{144}Ce via **a** inhalation of particulate aerosols of Type M compounds with the AMAD of 5 µm, **b** ingestion of compounds whose f_1 value is assumed to be 0.0005, **c** injection of ^{144}Ce in soluble forms.

Uranium-234 (half-life = 2.44×10^5 y), -235 (half-life = 7.04×10^8 y), -238 (half-life = 4.47×10^9 y)

In Publication 68 [94I1], ICRP assigned most hexavalent compounds of uranium, e.g. UF_6, UO_2F_2 and $UO_2(NO_3)_2$ to Type F (f_1 = 0.02), less soluble compounds, e.g. UO_3, UF_4, UCl_4 and most other hexavalent compounds to Type M (f_1 = 0.02) and highly insoluble compounds, e.g. UO_2 and U_3O_8 to Type S (f_1 = 0.002).

Principal isotopes of uranium (^{234}U, ^{235}U, ^{238}U) are α-emitting radionuclides and do not emit energetic photons except for ^{235}U. Internally deposited uranium-isotopes are therefore measured by α-spectrometry on biological samples following radiochemical separation. A typical detection limit is 10 mBq/l in urine and 10 mBq in faeces [97I2]. Uranium-235 emits γ-rays of 0.144 MeV (10.5 % yield), 0.186 MeV (54.0 %) and 0.205 MeV (4.7 %). They are used for lung counting of ^{235}U. A typical detection limit is 200 Bq [97I2]. For routine monitoring, the detection limits for α-spectrometry are adequate, but those for lung counting would not permit detection of intakes at annually limited levels [97I2].

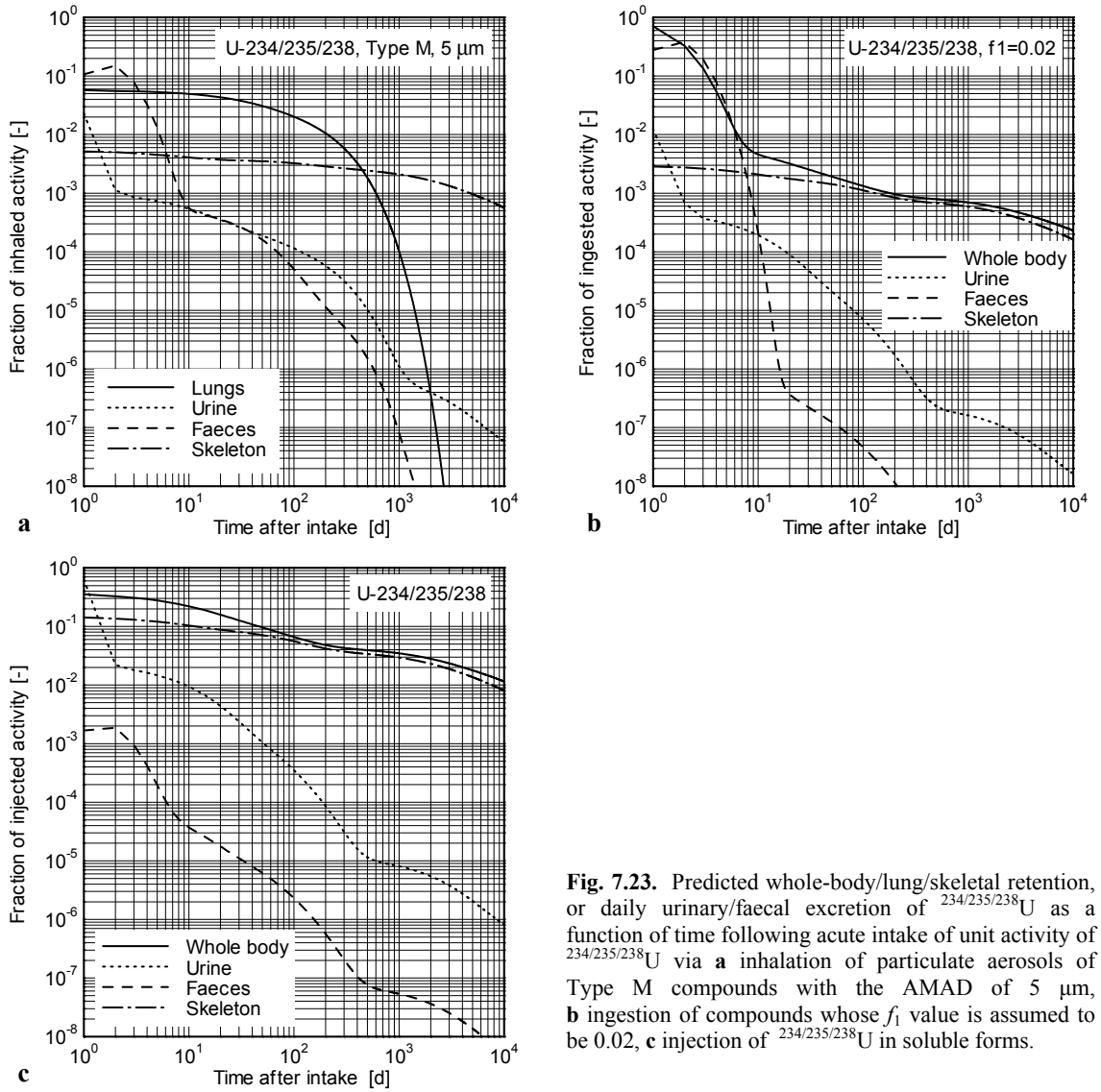
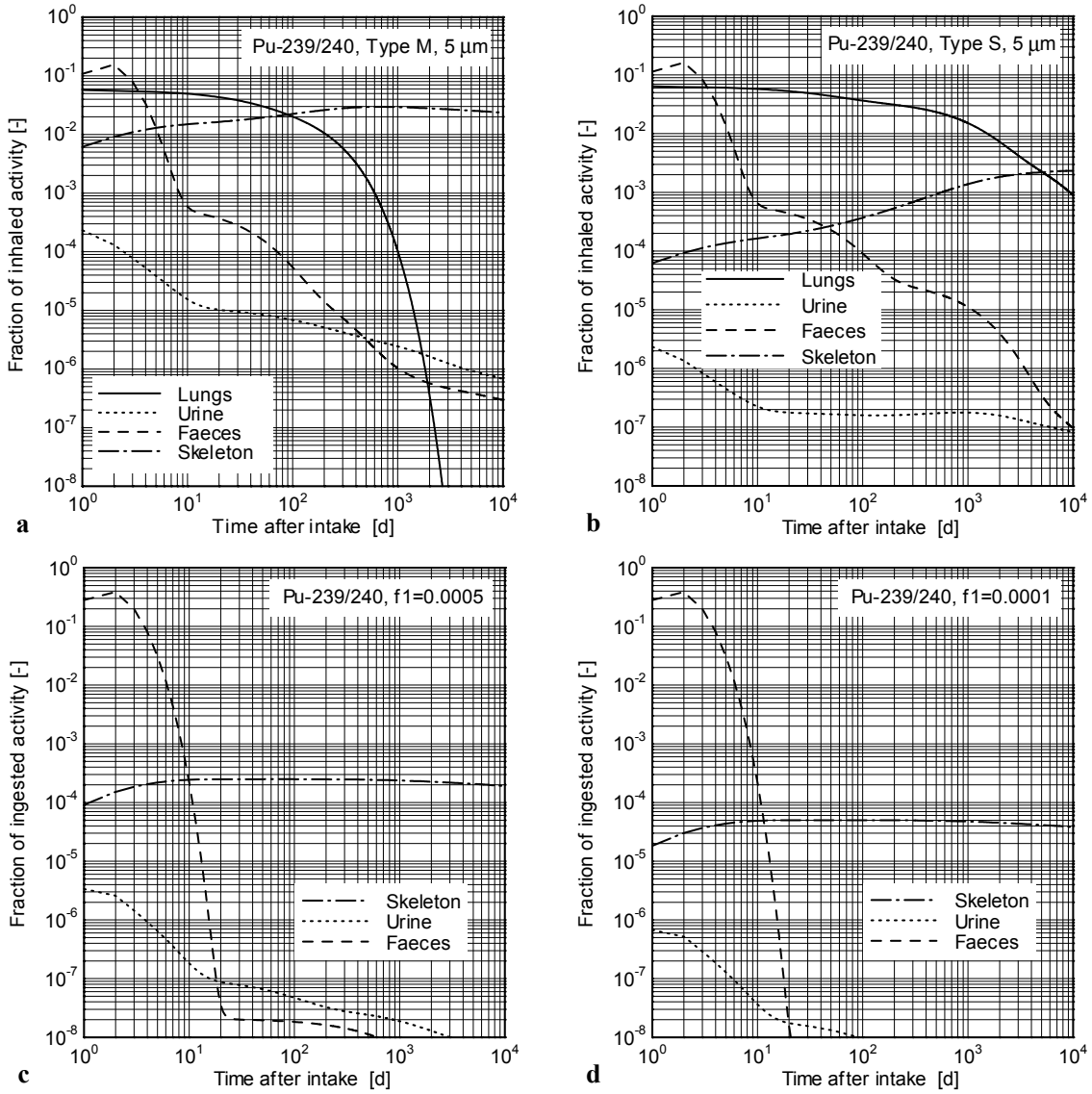


Fig. 7.23. Predicted whole-body/lung/skeletal retention, or daily urinary/faecal excretion of $^{234/235/238}$U as a function of time following acute intake of unit activity of $^{234/235/238}$U via **a** inhalation of particulate aerosols of Type M compounds with the AMAD of 5 µm, **b** ingestion of compounds whose f_1 value is assumed to be 0.02, **c** injection of $^{234/235/238}$U in soluble forms.

Plutonium-239 (half-life = 2.41×10^4 y), -240 (half-life = 6.54×10^3 y)

In Publication 68 [94I1], ICRP assigned insoluble oxides of plutonium, e.g. high-fired PuO_2, a common chemical form in nuclear industry, to Type S (f_1=0.00001) and all other compounds to Type M (f_1=0.0005). Among Type M compounds, f_1-value of nitrates is assumed to be 0.0001 [94I1].

Plutonium-239/240 are α-emitting radionuclides and do not emit energetic photons. Internally deposited $^{239/240}$Pu are therefore measured by α-spectrometry on biological samples following radiochemical separation. A typical detection limit is 1 mBq/l in urine and 1 mBq in faeces [97I2]. Emission of low energy characteristic X-rays (0.014 - 0.020 MeV) are used for lung counting of $^{239/240}$Pu. A typical detection limit is 2 kBq, which is not adequate for routine monitoring [97I2].



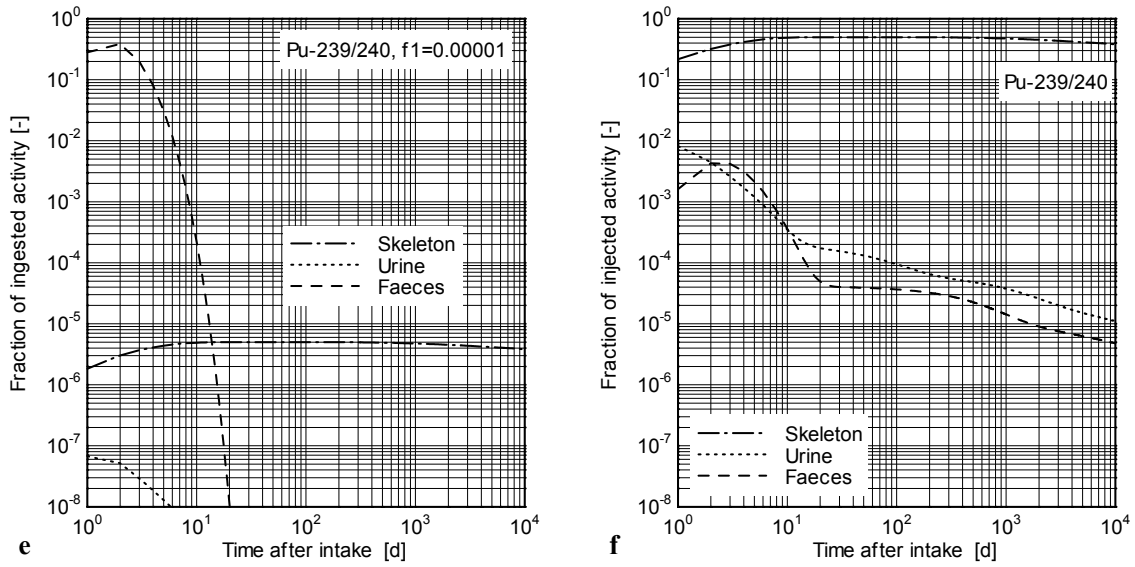


Fig. 7.24. Predicted lung/skeletal retention, or daily urinary/faecal excretion of $^{239/240}$Pu as a function of time following acute intake of unit activity of $^{239/240}$Pu via **a** inhalation of particulate aerosols with the AMAD of 5 μm of Type M compounds, **b** Type S compounds, **c** ingestion of compounds whose f_1 value is assumed to be 0.0005, **d** 0.0001, **e** 0.00001, **f** injection of $^{238/2390}$Pu in soluble forms.

Am-241 (half-life = 4.32×10^2 y)

All compounds of americium possibly present in work place are assigned to Type M (f_1=0.0005) by ICRP [94I1]. Based on several experimental results, ICRP considers that the trace contaminant ^{241}Am that has grown from ^{241}Pu in matrices of nuclear fuels behaves similarly to the bulk materials [95I2]. For this reason, Type S as well as Type M is taken in this publication.

Americium-241 is α-emitting radionuclide accompanied with low-energy (0.060 MeV) γ-ray emission with 35.7 % yield. Internally deposited ^{241}Am can be measured by both indirect and direct methods. A typical detection limit of α-spectrometry following radiochemical separation is 1 mBq/l in urine and 1 mBq in faeces [97I2]. These detection limits are adequate for both special and routine monitoring. Typical detection limits of *in vivo* measurements are 20 Bq for lungs and 20 Bq for skeleton. These detection limits are not necessarily adequate for routine monitoring [97I2].

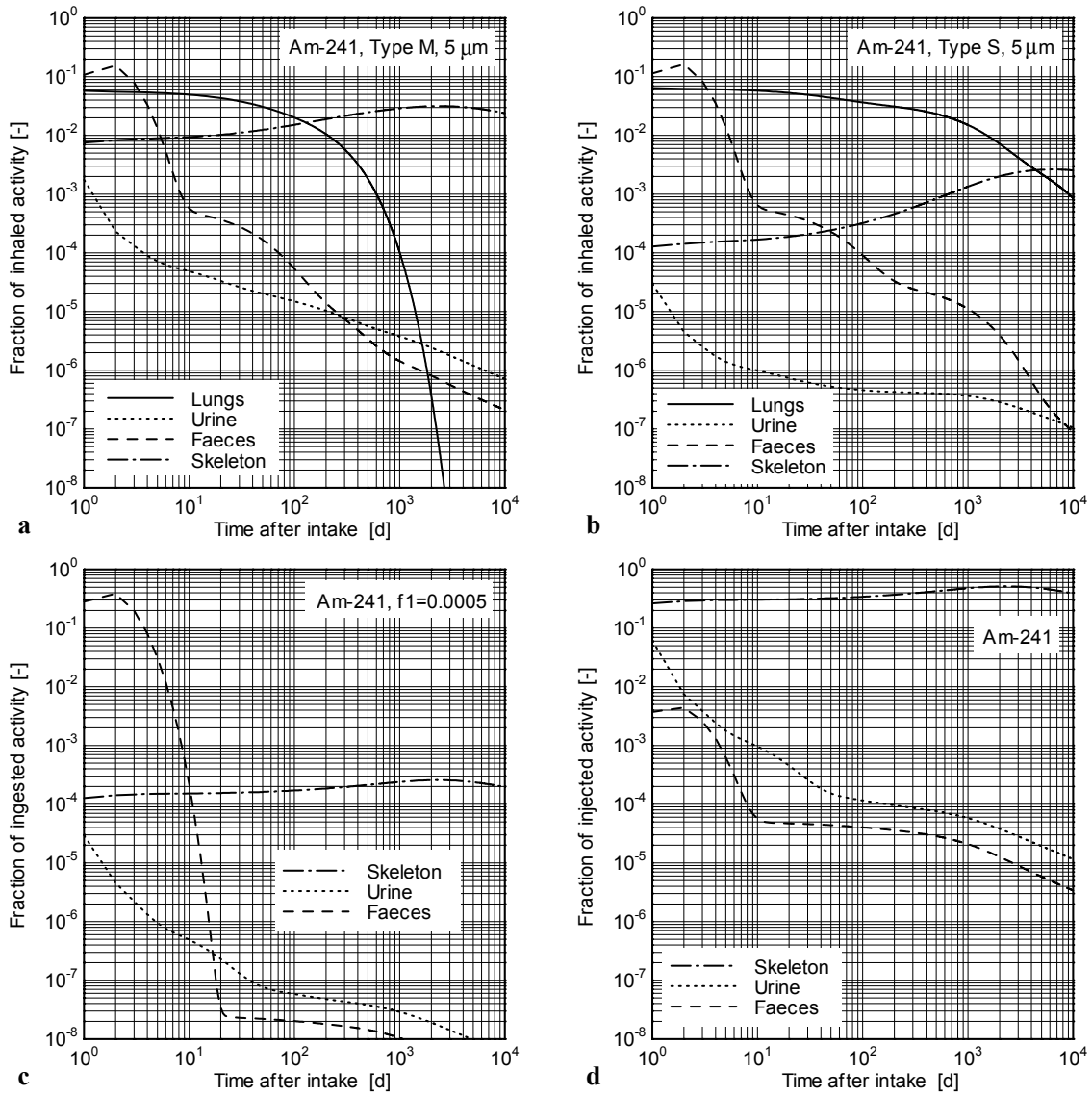


Fig. 7.25. Predicted lung/skeletal retention, or daily urinary/faecal excretion of ^{241}Am as a function of time following acute intake of unit activity of ^{241}Am via **a** inhalation of particulate aerosols with the AMAD of 5 µm of Type M compounds, **b** Type S compounds, **c** ingestion of compounds whose f_1 value is assumed to be 0.0005, **d** injection of ^{241}Am in soluble forms.

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